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A
TEXT-BOOK OF MEDICINE

FOR STUDENTS AND PRACTITIONERS

BY

DR. ADOLF STRÜMPELL
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Second American Edition

TRANSLATED BY PERMISSION FROM THE SECOND AND THIRD, AND
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WITH ONE HUNDRED AND NINETEEN ILLUSTRATIONS

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PREFACE TO THE SIXTH EDITION.

ALTHOUGH the fifth edition of this text-book was very large, a year's time has brought so pressing a demand for a new one, that the author, in preparing it, has been obliged to confine himself to the most essential improvements and additions. There is an entirely new chapter on influenza, a disease which had almost been forgotten, when the last great epidemic brought it prominently to medical attention. Quite important changes and additions have been made in the chapters on cholera, malaria, diseases of the nose and larynx, syringomyelia, and diabetes. The appendix on poisons has also been considerably enlarged.

The author sends out this new edition with the wish that it may not only be of practical benefit to those for whom it is written, but may also add to their pleasure and interest in their vocation.

ADOLF STRÜMPELL.

ERLANGEN, *May*, *1890*.

PREFACE TO THE FIRST EDITION.

In the work which is now offered to the public I have made an attempt to give an account of our present knowledge in the field of the special pathology and treatment of internal diseases. This account, although brief, I have endeavored to make as complete as possible in regard to all important and certainly established facts. While everything hypothetical has been wholly omitted or only briefly referred to, I have tried, on the other hand, not only to enumerate the facts of clinical experience with sufficient accuracy, but also and especially to make the reader comprehend the development and the internal connection of the different morbid phenomena by constantly referring to the data of general pathological and anatomical research. In regard to treatment, the limits of our knowledge will often be apparent, but I believe that I have paid a sufficient regard to the needs of practice. In order to avoid repetitions, only a small number of complete prescriptions have been inserted in the text, but an abundant and well-arranged formulary has been added as an appendix at the end of the work.*

Although in the composition of this text-book I have of course made very great use of the later literature of medicine, still the experienced reader will recognize in not a few places the results of the author's own experience and observation. These results are drawn from more than six years' active work in the medical clinique here, to the abundant material of which I have been fortunate enough to have access as assistant first to C. Wunderlich and then to E. Wagner.

ADOLF STRÜMPELL.

LEIPSIC, *1 March, 1883.*

[* Owing to the differences between our Pharmacopœia and practice and those of Germany, it has been thought best to omit this appendix.—TRANS.]

TRANSLATORS' PREFACE
TO THE REVISED EDITION.

THIS translation is already used as a text-book or as a work of reference in some twenty-eight medical schools in America, and we trust that it will continue to meet with approval. After preparing this revised edition, the translators feel moved to express their admiration of the industry and carefulness as well as the sound judgment and great learning of their author. Scarcely a page has escaped some correction or addition; so that the work is thoroughly brought up to date.

THE TRANSLATORS.

BOSTON, *June, 1892.*

TRANSLATORS' PREFACE
TO THE FIRST AMERICAN EDITION.

THIS translation was made from the second German edition. After the work had been sent to the press in May, we learned that a third edition of the volume on nervous diseases had appeared in Germany. We therefore recalled our manuscript, and incorporated into it all the changes and additions that had been made in that edition.

We have tried to make the translation as exact as possible, but, in a few instances, we have taken the liberty of adding a word or a phrase to make the meaning clearer. With Dr. Shattuck's approval we have added a few foot-notes to the section on nervous diseases, embodying the results of investigations made subsequently to the appearance of the original.

In regard to the nomenclature of physical signs in diseases of the lungs, we have departed somewhat from the original in order to have our nomenclature conform to that proposed at the meeting of the American Medical Association in May, 1885, by the late Dr. Austin Flint, chairman of a committee appointed to prepare such a nomenclature at the International Medical Congress in 1881. This may explain certain unusual terms, such as "small râles."

We have not attempted to adapt the treatment to the United States Pharmacopœia. As a rule, when the preparation mentioned was described in Stillé and Maisch's National Dispensatory (second edition, 1879), we have made no comment. In other cases we have added the formula of the preparation either in a foot-note or in parenthesis. In a very few cases we have substituted an officinal (U. S. P.) preparation which was almost identical. As the metric system is not yet in active use, we have substituted for it approximate equivalents in the old system. We have considered it needlessly precise, however, to give the exact equivalents in tenths of a degree or hundredths of a grain. In every instance we have retained the author's figures in parenthesis, and we have added tables of weights and measures in an appendix. Measures of length have been left in the metric system.

In place of the original Fig. 106, page 763—specimens of handwriting in general paralysis, in German script, and in the German language—we have substituted other specimens selected from a large number kindly sent us by Dr. E. P. Elliot, first assistant at the Danvers Lunatic Hospital.

Our thanks are due to Dr. G. L. Walton, of this city, for his assistance at a critical moment in the work, and to other friends who have given us aid and encouragement during the progress of our labors.

THE TRANSLATORS.

BOSTON, *November, 1886.*

EDITOR'S PREFACE.

THE appearance of a sixth German edition, and the demand for a revised edition of the American translation, seem to show that the high estimate formed of this work was reasonable.

In the present edition some notes have been omitted, others changed, and some new ones added. For most of the new notes in the section on Nervous Diseases I have to thank Dr. Knapp, one of the translators. His additions have been signed "K." For the others, with all sins of omission and commission, I alone am responsible.

FREDERICK C. SHATTUCK.

BOSTON, *September, 1892.*

CONTENTS.

Acute General Infectious Diseases.

CHAPTER PAGE
I. Typhoid Fever . . . 1
Phenomena and Complications relating to the Separate Organs . . . 9
Peculiarities in the Course of the Disease . . . 18
Relapses of Typhoid Fever . . . 19
II. Typhus Fever . . . 28
III. Relapsing Fever . . . 31
IV. Scarlet Fever . . . 36
V. Measles . . . 46
VI. Rötheln . . . 51
VII. Small-pox . . . 52
Variola Vera . . . 53
Varioloid . . . 54
VIII. Varicella . . . 60
IX. Erysipelas . . . 61
X. Diphtheria . . . 65
XI. Influenza . . . 74
XII. Dysentery . . . 77
XIII. Cholera . . . 81
XIV. Malarial Diseases . . . 90
Intermittent Fever . . . 92
Pernicious Intermittent Fever . . . 94
Remittent and Continuous Forms of Malarial Fever . . . 95
Chronic Malarial Cachexia . . . 95
Masked Intermittent Fever . . . 95
XV. Typho-malarial Fever . . . 98
XVI. Dengue . . . 99
XVII. Yellow Fever . . . 100
XVIII. Epidemic Cerebro-spinal Meningitis . . . 103
XIX. Septic and Pyæmic Diseases . . . 108
XX. Hydrophobia (Rabies canina) . . . 113
XXI. Glanders (Farcy) . . . 116
XXII. Malignant Pustule (Anthrax. Mycosis intestinalis) . . . 117
XXIII. Trichinosis . . . 121

Diseases of the Respiratory Organs.

SECTION I.

DISEASES OF THE NOSE.

I. Coryza . . . 125
II. Chronic Rhinitis . . . 126
III. Nose-bleed . . . 129

SECTION II.

DISEASES OF THE LARYNX.

CHAPTER PAGE
I. Acute Laryngeal Catarrh (Acute Laryngitis) 130
II. Chronic Laryngitis (Chronic Laryngeal Catarrh) 132
III. Laryngeal Perichondritis 134
IV. Œdema of the Glottis 135
V. Tuberculosis of the Larynx (Consumption of the Larynx) 136
VI. Paralyses of the Laryngeal Muscles 139
Paralyses in the Distribution of the Superior Laryngeal Nerve 139
Paralyses in the Distribution of the Inferior Laryngeal or Recurrent Nerve . 139
VII. Spasm of the Glottis 142
VIII. Disturbances of Sensibility in the Larynx 143
IX. New Growths in the Larynx 144
Benignant New Growths in the Larynx 144
Malignant New Growths. Carcinoma of the Larynx 145

SECTION III.

DISEASES OF THE TRACHEA AND THE BRONCHI.

I. Acute Catarrh of the Trachea and the Bronchi 146
The Milder Forms of Acute Bronchitis 148
Severe Febrile Acute Bronchitis 148
Catarrh of the Finer Bronchi. Capillary Bronchitis 148
II. Chronic Bronchitis 151
III. Fœtid Bronchitis (Putrid Bronchitis) 155
IV. Croupous Bronchitis (Fibrinous Bronchitis) 158
V. Whooping Cough (Pertussis) 160
VI. Bronchiectasis 164
VII. Stenosis of the Trachea and Bronchi 166
Tracheal Stenosis 166
Bronchial Stenosis 167
VIII. Bronchial Asthma 168

SECTION IV.

DISEASES OF THE LUNGS.

I. Pulmonary Emphysema 173
Clinical History and Symptoms 176
Other Pulmonary Symptoms and Phenomena in other Organs 178
II. Pulmonary Atelectasis (Compression of the Lungs. Aplasia of the Lungs) . 181
III. Pulmonary Œdema 183
IV. Catarrhal Pneumonia (Broncho-pneumonia. Lobular Pneumonia) . . . 184
V. Croupous Pneumonia 189
Description of Single Symptoms and Complications 193
Special Peculiarities and Anomalies in the Course of Pneumonia . . . 200
VI. Tuberculosis of the Lungs (Pulmonary Phthisis. Pulmonary Consumption) . 207
General Pathology and Ætiology of Tuberculosis 207
Ætiology of Tuberculosis in Man 208
Pathological Anatomy of Tuberculosis, especially of Pulmonary Tuberculosis . 212
Clinical History of Tuberculosis in General, and of Pulmonary Tuberculosis in Particular 215
Special Symptoms and Complications 218
Contraction of the Lungs (Fibroid Phthisis) 223
Disseminated Pulmonary Tuberculosis 224

CHAPTER PAGE
VII. Acute General Miliary Tuberculosis . . . 236
VIII. Gangrene of the Lungs . . . 241
IX. Diseases from the Inhalation of Dust (Pneumonoconiosis) . . . 245
X. Embolic Processes in the Lungs (Hæmorrhagic Infarction of the Lungs) . . . 247
XI. Brown Induration of the Lungs (Lungs of Heart Disease) . . . 249
XII. Tumors of the Lungs. Cancer of the Lungs. Echinococcus in the Lungs. Pulmonary Syphilis . . . 250

SECTION V.

DISEASES OF THE PLEURA.

I. Pleurisy . . . 253
Physical Signs . . . 257
Different Forms of Pleurisy . . . 261
II. Peripleuritis . . . 267
III. Pneumothorax . . . 267
IV. Hydrothorax. Hæmatothorax . . . 271
V. New Growths of the Pleura . . . 272
VI. Mediastinal Tumors . . . 273
VII. Actinomycosis of the Thoracic Cavity . . . 274

Diseases of the Circulatory Organs.

SECTION I.

DISEASES OF THE HEART.

I. Acute Endocarditis (Endocarditis verrucosa and ulcerosa) . . . 276
II. Valvulvar Disease of the Heart . . . 280
General Pathology of Valvular Disease of the Heart . . . 281
Insufficiency of the Mitral Valve . . . 283
Stenosis of the Mitral Orifice (Mitral Stenosis) . . . 285
Insufficiency of the Semilunar Valves of the Aorta . . . 287
Stenosis of the Aortic Orifice . . . 290
Insufficiency of the Tricuspid Valve . . . 291
Stenosis of the Tricuspid Orifice . . . 292
Insufficiency of the Pulmonary Valve . . . 293
Stenosis of the Pulmonary Orifice (Pulmonary Stenosis) and the other Congenital Lesions of the Heart . . . 293
Combined Valvular Diseases of the Heart . . . 294
General Comparison of the most Important Physical Signs in Valvular Disease of the Heart . . . 295
General Sequelæ and Complications of Valvular Disease of the Heart . . . 295
General Course and Prognosis of Valvular Disease of the Heart . . . 301
Treatment of Valvular Heart Disease . . . 303
III. Myocarditis (Indurated Degeneration. Myodegeneration) . . . 307
IV. Idiopathic Hypertrophy and Dilatation of the Heart (Overexertion of the Heart. Weakened Heart) . . . 312
V. Fatty Heart . . . 315
Appendix. Remarks on the So-called Mechanical Treatment of Circulatory Disturbances . . . 316
VI. Neuroses of the Heart . . . 318
Angina Pectoris (Stenocardia) . . . 318
Nervous Palpitation . . . 319
Tachycardia . . . 320

SECTION II.

DISEASES OF THE PERICARDIUM.

CHAPTER PAGE
I. Pericarditis 321
Pericarditis externa and Mediastino-pericarditis (Pleuro-pericarditis) . . 325
Obliteration of the Pericardial Cavity (Adhesive Pericarditis) 326
Tubercular Pericarditis 327
II. Hydro-pericardium, Hæmo-pericardium, and Pneumo-pericardium . . . 329
Hydro-pericardium (Dropsy of the Pericardium) 329
Hæmo-pericardium (Blood in the Pericardial Sac) 330
Pneumo-pericardium (Air in the Pericardial Sac) 330

SECTION III.

DISEASES OF THE VESSELS.

I. Arterio-sclerosis (Endarteritis chronica deformans. Atheroma of the Vessels) 331
II. Aneurism of the Thoracic Aorta 334
III. Aneurisms of the Other Vessels 339
IV. Rupture of the Aorta 339
V. Narrowing of the Aorta 340

Diseases of the Digestive Organs.

SECTION I.

DISEASES OF THE MOUTH, TONGUE, AND SALIVARY GLANDS.

I. Stomatitis (Inflammation of the Mouth) 341
II. Ulcerative Stomatitis (Stomacace) 342
III. Aphthæ (Aphthous Stomatitis) 343
IV. Thrush (Soor. Muguet) 344
V. Glossitis 345
VI. Noma (Water-cancer. Cancrum oris) 347
VII. Parotitis (Mumps) 348
Idiopathic, Primary Parotitis 348
Secondary, Metastatic Parotitis 349
VIII. Angina Ludovici 349
IX. Anomalies of Dentition 350

SECTION II.

DISEASES OF THE SOFT PALATE, TONSILS, PHARYNX, AND NASO-PHARYNX.

I. Sore Throat (Tonsillitis. Angina) 351
Catarrhal Sore Throat 352
Follicular Tonsillitis 353
Tonsillar Abscess (Parenchymatous Sore Throat) 353
Necrotic Tonsillitis (Necrotic Sore Throat) 354
II. Chronic Hypertrophy of the Tonsils 356
III. Chronic Pharyngitis 357
Chronic Catarrh of the Naso-pharynx 357
Pharyngitis Sicca 358
Hypertrophic Catarrh of the Pharynx and Naso-pharynx 358
IV. Retropharyngeal Abscess 360

SECTION III.

DISEASES OF THE ŒSOPHAGUS.

CHAPTER PAGE
I. Inflammation and Ulcer of the Œsophagus 361
II. Dilatation of the Œsophagus 363
Diffuse Dilatation 363
Diverticula 363
III. Stenosis of the Œsophagus 366
IV. Cancer of the Œsophagus 369
V. Rupture of the Œsophagus 371
VI. Neuroses of the Œsophagus 371
Spasm of the Œsophagus 371
Paralysis of the Œsophagus 371

SECTION IV.

DISEASES OF THE STOMACH.

I. Acute Gastric Catarrh 372
II. Chronic Gastric Catarrh 374
Appendix. Hyperacidity and Hypersecretion of the Gastric Juice . . . 382
III. Phlegmonous Gastritis 383
IV. Gastric Ulcer (Simple or Round Ulcer of the Stomach) 384
Appendix. Melæna Neonatorum 390
V. Cancer of the Stomach 391
VI. Dilatation of the Stomach 396
VII. Nervous Disorders of the Stomach 400

SECTION V.

DISEASES OF THE INTESTINES.

I. Intestinal Catarrh (Catarrhal Enteritis) 403
Different Forms of Intestinal Catarrh 406
II. Cholera Morbus (Cholera Nostras, Cholera Infantum) 410
III. Intestinal Catarrh of Children (Pedatrophy) 413
IV. Typhlitis and Perityphlitis (Inflammation of the Cæcum) 418
V. Perforating Ulcer of the Duodenum 423
VI. Tuberculosis of the Intestines 423
VII. Syphilis of the Rectum 425
VIII. Cancer of the Intestines 426
IX. Hæmorrhoids 428
X. Habitual Constipation 430
XI. Stricture and Obstructions of the Intestines 432
XII. Intestinal Parasites 440
Tape-worms (Tænia and Bothriocephalus) 440
Round-worms (Ascaris lumbricoides) 445
Oxyuris vermicularis (Seat-worms) 446
Anchylostomum duodenale (Dochmius s. Strongylus duodenalis) . . . 448
Trichocephalus dispar (Whip-worm) 448

SECTION VI.

DISEASES OF THE PERITONEUM.

I. Acute Peritonitis 449
II. Chronic Peritonitis. Tubercular Peritonitis 457

CHAPTER PAGE
III. Ascites (Hydroperitoneum) 460
IV. Cancer of the Peritoneum 463

SECTION VII.

DISEASES OF THE LIVER, BILE-DUCTS, AND PORTAL VEIN.

I. Catarrhal Jaundice (Icterus catarrhalis. Gastro-duodenal Catarrh with Jaundice) 464
Appendix. Acute Febrile Jaundice. Weil's Disease 469
II. Biliary Calculi (Hepatic Colic. Cholelithiasis) 470
III. Suppurative Hepatitis (Hepatic Abscess) 476
IV. Cirrhosis of the Liver (Chronic Diffuse Interstitial Hepatitis. Laennec's Cirrhosis. Gin-drinkers' Liver) 478
V. Biliary and Hypertrophic Cirrhosis of the Liver 483
VI. Acute Yellow Atrophy of the Liver 485
Appendix. Pernicious Jaundice. Cholæmia and Acholia 489
VII. Icterus Neonatorum (Jaundice of the Newborn) 490
VIII. Syphilis of the Liver 490
IX. Cancer of the Liver and Bile-ducts 492
X. Echinococcus of the Liver 494
XI. Circulatory Disturbances in the Liver 496
XII. Atrophy, Hypertrophy, and Degenerations of the Liver 498
XIII. Anomalies in the Shape and Position of the Liver 499
XIV. Suppurative Pylephlebitis (Purulent Inflammation of the Portal Vein and its Branches) 500
XV. Thrombosis of the Portal Vein (Chronic Adhesive Pylephlebitis. Pylethrombosis) 502
Appendix. Diseases of the Pancreas 503

Diseases of the Nervous System.

I. The Diseases of the Peripheral Nerves.

SECTION I.

DISEASES OF THE SENSORY NERVES.

I. General Remarks upon the Disturbances of Sensibility 505
The Different Varieties of Cutaneous Sensibility and the Methods of testing them 505
The Sensibility of the Muscles and Joints 509
II. Anæsthesia of the Skin 510
Anæsthesia of the Trigeminus 513
III. Neuralgia in General 515
IV. The Individual Forms of Neuralgia 521
1. Neuralgia of the Trigeminus 521
2. Occipital Neuralgia 523
3. Neuralgias in the Region of the Brachial Plexus 524
4. Intercostal Neuralgia 524
5. Neuralgias in the Region of the Lumbar Plexus 526
6. Sciatica 526
7. Neuralgia of the Genitals and the Rectal Region 528
V. Neuralgia of the Joints 528
VI. Habitual Headache 530
VII. Anomalies of the Sense of Smell 532
VIII. Anomalies of the Sense of Taste 533

SECTION II.

DISEASES OF THE MOTOR NERVES.

CHAPTER PAGE
I. General Remarks upon the Disturbances of Motility 534
1. Paralysis 534
2. Symptoms of Motor Irritation 539
3. Ataxia 542
4. General Remarks upon testing the Reflexes and the Condition of them . 543
Mechanical Muscular Irritability and Paradoxical Contraction 546
5. General Remarks upon the Changes of Electrical Excitability in the Motor Nerves and Muscles 546
II. The Different Forms of Peripheral Paralysis 555
1. Paralysis of the Ocular Muscles 555
2. Paralysis of the Motor Branch of the Trigeminus 557
3. Facial Paralysis 558
4. Paralyses in the Region of the Muscles of the Shoulder 562
5. Paralyses of the Muscles of the Back 564
6. Paralyses in the Region of the Upper Extremity 564
Radial (Musculo-spiral) Paralysis 564
Ulnar Paralysis 566
Median Paralysis 566
Combined Paralyses of the Arm 567
7. Paralysis of the Diaphragm 568
8. Paralyses in the Region of the Lower Extremity 568
9. Toxic Paralyses 569
Lead Paralysis 569
Arsenical Paralysis 571
III. The Different Forms of Localized Spasms 571
1. Spasms in the Motor Distribution of the Trigeminus 571
2. Clonic Facial Spasm 572
3. Spasm in the Region of the Hypoglossal Nerve. Lingual Spasm . . . 573
4. Spasms in the Muscles of the Neck 573
5. Spasms in the Muscles of the Shoulder and Arm 575
6. Spasms in the Muscles of the Lower Extremity 575
Saltatory Reflex Spasm 575
Arthrogryposis 576
Paramyoclonus Multiplex 576
Electrical Chorea 576
7. Spasms in the Respiratory Muscles 576
IV. Writer's Cramp and Allied Professional Neuroses 577
V. Simple and Multiple Degenerative Neuritis 579
Clinical History of the Different Forms of Neuritis 581
1. Secondary Neuritis 581
2. Primary Simple Neuritis 582
3. Primary Multiple Degenerative Neuritis 582
4. The Chronic Neuritis of Alcoholic Subjects 584
VI. New Growths in the Peripheral Nerves 585

II. Vasomotor and Trophic Neuroses.

I. Preliminary Remarks upon Vasomotor, Trophic, and Secretory Disturbances . 587
Acute Angioneurotic Œdema 588
Myxœdema 589
Acromegaly 589
Hydrops Articulorum Intermittens 590
II. Hemicrania 591

CHAPTER PAGE
III. Progressive Facial Hemiatrophy . . . 594
IV. Exophthalmic Goître . . . 595

III. The Diseases of the Spinal Cord.

I. Diseases of the Spinal Meninges . . . 599
1. Acute Inflammations of the Spinal Meninges . . . 599
2. Chronic Spinal Leptomeningitis . . . 601
3. Pachymeningitis cervicalis hypertrophica . . . 602
4. Hæmorrhages of the Spinal Meninges . . . 603
II. Disturbances of Circulation, Hæmorrhages, Functional Disturbances, and Traumatic Lesions of the Spinal Cord . . . 604
1. Disturbances of Circulation . . . 604
2. Spinal Apoplexy. Hæmatomyelia . . . 604
3. Functional Disturbances . . . 605
4. Traumatic Lesions . . . 606
5. Diseases of the Spinal Cord after a Sudden Reduction of the Atmospheric Pressure (Caisson Disease) . . . 608
III. The Pressure Paralyses of the Spinal Cord . . . 608
IV. Acute and Chronic Myelitis . . . 615
Localization of the Functions of the Segments of the Spinal Cord . . . 624
V. Multiple Sclerosis of the Brain and Spinal Cord . . . 627
VI. Tabes Dorsalis . . . 632
Appendix. Hereditary Ataxia. Friedreich's Form of Locomotor Ataxia . 648
Ataxic Paraplegia . . . 649
VII. Amyotrophic Lateral Sclerosis . . . 650
VIII. Progressive (Spinal) Muscular Atrophy . . . 653
Appendix. The Primary Myopathic Forms of Muscular Atrophy . . . 658
IX. The So-called Spastic Spinal Paralysis . . . 663
X. Acute and Chronic Poliomyelitis . . . 667
1. Spinal Paralysis of Children . . . 667
2. Acute Poliomyelitis of Adults . . . 671
3. Subacute and Chronic Poliomyelitis . . . 673
XI. Acute Ascending Spinal Paralysis . . . 674
XII. New Growths of the Spinal Cord and of its Membranes . . . 676
XIII. The Formation of Cavities and Fissures in the Spinal Cord . . . 677
Morvan's Disease . . . 679
Appendix. Spina Bifida . . . 679
XIV. Secondary Degenerations in the Spinal Cord . . . 680
XV. Unilateral Lesion of the Spinal Cord . . . 682

IV. The Diseases of the Medulla Oblongata.

I. Progressive Bulbar Paralysis . . . 685
Appendix. The Rarer Forms of Chronic Bulbar Paralysis, and Progressive Ophthalmoplegia . . . 690
II. Acute and Apoplectiform Bulbar Paralysis . . . 691
1. Hæmorrhage into the Medulla Oblongata and the Pons . . . 691
2. Embolism and Thrombosis of the Basilar Artery . . . 693
3. Acute or Inflammatory Bulbar Paralysis . . . 694
III. Compression of the Medulla . . . 695

V. *THE DISEASES OF THE BRAIN.*

SECTION I.

DISEASES OF THE CEREBRAL MENINGES.

CHAPTER PAGE
I. Hæmatoma of the Dura Mater 696
II. Purulent Meningitis 698
III. Tubercular Meningitis 702
Tubercular Meningitis in Children 705
IV. Thrombosis of the Cerebral Sinuses 707

SECTION II.

DISEASES OF THE BRAIN-SUBSTANCE.

I. Disturbances of Circulation in the Brain 708
II. General Preliminary Remarks upon the Localization of Cerebral Diseases (Topical Diagnosis of Cerebral Lesions) 710
1. The Motor Region of the Cortex Cerebri 711
2. The other Parts of the Cortex Cerebri, except the Center for Speech . . 715
3. The Centers of Speech and the Disturbances of Speech (Aphasia and Allied Conditions) 716
4. The Centrum Ovale, Internal Capsule, Central Ganglia, and Region of the Corpora Quadrigemina 720
5. The Cerebellum 723
General Diagnostic Principles 724
III. Cerebral Hæmorrhage 725
IV. Cerebral Embolism and Thrombosis 737
V. Inflammation of the Brain 741
1. Abscess of the Brain (Suppurative Encephalitis) 741
2. Acute and Chronic Non-suppurative Encephalitis 743
Idiopathic Softening of the Brain 744
Curable Form of Encephalitis 744
Diffuse Cerebral Sclerosis 744
The Acute Encephalitis of Children (Cerebral Paralysis of Children) . . . 744
VI. Insolation. Sunstroke. Heat Prostration. Thermic Fever 746
VII. Tumors of the Brain 748
Varieties of Cerebral Tumor 748
The General Symptoms of Cerebral Tumors 749
Tumors in the Different Parts of the Brain. Their Focal Symptoms . . . 751
General Course of Cerebral Tumors 754
APPENDIX. Hydatids of the Brain 756
VIII. Cerebral Syphilis 757
IX. Progressive General Paralysis of the Insane (Paralytic Dementia) . . . 760
X. Chronic Hydrocephalus 767
XI. Ménière's Disease 769

VI. *NEUROSES WITHOUT KNOWN ANATOMICAL BASIS.*

I. Epilepsy 770
APPENDIX. Infantile Convulsions 779
II. Chorea 780
III. Paralysis Agitans 784
IV. Athetosis 787
V. Tetany 789
VI. Tetanus 791

CHAPTER PAGE
VII. Congenital Myotonia (Thomsen's Disease) 795
VIII. Catalepsy 796
IX. Hysteria 797
X. Neurasthenia 815
XI. The Traumatic Neuroses 819

Diseases of the Kidneys, the Pelvis of the Kidney, and the Bladder.

SECTION I.

DISEASES OF THE KIDNEYS.

I. General Preliminary Remarks upon the Pathology of Renal Disease . . . 822
  1. Albuminuria 823
  2. Casts and other Abnormal Morphological Constituents of the Urine in Renal Disease 826
  3. The Dropsy of Renal Disease 828
  4. Uræmia 829
  5. The Changes in the Circulatory Apparatus in Renal Disease . . . 834
II. Acute Nephritis (Acute Bright's Disease) 836
III. The Subchronic and Chronic Forms of Nephritis, with the Exception of the Genuine Contracted Kidney 849
IV. Contracted Kidney 856
V. Amyloid Kidney 864
VI. Purulent Nephritis and Perinephritis 868
  Perinephritic Abscess 870
VII. Disturbances of Circulation in the Kidneys 871
  1. The Congested Kidney 871
  2. Embolic Infarction in the Kidneys 871
VIII. New Growths in the Kidneys 872
IX. Parasites of the Kidneys and of the Urinary Passages. Chyluria . . . 874
X. Movable Kidney (Floating Kidney. Ren Mobilis) 876
  APPENDIX. The Diseases of the Suprarenal Capsules and Addison's Disease (Bronzed Skin) 878

SECTION II.

DISEASES OF THE PELVIS OF THE KIDNEY AND OF THE BLADDER.

I. Inflammation of the Pelvis of the Kidney. Pyelitis 881
II. Nephrolithiasis 884
III. Tuberculosis of the Genito-urinary Apparatus 888
IV. Hydronephrosis 890
V. Cystitis (Vesical Catarrh) 893
VI. New Growths in the Bladder 897
VII. Enuresis Nocturna (Nocturnal Incontinence of Urine) 898

Diseases of the Organs of Locomotion.

I. Acute Articular Rheumatism 900
II. Chronic Articular Rheumatism (Chronic Polyarthritis) and Arthritis Deformans 911
III. Acute and Chronic Muscular Rheumatism 916
  APPENDIX. Acute Polymyositis 919
IV. Rachitis 920
V. Osteomalacia 925

Diseases affecting the Blood and Tissue-metamorphosis.

(CONSTITUTIONAL DISEASES.)

CHAPTER PAGE

I. Anæmia and Chlorosis 928
II. Progressive Pernicious Anæmia 938
III. Leukæmia 945
IV. Pseudo-leukæmia 951
V. Hæmoglobinæmia and Hæmoglobinuria 953
VI. Scurvy 956
VII. Purpura. Morbus Maculosus Werlhofii. Peliosis 961
VIII. Hæmophilia 963
IX. Diabetes Mellitus 965
X. Diabetes Insipidus 982
XI. Gout 984
XII. Obesity 992
XIII. Scrofula 999

APPENDIX I. Summary of the Symptoms and Treatment in Cases of Poisoning 1003
APPENDIX II. Table of Weights and Measures 1011
INDEX 1012

LIST OF ILLUSTRATIONS.

FIG. PAGE

1. Typhoid Bacilli 1
2. Temperatures in typhoid fever 6
3. Example of the temperature curve in relapsing fever 33
4. Spirilli of relapsing fever in the blood 34
5. Example of a normal scarlet-fever curve 38
6. Example of the temperature curve in measles 48
7. Example of the temperature curve in true small-pox 55
8. The cocci of erysipelas 62
9. Comma bacilli 83
10. Quotidian intermittent fever 93
11. Tertian intermittent fever 93
12 A, 12 B. Anthrax bacilli 118
13. Trichinæ 122
14. Paralysis of left vocal cord 140
15. Bilateral paralysis of the posticus 141
16. Paralysis of both internal thyro-arytænoid muscles 141
17. Paralysis of the arytænoideus 142
18. Paralysis of the thyro-arytænoids and arytænoideus 142
19, 20. Pediculated fibromata 144
21. Crystals of fat acids 156
22. Asthma crystals and Curschmann's spirals 169
23. Example of the temperature curve in croupous pneumonia 199
24. Example of the temperature curve in "intermitting" pneumonia 199
25. Cholesterine crystals 203
26. Elastic fibers 219
27. Tubercle bacilli in the sputum 220
28. Masses of actinomyces 274
29. Pulse curve in marked mitral stenosis 285
30. Pulse curve in aortic insufficiency 289
31. Pulse curve in stenosis of the aortic orifice 291
32. Pulsus bigeminus 298
33. Plan of the dentition 350
34. Sarcini ventriculi and yeast-cells 376
35. Hæmine crystals 392
36. Stomach-tube with Hegar's funnel 399
37. Head of tænia solium 440
38. Head of cysticercus of the brain 440
39. Tænia solium 440
40. Eggs of intestinal parasites 441
41. Head of tænia mediocanellata 442
42. Tænia mediocanellata 442
43. Head of bothriocephalus latus 442
44. Bothriocephalus latus 442

FIG. PAGE
45. Embryo of bothriocephalus latus 443
46. Ascaris lumbricoides 445
47, 48. Oxyuris vermicularis 447
49, 50. Anchylostomum duodenale 448
51. Trichocephalus dispar 449
52. Leucine and tyrosine crystals 488
53. Tænia echinococcus 494
54, 55. Echinococcus scolices 495
56. Echinococcus hooklets 495
57, 58. Distribution of the sensory cutaneous nerves in the head 513
59, 60. Distribution of the sensory cutaneous nerves in the trunk and upper extremities 514
61. Detailed distribution of the nerves to the dorsal surface of the fingers . . . 514
62, 63. Distribution of the sensory cutaneous nerves to the lower extremities . . 515
64. Horizontal section through the right cerebral hemisphere 534
65. Transverse section through the crura cerebri in secondary degeneration . . . 535
66. Transverse section through the cervical enlargement 535
67. Transverse section through the lumbar enlargement 535
68. Motor points of face 547
69, 70. Motor points of arm 548, 549
71. Motor points of thigh 550
72, 73. Motor points of leg 551, 552
74. Right facial paralysis 558
75. Trunk of the facial 560
76. Paralysis of the right serratus 563
77. Position of the hand in paralysis of the radial nerve 565
78. Claw-shaped hand, *main en griffe* 566
79. Spasm of the right splenius capitis 574
80. Left facial hemiatrophy 594
81. Position of the hand in pachymeningitis cervicalis hypertrophica 602
82. Vertebral displacement in spondylitis 609
83. Relations of the vertebræ to the spinal segments 622
84. Areas of anæsthesia at various levels of the spinal cord 623
85. Example of disease of the cord in multiple sclerosis 628
86. Distribution of the sclerosed nodules on the surface of the pons 628
87. Transverse section through the lumbar region in locomotor ataxia 634
88. Transverse section through the cervical region in locomotor ataxia 634
89, 90. Transverse section of cord in beginning locomotor ataxia 634
91. Tabetic arthropathy of the right knee and left ankle 644
92. Positions of a child with pseudo-hypertrophic paralysis on rising 659
93. Pseudo-hypertrophy of the muscles 660
94. Juvenile myopathic muscular atrophy 662
95. Section of the cord in anterior poliomyelitis 667
96. Secondary descending degeneration of the pyramidal tracts 681
97. Secondary ascending and descending degeneration 682
98. Course of the main tracts in the cord 683
99. Representation of the chief symptoms in unilateral lesion 684
100. Diagram of focal diseases in the pons 692
101, 102. Lateral aspect of the brain 712, 713
103. Aspect of the median surface of the cerebrum 713
104. Topographical relations between the surface of the brain and the skull . . . 714
105. Diagram of the course of the optic fibers in the chiasma 716
106. Examples of handwriting in general paralysis 763
107. Characteristic position of the body in paralysis agitans 785
108. Example of the position of the fingers in the movements of athetosis . . . 788
109. Hysterical contracture 804
110. Hysterical *arc de cercle* 807

FIG. PAGE
111. Different forms of casts 826
112. Acute nephritis (scarlatinal, early stage) 837
113. Distoma hæmatobium 875
114. Embryos of filaria 876
115. Pelvic renal epithelium 883
116. Crystals of triple phosphate and ammonic urate 895
117. Deformity of the hand in protracted arthritis deformans 914
118. Changes in the red blood-corpuscles in pernicious anæmia 943
119. Anæmic blood 948

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ACUTE GENERAL INFECTIOUS DISEASES.

CHAPTER I.

TYPHOID FEVER.

(*Typhus abdominalis. Enteric Fever. Ileotyphus.*)

Ætiology.—According to our present views, the cause of typhoid fever must be sought in some specific, organized, pathogenic poison. The later investigations in bacteriology have apparently revealed what this poison is. Koch and Eberth were the first to point out a clearly specific variety of short, rod-shaped bacteria (bacilli), which appear in this disease alone. They take up the aniline colors. Koch and Eberth, and later W. Meyer, Friedländer, and Gaffky, found them in the intestine, especially in its lymphatic apparatus, and also in the mesenteric glands, the spleen, liver, and kidneys. The subjects in whom these bacteria were detected had died in the beginning or during the fastigium of typhoid fever.

The length of these bacilli (see Fig. 1) is about one third the diameter of a red blood-globule, and their breadth equals one third their length. Their ends are rounded off, and in their interior the formation of spores can sometimes be plainly recognized. They are found for the most part lying together in little clumps (foci of bacilli) in the organs. They have also been demonstrated in the dejections of typhoid patients, and sometimes in the blood taken from rose-spots.

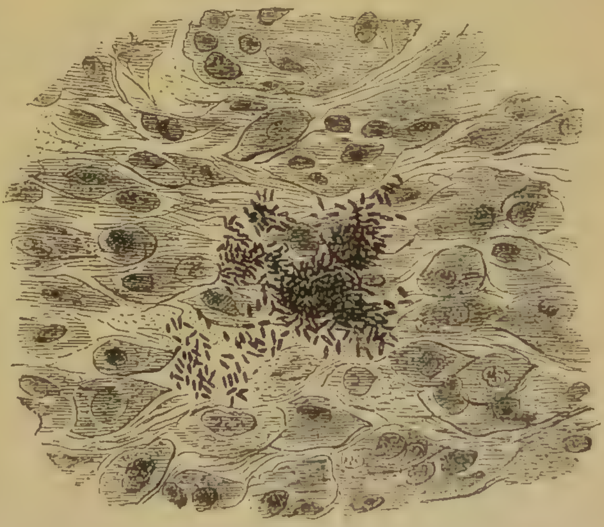

FIG. 1.—Typhoid bacilli. Section of the spleen, × 800. (After FLÜGGE.)

That these typhoid bacilli are specific, is shown, however, as in the case of many micro-organisms, less by their external form than by their peculiarities, as observed in pure cultures of them. Gaffky, who first succeeded with such cultivations, found that the colonies of these bacilli, reared in a mass of stiff gelatin, are made up of very minute, brownish-yellow clumps, and that in their growth they are always limited to the spots where they have been implanted, and never liquefy the jelly in which they grow. The growth of typhoid bacilli upon the cut surface of boiled potatoes is also very characteristic. The bacilli cover the entire surface with a very thin though tough pellicle scarcely recognizable to the naked eye. Examined in water, the typhoid bacilli exhibit quite an active individual motion. The formation of spores takes place only when the temperature is between 86° and 108° (30°–42° C.), ceasing at lower temperatures.

Numerous attempts have been made to produce typhoid fever artificially by introducing pure cultures of the typhoid bacilli into the bodies of animals, but the results of these efforts have not yet proved perfectly harmonious. The main cause of the discrepancy is probably that animals are in general very slightly susceptible to the disease. At any rate, the attempts at artificial infection up to this date have proved successful only in cases when the animals subjected to the experiment (rabbits, guinea-pigs) have received large amounts of the typhoid bacilli directly into a vein or into the abdominal cavity (E. Fränkel, Simmonds), or when the bacilli have been introduced into the duodenum (A. Fränkel). Probably, however, we have here to do rather with the intoxication of the animals caused by the poisonous matters generated in the cultures of bacilli than with an actual infection, for the pathological changes of typhoid fever are but little developed in the animals, and the injected bacilli themselves appear to be for the most part destroyed within the body of the animal experimented upon (Flügge and Sirotinin, and others). Attempts to produce the disease by mixing the dejecta of typhoid patients with the animal's food have thus far proved invariably unsuccessful. Probably the bacilli are immediately destroyed by the hydrochloric acid in the stomach.

Investigation of the ætiology of typhoid fever must consequently be directed to ascertaining in what manner and through what channels the specific typhoid bacilli penetrate* into the human body, and what circumstances are then essential to their further development and to the display of their pathogenic properties. It must be confessed that the ability to answer these questions accurately is a goal from which we are quite distant.

It is almost universally believed that, as a rule, typhoid bacilli do not have any permanent, independent existence outside the human body. Often, however, the conditions essential to an abundant development of the bacilli arise in certain places, and thus make it possible for a greater or less number of persons to absorb the pathogénetic poison, and, as a result, to be attacked by typhoid fever. In this way occur the numerous greater or smaller epidemics of typhoid fever in contrast to the sporadic cases, which are likewise possible, and are not infrequent. If an epidemic of typhoid appears in a place till then entirely free from the disease, we must always refer it to an importation of the disease-germs, and seek their source in some previous case of typhoid. We must, therefore, take for granted that the poison of typhoid can in some way escape from the body of the patient into the outer world. If we believe this, we shall be sure to think, first of all, of the intestinal discharges as the source of infection. These discharges, as already stated, are known to contain the typhoid bacilli or their spores.

As to the exact manner of infection, views are still widely different. Up to the present time there are chiefly two contrasted theories, called, respectively, the "ground-soil" and the "drinking-water"† theories. According to the former, which is maintained principally by Pettenkofer and his pupils, the ground-soil is to be regarded as the chief place of development for the schizomycetic fungus of typhoid fever. Whether this will flourish depends chiefly on the condition of the soil (varying at different times and in different places), and this alone should explain all the peculiarities observable in the spread of the disease—e. g., that single houses, streets, or wards of a city should suffer. According to Pettenkofer, a soil that air and water easily penetrate—e. g., one made up of alluvial or detrital deposits—is most favorable for the spread of the disease, while a firm, rocky bottom makes its further development impossible; and, where this "tendency of the ground-soil" is wanting, the disease can neither be introduced nor, if brought in, spread any further; for, according to Pettenkofer, the typhoid poison is seldom if ever transferred directly from one person to another. The poison in the stools must first be changed by the soil before it becomes infectious. The "ground-air," which is continually rising, carries the poison not only into the open atmosphere, but into the air of dwelling-rooms, and, being then inhaled, produces infection. We can thus understand why Pettenkofer regards typhoid fever as not directly contagious. The chief support of the ground-soil theory, beyond the results of comparing the character of the soil with the extent of the epidemics, consists in the proof which Buhl and Pettenkofer have given (taking Munich as an example) that a relation exists between the variations of the standing water in the soil and the frequency of typhoid cases. It appears that, when the water stands high (near the surface), fewer cases occur, and when it falls below the mean height cases are more numerous. This relation, which is said to hold true also for Berlin and some other places, is not yet, we may add, explained with certainty.

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* Perhaps it is not useless once more to call attention expressly to the fact that typhoid fever can result only from an infection of the body with actual typhoid bacilli, and never through any other bacteria, through the products of decay and decomposition, tainted food, and the like; nor does there yet exist the slightest proof that typhoid bacilli can be developed from any other micro-organisms.

† Compare with what follows the statements concerning the ætiology of cholera, where the same disputed points are considered.

To be contrasted, or rather compared, with the soil-gas theory is the view held by many physicians, despite the vigorous protest of Pettenkofer, that drinking-water plays an important *rôle* in the origin of many epidemics of typhoid. In fact, in the case of numerous epidemics, whose extent bears an unmistakable relation to the water-supply, we seem perfectly justified in supposing that the typhoid germs are brought into the body by means of water used in drinking or otherwise. Even then we are by no means wholly to disregard the character of the soil, for the disease-producing poison—not to speak of direct pollution—is probably often communicated to the well-water from the soil. The possibility of this will be especially great if the wells are near drains or cess-pools containing typhoid discharges. In epidemics spread by drinking-water, the typhoid bacilli have lately been repeatedly found in the suspected water.

We believe the idea is continually gaining ground that no single "theory" can fully explain all the facts, and that the possibility of infection occurring in several different ways must be considered. Beside the possible inhalation of the poison, or the ingestion of polluted water, it may be that sometimes the disease is conveyed by food. For example, it has been remarked in England, and lately in Cologne, that the fever in certain epidemics was limited to individuals who had their milk from one common source. In such cases, however, the probable cause is not a disease in the cows, but a pollution of the milk or the milk-cans by water. It is as yet doubtful if animals can have typhoid fever; at any rate, all attempts at artificial inoculation have had a negative result. This fact makes it uncertain whether the illnesses which have been observed to follow the ingestion of the flesh of diseased calves (e. g., the epidemic of Kloten) are actually to be considered typhoid fever, although the pathological changes are said by Huguenin to be very similar to those found in typhoid. Finally, it seems very probable that persons who come into direct contact with typhoid discharges are thereby exposed to the danger of infection. Many deny this (*vide supra*), but it would explain why nurses and laundresses, who have to handle clothing soiled by the discharges of patients, are comparatively often attacked by typhoid fever.

Through the agency of dirty linen, utensils, etc., the poison may be spread even further.

[It is not probable that sewer-gas in itself is an exciting cause of typhoid fever. Especially in large cities typhoid dejections are constantly finding their way into the sewers, which afford all the conditions favorable to the further growth and development of the poison. If, then, the drainage of any house is defective, the seeds of the disease can readily gain access to the interior of the house and infect susceptible individuals.

One of the most instructive epidemics on record is that in Plymouth, Pennsylvania, a town of eight thousand inhabitants. In the spring of 1885 a disease, at first supposed to be of a strange character, broke out in the place, and, before it ceased, affected twelve hundred persons, causing one hundred and thirty deaths. It was soon found that the malady was typhoid fever, which arose from one case, briefly in this wise: In January, February, and March there was a case of typhoid in a house on a hill sloping toward a water-supply of the town. The dejections were thrown out on the snow, under which the ground was deeply frozen. On March 25th a sudden and great thaw occurred, the water did not sink into the ground, but ran immediately into the natural surface channels, and on April 10th the epidemic began. There were reasons, which it is not necessary here to detail, why the above source of water-supply was drawn upon to an unusual degree just at that time, but it has been shown that those who derived their water from other sources were spared by the disease. The original case came from Philadelphia.]

In almost all cases the intestine seems to be the actual gate of entrance for the typhoid poison into the human system. This is shown by the fact that in all cases which come to autopsy in early stages of the disease, the typhoid bacilli are mainly confined to the lymphatic tissues of the intestine. The typhoid poison (bacilli or spores) is probably swallowed, either directly with water or polluted food, or after being inhaled or in some other way introduced into the mouth. If not destroyed in the stomach, it passes on in viable condition into the alkaline contents of the intestine, and here finds the conditions essential to its further development. It penetrates at first into the follicles and Peyer's patches, and thence goes on into the mesenteric glands, the blood-current, the spleen, and other organs.

As in the case of most other infectious diseases, the occurrence of infection is dependent not only on outward conditions, but also on an individual predisposition. Details of the circumstances attending this latter are as yet not at all accurately understood. Even in the worst typhoid centers, where the possibility of infection must be universal, many escape the disease.

Age has an indubitable influence upon the liability to the disease. Typhoid is especially a disease of youthful, vigorous individuals, of fifteen to thirty years. Above that age it is noticeably less frequent, although cases do occur at sixty and even seventy years. Formerly it was often said that young children were never attacked; but this was because the disease was not recognized, for in reality it is only children under one year old who seem to be seldom infected. At a later age, cases are by no means rare.

Sex can not be shown with certainty to have an especial predisposing influence upon the frequency of typhoid fever.

Mental excitement and gross errors in diet seem to predispose to the disease. On the other hand, a certain immunity has been alleged to be given by many circumstances, especially pregnancy, the puerperal state, and other diseases already existing (tuberculosis, heart disease). Most of these statements are shown, however, by more extended experience, to be very doubtful. It does seem to be certain that the occurrence of typhoid fever gives very probable though not absolute immunity against any later new attack.

Finally, it must be mentioned that the necessary conditions for an abundant development and conveyance of the typhoid germs are beyond doubt dependent on the season. According to statistics, most of the typhoid epidemics come in the months from August to November, while generally the number of cases greatly diminishes from December to spring.

General Course of the Disease.—Extended experience shows that, after infection with the typhoid poison has taken place, a certain time must elapse before the symptoms of the disease appear. The length of this time, the "stage of incubation," is, unlike that of many other infectious diseases, not perfectly definite. On the average, it lasts two to three weeks, sometimes less time, sometimes longer. During this period the patient either feels perfectly well, or has certain slight symptoms, to which he pays more or less attention, according to his individual susceptibility. These prodromata consist of languor, disinclination to exertion, anorexia, slight headache, pain in the limbs, etc. Often they last only a few days. Not infrequently the patients state afterward that they had felt the disease coming on for weeks.

The transition of the prodromata into the regular disease takes place sometimes so gradually that it is utterly impossible to take any one day as the first of the illness, in order to reckon from it its duration. It is usually, however, the first symptoms of a high temperature, chilliness, feverishness, and the accompanying increase in general discomfort, which allow one to fix, with at least some accuracy, the beginning of the disease. A decided initial rigor is certainly exceptional.* After the fever begins, most patients soon take to their beds, although it happens often enough that the sick feel either unable or unwilling to give up, and keep on at work for days!

There have been manifold attempts to divide the whole course of the disease into separate periods. The most natural division seems to be into the three stages of development, height or fastigium, and decline (*stadium incrementi*, *s. acmes*, *s. decrementi*). Usually, however, physicians reckon according to the week of the disease. The first week corresponds to the developmental stage, the second, and in all severer cases the third as well, to the fastigium, the fourth (in light cases the third) to the decline. The course of the disease is very variable, however, and naturally there is the greatest diversity in the departures from this general plan.

In the first week, the initial period, the general symptoms augment rapidly. The patients become, in severe cases, very languid and feeble, have generally an intense headache, and complete anorexia, with great thirst. The fever, which is all the time gradually rising, is recognizable subjectively by alternating sensations of heat and cold, and objectively by the hot, dry skin, the parched lips, and the dry and coated tongue. The sleep is disturbed. For the most part there are no prominent thoracic or abdominal symptoms, except that at times there is a sense of oppression in the chest, or some cough. The pulse is quickened, sometimes even now dicrotic. There is often a temporary epistaxis. The belly is not much swollen as a rule, and but little if at all tender. There is generally constipation. Usually the spleen, even at this time, exhibits a swelling that can be easily demonstrated.

Generally the fastigium has begun before the end of the first week. The severe general symptoms persist or even increase. The fever maintains constantly a considerable elevation. The patients become more stupid. Often delirium appears, especially at night. In the lungs there is developed a more or less intense and extensive bronchitis. The abdomen becomes more swollen. On the skin of the trunk appear, generally at the beginning of the second week, a number of small, pale-red spots, roseolæ. Instead of constipation, there is a moderate diarrhœa. There are daily about two to four soft, thin, bright-yellow dejections.

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* According to the representations of many authors, a marked initial rigor seems to occur rather often in some places. In Leipsic, and also in Erlangen, it is very rare.

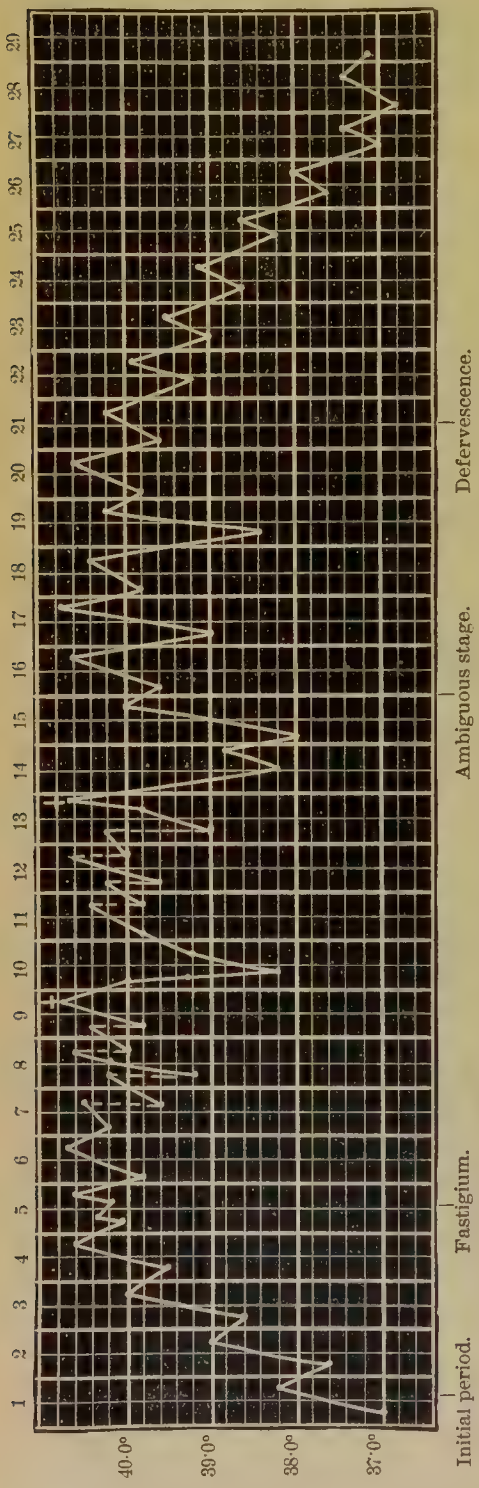

FIG. 2.—*Temperatures in Typhoid Fever.*—The dotted lines represent the remissions in temperature after cool baths, the crosses (+) the exhibition of quinine.

The third week, during which in the severe cases the symptoms already mentioned persist, is the chief time of the numerous complications and of especial clinical events, about which we shall speak below at length. If the disease takes a favorable course, there comes at the end of the third week a decline of the fever; and then the general symptoms also improve as a rule. The mind becomes clearer, the patient sleeps better, and gains some appetite. The pulmonary and digestive symptoms abate, and convalescence gradually begins.

This short sketch of the course of the disease corresponds to most of the cases of medium severity. There are, however, besides these, so many forms and so many variations from the usual picture, that it seems almost impossible to enumerate completely all the events of typhoid fever. And besides, the separate epidemics vary in their general character according to the time and place of their occurrence. In many epidemics the cases run a peculiar course and have certain special complications not seen in others.

We will begin the presentation of the chief peculiarities by speaking of the course of the fever.

Course of the Fever.—Observation of the temperature in typhoid is so absolutely essential for the estimation of each individual case that no scientific physician ought to treat a case without regular measurement of the temperature. The measurements should be taken, if possible, in the rectum. Their frequency must of course be modified by circumstances, but it will probably be possible to have three or four measurements daily. At night, especially if the patients are asleep, it is generally not requisite to take the temperature. A general idea of the course of the fever can be gained only by representing the separate measurements graphically in a continuous "temperature curve."

The typical curve of typhoid fever (see Fig. 2) falls naturally into three or four divisions. The first division is the initial period, or the pyrogenetic stage, and is seldom observed, since at this time the patients are generally not yet under the doctor's care. The initial period of the fever lasts, as a rule, some three or four days, seldom longer; and during this time the temperature rises, generally by gradual steps, so that the morning as well as the evening temperature is each day 2° or 3° (1°–1·5° C.) higher than on the day before. A sudden and considerable rise of temperature, such as occurs in many other diseases, is very rarely seen in the beginning of typhoid fever.

The second division of the curve represents the so-called fastigium, and corresponds to the height of the disease. During this time the fever presents, in most of the severer cases, the general character of "*febris continua*"—i. e., the spontaneous remissions of the fever seldom exceed 2° (1° C.). Almost always the lower temperatures come in the morning hours and the higher in the evening. In cases of average severity the morning remissions touch 102°–103° (39°–39·5° C.), and the evening exacerbations 104°–105° (40°–40·5° C.). Temperatures which reach or exceed 106° (41° C.) are seen only in very severe cases. Considerable morning remissions are always a favorable symptom, while morning temperatures of 104° (40° C.) or higher generally show the case to be severe. The duration of the fastigium varies with the severity and obstinacy of the case. It may last only a few days or one and a half to two weeks; in violent cases still longer.

In many cases of slight or average severity the period of decline follows directly on the fastigium; but in severe cases there frequently intervenes another stage, which Wunderlich has graphically named the "ambiguous" period. The temperature curve becomes irregular and more variable. The morning remissions may be great, even reaching the normal, while the evening temperatures are often still very high. This stage has accordingly been termed the "period of the steep curves." It may be said that in general the longer a case of typhoid lasts the more irregular will be the course of the fever.

The last stage—i. e., in cases of slight or average severity the third stage, and in severe cases commonly the fourth—is the period of defervescence or recovery. The peculiarity of this period in typhoid fever is that the fall of the fever is never by crisis, but always gradually, by lysis. Commonly the temperature descends by degrees, so that on each new day the morning remissions as well as the evening exacerbations are 1° to 2° (0·5°–1° C.) lower. The zigzag form of curve, in which there are of course very frequently slight irregularities, must be taken as the rule. The duration of the defervescence generally exceeds that of the initial period. It lasts five to eight days, often longer. It is not very seldom that in defervescence the morning remissions become from the first very marked, even reaching the normal temperature, while the evening exacerbations become daily less and less, until they too are not above the normal. A third form of decline is much less frequent, in which the morning remissions become every day greater, while the evening temperature persists for some days at about the same height. Several times we have seen the fever take on a tertian type during recovery.

To this outline must be added a number of observations of practical importance.

The initial period does not exhibit especial variations from the course we have stated. Its entire duration is bounded by certain relatively narrow limits.

The fastigium presents, as already mentioned, the greatest varieties in its duration. In light cases it is wholly wanting, so that these consist only of a period of gradually rising fever, and of a gradual defervescence almost immediately consecutive to the rise. The entire duration of such light cases is only one and a half to two weeks. In other and tolerably frequent cases, which are often tedious, but still for the most part are light, the fever is not continuous, but remittent. We have seen in Leipsic, notably in the autumn epidemic of 1878, a number of cases where the fever was even perfectly intermittent during almost the entire illness, and where for two to three weeks afternoon elevations reaching 104° (40° C.) or more daily succeeded normal morning temperatures. These cases had the general course of light attacks.

Various influences, not to speak of therapeutic interference, may produce a considerable temporary remission of temperature in the course of the fastigium. Such a remission sometimes occurs spontaneously from the seventh to tenth day of the disease. If a marked intestinal hæmorrhage occurs (*vide infra*), the temperature generally falls several degrees centigrade, and the less frequent instances of severe epistaxis have the same effect. If, in female patients, abortion or premature delivery occurs, we often observe a similar considerable fall of temperature, even without severe attendant hæmorrhage. Perforation of the intestine often causes the temperature to fall rapidly. At times the occurrence of mental disturbances effects a moderate though noticeable lowering of temperature. Those great and sudden depressions of temperature remain to be mentioned which are accompanied by a very small but exceedingly rapid pulse and general prostration. Every such collapse, if severe, is a most dangerous event, and demands prompt and energetic medical treatment (*vide infra*).

The occurrence of local complications, such as pneumonia or inflammation of the parotid gland, is generally accompanied by a considerable rise of temperature. The fever in such cases often becomes more irregular.

The period of defervescence departs most frequently from its typical behavior by being lengthened out into a "stage of retardation." The morning temperature is then generally normal, while in the evening slight or moderate elevations continue. The reason for this long continuance of the fever may frequently be found in some not yet completely healed local complication, but often no such lesion can be demonstrated. Then we are commonly inclined to surmise sluggish intestinal ulcers which will not heal, or trouble in the mesenteric glands, etc. This sluggish fever may continue for weeks. It is prone to follow severe cases, but lighter attacks, especially in elderly or feeble patients, may also take on this sluggish character at a relatively early period.

Entrance into complete convalescence is shown with far greater certainty by the absence of elevations of temperature than by any other single symptom. There sometimes come, however, temporary elevations of temperature during convalescence, following some error in diet, long-continued constipation, or mental excitement. In other cases the new fever depends on some local sequela, e. g., a boil or a glandular abscess. Often, however, the most accurate investigation fails to demonstrate a cause. Especially in the beginning of convalescence there sometimes comes a high fever, or even a rigor, which may recur several times, but soon gives place to a normal temperature. Generally no certain cause for these brief but decided elevations of temperature can be pointed out. Perhaps we might consider the possibility of some affection of the mesenteric lymph-glands. These sudden and great elevations have seldom any grave significance.

This new fever which we have just described is best termed recurrent fever-attack, in contrast with the proper typhoid relapse. That is, after typhoid fever has ended, the whole process may be repeated; and this occurrence is called a relapse. Particulars as to the behavior of the fever in such cases will be considered below, in connection with all the other peculiarities of typhoid relapses.

PHENOMENA AND COMPLICATIONS RELATING TO THE SEPARATE ORGANS.*

1. **Digestive Organs.**—We think it best to begin our consideration of the more special symptoms with the phenomena referable to the intestinal canal, for the reason that the anatomical changes in the intestine are pathognomonic. Indeed, these alterations may sometimes become of surpassing import in a clinical point of view, although in the majority of cases the intestinal symptoms are clinically not nearly so prominent as the general symptoms that result from the infection of the system as a whole.

The characteristic typhoid lesion of the intestine consists of an affection of Peyer's patches, most marked in the lower part of the ileum. In the first week the patches swell gradually (stage of medullary infiltration). The rest of the mucous membrane exhibits at the same time more or less marked symptoms of simple catarrhal inflammation. In the second week, necrotic crusts form on the surface of the patches, which are cast off in the third week, leaving behind the typhoid ulcers. Toward the end of the third week the ulcers clean up, and then in the fourth week, if the case takes a favorable course, the ulcers heal. Smooth scars are formed, often diffusely pigmented. Experience shows that these scarcely ever lead to stricture of the intestine. The same process also goes on in a greater or less number of the solitary follicles as well as in the Peyer's patches themselves. We may add that probably in lighter cases of typhoid (*vide infra*) there is often no actual ulceration. The swelling of the lymphatic tissue subsides in this case before sloughing occurs. We have already mentioned the occurrence of typhoid bacilli in Peyer's patches and the intestinal follicles.

The number and extent of the ulcers formed have no direct relation whatever to the severity of the case. Although very extensive lesions in the intestine are often found in cases that end fatally, yet, on the other hand, we observe fatal cases in which only a few ulcers are found in the intestine. In cases with extensive intestinal lesions we often see follicular ulcers in the colon as well as in the small intestine (colo-typhoid).

The clinical symptoms referable to the intestinal canal are, as we have said, prominent only in exceptional cases. In the beginning of typhoid fever there is usually constipation. This may last throughout the illness, so that the patients have but one dejection in every two or three days, or often none at all unless an enema be given. As a rule, a moderate diarrhœa begins during the second week. There are two to four stools, or sometimes more, each day. They usually have a characteristic bright-yellow color. On standing, they divide into an upper, cloudy, and quite liquid layer, and a lower layer composed of yellow, crumby masses. They have generally an alkaline reaction, and upon microscopic examination they are found to contain, besides remnants of the ingesta and granular detritus, a few epithelial cells, round cells, many crystals of triple phosphate, and numberless bacteria. Pfeiffer and other investigators have been able frequently, although not invariably, to demonstrate the true typhoid bacilli in the dejecta by means of special methods of cultivation.

Severe diarrhœa (ten to twenty stools daily) is relatively infrequent. In some severe cases we have seen the stools take on a dysenteric character. The autopsy showed in these cases unusually severe lesions of the colon and a diphtheritic inflammation of its mucous membrane.

Gaseous distention affecting the intestine, and especially the colon, is very frequent, but in most cases it is not excessive. Indeed, severe cases of typhoid are observed in which the abdomen always remains concave. Marked tympanites is always an unpleasant complication. We saw one case, which ended fatally, with very great tympanites, in which the lesions were almost exclusively in the colon, and it was the enormous distention of its entire length which had so swollen the abdomen. The noise that can often be produced by pressure in the ileo-cæcal region (gurgling) used to be regarded, but probably erroneously, as especially characteristic of typhoid fever. Abdominal pain is often entirely absent. Some patients, however, complain of abdominal pain during almost the entire illness. On pressure, the belly is generally somewhat sensitive, but the tenderness is seldom extreme. It is more apt to be marked when there is constipation. Often such tenderness is due to a participation of the peritoneum in the disease, even when there is no perforation (*vide infra*).

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* To avoid repetition, we have in what follows united a description of the anatomical changes with the presentation of the clinical symptoms.

There still remain two symptoms of the greatest practical importance, both of which have a direct connection with the intestinal lesions: they are intestinal hæmorrhage and perforation.

Intestinal hæmorrhages in the course of typhoid are almost always due to the erosion of the walls of blood-vessels in connection with the formation and throwing off of the crusts of the ulcers. The hæmorrhages occur, therefore, most frequently toward the end of the second and during the third week. The blood pours out into the intestine, and is passed with the stools. Its amount may be small, or it may reach to one or two pints, or even more. Its color is generally rather dark. The later discharges are generally tarry. Liebermeister states that he has observed intestinal hæmorrhages in 7·3 per cent. of typhoid patients, and Griesinger in 5·3 per cent. We have ourselves seen, in the medical clinique at Leipsic, 45 intestinal hæmorrhages in 472 cases, i. e., in 9·5 per cent. In individual epidemics the frequency varies greatly. It rose in 1880 to eighteen per cent.

Intestinal hæmorrhage is always a grave symptom. Even slight hæmorrhages deserve consideration, for they may be the precursors of severer ones. And yet intestinal hæmorrhage, even if profuse, is not necessarily fatal. Of the above forty-five cases of typhoid with hæmorrhage, twenty-six ended in complete recovery. In eight cases, death occurred as the immediate result of the loss of blood. Eleven ended fatally after a time.

After every considerable intestinal hæmorrhage, the symptoms of general anæmia, often even of collapse, appear. The fall of the bodily temperature has been already mentioned. The hæmorrhage has sometimes a favorable influence on severe cerebral symptoms, for consciousness succeeds to the previous stupor or delirium. Often the hæmorrhage is directly followed by recovery from the disease.

Much more ominous than the intestinal hæmorrhage is the occurrence of perforation, as a result of the breaking through of a typhoid ulcer into the abdominal cavity, because, almost without exception, this is followed by a purulent or even ichorous peritonitis. The occurrence of perforation is sometimes marked by a violent pain suddenly felt by the patient; but it may also, even in severe cases, take place insidiously. The abdomen is generally (not always) greatly distended and very tender on pressure, so that even in stupor patients groan while being examined. If gas has entered through the opening into the peritoneal cavity, we often observe absence of the ordinary dullness over the liver; but this symptom is to be employed cautiously as a factor in diagnosis, for absence of hepatic dullness may also result from distended intestines lying in front of the liver. When perforation has occurred, the patient soon looks collapsed, with cheeks fallen in and sharp, cool nose. Frequent eructations and vomiting often follow. The pulse becomes small and very frequent. The temperature generally falls as the peritonitis begins, and later it usually undergoes great variations.

Perforation of the intestine occurs most frequently in the third or fourth week of the disease. In sluggish cases, however, we can not be without apprehensions of it till a late period. The perforation generally takes place in a coil of the lower part of the small intestine, and with marked relative frequency in the right side of the pelvis—seldom in the vermiform appendix or in the colon. With few exceptions, death comes quickly, after a few days at latest. Out of fifty-six fatal typhoid cases in the Leipsic medical clinique we lost five, or nine per cent., from peritonitis following perforation. Here and there a case of recovery has been reported, probably resulting from a limitation of the peritonitis through speedy adhesion of the intestines.

It should be mentioned here that sometimes in typhoid fever a local or general peritonitis may occur through direct extension of the process to the serous membrane without actual perforation. We have seen in one case, as a result of the peritonitic bands and false membranes, complete occlusion of the intestine (ileus), and death.

Swelling of the mesenteric lymph-glands (less often of the retro-peritoneal glands as well) is found in typhoid almost as constantly as the anatomical changes in the intestine. Sometimes they break down, i. e., suppurate. In cases that have passed through the disease we often find considerable deposits of lime in the glands. These changes have a certain clinical importance ; for, as already mentioned, we may often venture to refer a more or less tedious recurrent febrile state which has no other demonstrable cause to this lesion of the mesenteric glands. In some rare cases a general peritonitis has been observed as a result of the bursting of a suppurating gland.

The swelling of the spleen (acute splenic tumor) is, in typhoid fever as well as in many other acute infectious diseases, one of the most constant symptoms. The enlargement of the spleen can often be demonstrated as early as the end of the first week, and is therefore of considerable diagnostic importance ; but percussion of the spleen is sometimes decidedly difficult and deceptive in this disease because of the existence of tympanites. The surest demonstration of splenic enlargement is therefore always by means of palpation, which, after a little practice, gives a positive result in the majority of cases. Absence of splenic tumor is most frequently observed in elderly typhoid patients. The spleen may also diminish considerably in size after severe intestinal hæmorrhage. Pain in the splenic region, resulting from tearing of the distended capsule, is comparatively rare. The splenic infarctions which sometimes occur may, in exceptional cases, prove the starting-point of a peritonitis.

Hepatic symptoms are seldom seen in typhoid fever, except that there may be a moderate swelling of the organ. The anatomical changes of "parenchymatous degeneration," and the frequent formation in the liver of the small lymphomata which Wagner discovered, have no clinical significance. The bile secreted is generally pale and scanty. This is a partial explanation of the light color of the stools. A very rare complication, which we ourselves observed in one case, is acute yellow atrophy of the liver.

The stomach presents no especial anatomical changes in typhoid. Anorexia is an almost invariable symptom in the beginning and during the course of all severer cases. There is seldom any desire for food till recovery begins; but then, if convalescence is undisturbed, the appetite soon attains an enviable keenness. Vomiting in the beginning or course of the disease is an exception, unless after some error in diet. We have already mentioned it as a symptom of peritonitis.

The changes in the mouth and throat of typhoid patients deserve the careful attention of the physician. The lips and tongue are in severe cases dry and fissured. The lips are often covered with dry, black crusts, sometimes described as a "fuliginous coating." The tongue is apt to be thickly coated at first, but later cleans off from the edges and tip. In severe cases, especially if the mouth is not properly cleansed, a rather severe stomatitis may occur and produce superficial ulceration of the buccal mucous membrane and of the edges of the tongue. The gums sometimes become spongy, and are apt to bleed, as if scorbutic.

Actual sore throat, at least, according to our experience in Leipsic, occurs but seldom at the beginning of typhoid fever. The difficulty in swallowing, often complained of by patients, is generally due to dryness of the pharynx. In certain epidemics, however, the occurrence of sore throat at the beginning of the illness has been frequently observed. It may even happen that this early sore throat is accompanied by an erythema diffused over the body, so that at first suspicions of scarlet fever arise. In rare cases (so-called tonsillo-typhoid or pharyngo-typhoid) there are seen upon the tonsils peculiar whitish elevations, which later ulcerate. These are probably to be regarded as a specific typhoid lesion of the tonsils. It should also be mentioned that in severe cases there is often an extensive growth of thrush in the mouth and throat, and this may spread quite a distance down the œsophagus.

The changes in the mouth and throat are of especial interest, for the reason that they may be directly propagated to important neighboring organs. Starting from the pharyngeal cavity, the pathogenic agent, probably in most cases the staphylococcus, may penetrate through the Eustachian tube into the middle ear. Thus arise those inflammations of the middle ear which are not very rare in severe cases of typhoid, and which lead to perforation of the membrana tympani and to purulent discharges from the ear. The not infrequent inflammation of the parotid gland is also, as we believe, occasioned in a similar way, the inflammatory agent reaching the parotid gland from the mouth by way of Steno's duct. We do not regard the otitis and parotitis as especial localizations of the typhoid poison, but as genuine complications (secondary disease), for the occurrence of which typhoid fever merely furnishes the occasion, as when the mouth is imperfectly cleansed. The parotitis appears most frequently in the third week, and generally on one side, though sometimes on both. It almost always becomes purulent, and discharges either externally or into the external auditory meatus, unless there is a timely incision.

2. **Organs of Respiration.**—Affections of the lungs are among the most frequent and important complications of typhoid fever, but are for the most part not a direct result of the typhoid infection. The bronchitis very often found in severe cases, and especially in patients who do not come till late under proper care, certainly is chiefly dependent on the imperfect expectoration of the bronchial secretions and on the inhalation of inflammatory agents coming from the mouth and throat.

Numerous cases of typhoid of slight or average severity, under proper care, run their course without any considerable bronchitis. In many other cases, and even severe ones, the bronchitis remains within moderate bounds, especially if the patient is brought promptly under proper care and treatment; but in severe cases, where marked disturbances of the nervous system arise, and the patient in his stupor expectorates little, swallows things the wrong way, and lies all the time on his back, passive and collapsed, the occurrence of a severe, diffuse bronchitis, especially in the lower lobes of the lungs, can hardly be avoided. Nor in such cases is there generally a mere bronchitis, but a more or less extensive catarrhal, lobular pneumonia, to be classed therefore under the so-called inhalation pneumonias (cf. chapter on lobular pneumonia). What was formerly termed "hypostatic pneumonia" is also almost invariably to be put in this group.

From the way in which these pulmonary disorders arise, we can understand why the bronchitis sometimes takes on a putrid character, and why the lobular infiltrations are, in severe cases, transformed into genuine gangrene. If such spots touch the pleura, they occasion the development of a pleurisy which is almost always purulent. In rare cases, pneumothorax may arise as a sequel to the perforation of a gangrenous infiltration into the pleural cavity. Various circumstances promote the occurrence of pulmonary symptoms. Thus we find it especially easy for a severe bronchitis and its sequelæ to be developed, in the case of elderly persons, or the kyphoskoliotic, or the corpulent, or patients who have previously suffered from emphysema or cardiac disease.

The subjective thoracic symptoms, in typhoid patients who have pulmonary complications, are generally not very prominent. It is only occasionally that patients complain in the early stages of typhoid fever of pain, and of a sense of oppression in the chest, or of cough, or of a stitch in the side; and even when such symptoms exist, the physical examination may give comparatively insignificant results. The severer pulmonary complications are seen mainly in those whose intelligence is more or less blunted, and who, therefore, make little complaint, are not much disturbed by the dyspnœa, and cough and expectorate little. A careful physical examination alone can enlighten us as to their condition. On auscultation, sibilant rhonchi are the chief signs observed in the milder cases. In the severer ones there are moist, fine, and coarse râles, especially numerous toward the base of the chest. If there are abundant moist râles, we may infer that there is a lobular pneumonia, although this can not be demonstrated with certainty till the separate islets of infiltration unite into a more extensive solidification, so as to afford dullness on percussion.

In addition to the pulmonary lesions already mentioned, genuine croupous or lobar pneumonia does occur in typhoid fever. Probably this must be regarded as a direct result (localization) of the typhoid poison, although this croupous pneumonia is not anatomically distinguishable from the common, genuine pneumonia. It often appears as early as the second week, and attacks the lower as well as the upper lobes. Liebermeister states that he has sometimes observed it during convalescence.

Especial interest attaches to those cases of typhoid fever which begin with a lobar pneumonia. Often there is at first not the slightest suspicion of a typhoid fever, for the disease is regarded as an ordinary croupous pneumonia; but it is usually to be noticed that the illness does not begin suddenly with a rigor, but more gradually, and that from its incipiency the constitutional symptoms, the headache and splenic tumor, are more prominent than is usually the case in pneumonia. At the end of the first week's illness there is no crisis, but persistent fever. Now the pulmonary symptoms often retreat more and more to the background, while, on the contrary, diarrhœa and rose-spots appear. The spleen is enlarged. In short, the clinical picture of typhoid is developed. It is not unnatural to suppose, although there is yet no absolute proof of the fact, that in these cases, which are fittingly termed "pneumo-typhoid," the infection with the typhoid bacilli has taken place exceptionally in the pulmonary area, and that, therefore, the first pathological changes are developed in the lungs.

Laryngeal Lesions.—The same causes which produce the bronchitis result also in a simple catarrhal laryngitis, with hoarseness. This is in severe cases accompanied by superficial ulcers on the vocal cords or the posterior wall of the larynx. Sometimes, again, the lesion is due to mechanical causes, constituting the so-called "*decubitus laryngis.*" The disorders which attack the less superficial structures of the larynx are fortunately rare. Chief among them is a laryngeal perichondritis of the arytenoid cartilages. This complication is justly regarded as of bad omen, and may lead to the rapid development of œdema of the glottis, with great laryngeal obstruction and threatening suffocation. These severe laryngeal affections in typhoid are regarded by some authorities, especially by Klebs, as always the direct effect of the infecting poison; but in most cases they are probably due to an invasion of staphylococci or some similar microbes. We have several times seen laryngeal croup in typhoid fever, and it is a very dangerous symptom. We are inclined, however, to the belief that it was in every case a secondary disease.

Among symptoms referable to the mucous membrane of the nose, epistaxis is important. It occurs in the beginning of typhoid with tolerable frequency, and is in one way not unfavorable, for it often mitigates the patient's headache. At a later period nose-bleed may become a very unpleasant complication, as it is sometimes very difficult to check. We have even seen one fatal case due to persistent nose-bleed. Other nasal symptoms are exceptional. There is an old saying that typhoid never begins with a coryza.

3. **Nervous System.**—The old term "nervous fever," which is still used by the laity, shows how frequent and severe are the nervous derangements which occur in typhoid. In cases of any severity there is almost always a certain dullness of intellect, often amounting to apathy and somnolence. The patients give monosyllabic and incomplete answers to all questions, and their statements about their previous history are often disordered and contradictory. There may even be sopor or a deep coma in the worst cases. All cases of this sort in which there was a condition of intellectual enfeeblement were termed by the old physicians "*febris nervosa stupida*," in contrast to the "*febris nervosa versatilis*," that form in which abnormal mental activity or delirium predominates. In severe cases delirium is very frequent. It is generally worse at night, and at times when the patient happens to be left alone. Very often he tries to leave his bed, because of his delusions, and talks of persons and things with which he was formerly familiar; or he is very noisy and restless, sometimes shrieking from groundless fears. We may add that these diverse nervous symptoms frequently succeed one another, or appear in combination. Sometimes a soporose patient may be heard softly whispering to himself in "muttering delirium."

Certain motor disturbances are often combined with considerable impairment of consciousness. There is a slight twitching of the muscles of the face and extremities. The old authorities gave the name *subsultus tendinum* to the sudden leaping into prominence of the sinews thus caused. It is best seen on the back of the hands. In severe cases the patient is sometimes observed to grind the teeth together; this is due to a cramp-like condition of the muscles of mastication, and is justly regarded as ominous. We often see persistent tremor of the extremities and lower jaw; and it is especially in these cases, as we have demonstrated upon numerous patients, that the tendon reflexes and the mechanical excitability of the muscles are much increased. If deep coma comes on, the muscles become lax, the motions of the eye are not co-ordinated, and reflex excitability diminishes, or is wholly extinguished.

Headache is one of the most constant symptoms in the beginning of the disease. It is usually referred to the forehead or temples. The pain may be very violent, and sometimes takes on almost a neuralgic character. It almost always subsides in the second week.

If we seek the cause of these nervous symptoms, which are often so severe, we find that the anatomical changes in the nervous system, including the brain, bear no relation whatever to the severity of the symptoms observed during life. We sometimes meet with minute hæmorrhages in the cerebral meninges, or meningeal opacity or œdema, or a moist condition of the cerebral parenchyma; but the connection of these and similar changes with the symptoms of the disease is often more than doubtful. Nor can the microscopic alterations in the brain, which have been reported, be regarded as important and authoritative. It is only in very rare cases that large cerebral hæmorrhages or purulent meningitis have been found. As to this last, we should always be very cautious in making a diagnosis, as symptoms which would seem to be most conclusively meningeal—such as stiffness of the neck, rigidity of the whole spinal column, and occipital headache—may appear in typhoid patients, and yet the autopsy show no trace of meningitis.

One theory, which has Liebermeister for its chief supporter, and which has won a tolerably wide-spread acceptance among physicians, is that the nervous symptoms are chiefly a direct result of the febrile temperature. It is impossible, however, for us to regard this view as universally true. The unprejudiced consideration of a large number of personal observations prevents it. Although it is undeniable that elevated temperature has a harmful influence on the nervous system, yet in numerous cases there is no relation between the height of the fever and the severity of the nervous derangements. There are cases in which the fever remains continuously high for days, while the patient feels perfectly comfortable and presents no symptoms of any important cerebral disturbance. The opposite class of cases is still more numerous, in which from the very start there is always a low temperature, and, notwithstanding, the most severe nervous symptoms arise. Fräntzel has published very striking cases of this sort.

Hence we must seek for some other special cause of the severe nervous symptoms, and according to our present views this cause must be the intoxication resulting from the specific infection. We know that all bacteria produce, by their own tissue-metamorphosis and the chemical processes which they excite in their neighborhood, certain chemical matters which, especially in the case of the so-called "pathogenic bacteria," seem to be similar to the alkaloids ("ptomaines" and "toxines"), and exercise a decided poisonous influence upon the body, and especially upon the nervous system. These products are formed by the typhoid bacilli, enter the blood, and are the chief cause of the nervous phenomena. The difference in the violence of the latter in different cases probably depends mainly on a difference in the amount, and perhaps also in the quality, of the toxines produced by the typhoid bacilli, and probably also in the different susceptibility of individuals to the poison. The reason that the influence of these poisons is not much greater than it is, is that they are in part destroyed within the body and in part excreted with great rapidity, the channel of exit being mainly the kidneys. Thus is explained the interesting fact discovered by Lépine, Bouchard, and others, that the urine of typhoid patients possesses poisonous properties not present in normal urine.

That the appearance of the nervous symptoms is dependent not only on the material causes, but also on the susceptibility of the individual, is shown by the fact that certain patients are especially prone to exhibit marked nervous phenomena; for example, hard drinkers, "nervous" individuals, and also those who have suffered violent emotional disturbances shortly before the onset of the disease.

Actual insanity is not very infrequent during the course of typhoid, or in convalescence. It generally takes the form of melancholia. We have repeatedly seen patients in such a state that they would lie almost motionless in bed, with eyes open, and perhaps assert that they were dead! In other cases there is mental excitement, sometimes combined with hallucinations, or there is confusion of ideas. In one case, in a girl who was evidently predisposed to nervous disorders, we saw typical hysterical insanity break out during the fever. Sometimes the mental excitement at the beginning of a relapse terminates in actual insanity.

Few of the psychoses which arise during or at the end of typhoid outlast convalescence.

We have still to mention a number of nervous diseases that develop in the course of typhoid or after its decline. Neuralgia is sometimes seen, as well at the beginning as at the end of the disease. It is most frequent in the regions supplied by the trigeminus and the occipital nerves. Great hyperæsthesia of the skin and muscles is not rare during convalescence. It attacks the lower extremities by preference. Paralysis of single muscles (e. g., of the serratus magnus), or paralysis of a single extremity, has been repeatedly observed as a sequela. The paralysis is generally of the atrophic variety, and is probably, as a rule, due to neuritis. Ataxia and spastic paralysis of the lower extremities are rare sequelæ. Finally, there are sometimes developed, either in the course or at the conclusion of typhoid fever, the symptoms of a localized cerebral disorder (e. g., hemiplegia and aphasia), the anatomical cause of which varies. There may be a hæmorrhage or an embolism, and probably in still other cases a localized encephalitis.

4. **Circulatory System.**—Disturbances of the heart such as to produce striking anatomical changes are very rare. Endocarditis and pericarditis are, however, possible. The slight mitral endocarditis sometimes found at the autopsy has no clinical significance. On the other hand, great weight is laid by some authors upon the parenchymatous or fatty degeneration of the heart. They say it is often the cause of cardiac failure. We can not admit this, for experience shows that the two do not stand in any constant relation to each other.

The pulse is almost always rapid, although often not so much so as the height of the temperature might lead one to expect. It averages from 90 to 110, and often more. When it keeps at 140 or higher, in adults, it is always an unfavorable symptom. This abnormal frequency is often in part due to the high temperature; but there are other factors. Temperature and pulse do not correspond in all cases. Sometimes the pulse will have a normal or even subnormal frequency throughout the entire attack, despite the fever. Temporary accelerations are easily produced by mental excitement or bodily exertion, as by sitting up in bed. In convalescence the rate is frequently subnormal.

Slight irregularities of the pulse are not rare, either in the acme or the decline of typhoid. Marked irregularity is always a grave symptom, although in many cases it passes off and the patient recovers.

Dicrotism is so frequent that many elderly physicians still regard it as characteristic of the disease. It is often, however, equally marked in other acute diseases. Its cause is diminished arterial tension.

The diminished cardiac activity may result in venous thrombosis, especially in the lower extremities. This sometimes causes swelling of one of the lower extremities during convalescence. The swollen member generally regains its normal size after some weeks. In other cases the thrombosis occurs earlier, and in patients who are still too vigorous to suffer from cardiac weakness, so that we are forced to the conclusion that there is some local specific cause. A possible, but fortunately infrequent, result of these thrombi in the lower limbs is pulmonary embolism and sudden death.

In severe cases, which end in death, cardiac thrombi are sometimes found, with emboli in the lungs, spleen, kidneys, or other organs.

Œdema of the ankles and legs is very often seen in convalescents, especially when they first get out of bed. It is due to the weakness of the heart and changes in the vascular walls. Once we saw a general dropsy develop at the end of a severe attack in a girl of fourteen. The autopsy disclosed no other possible cause for it than the extreme atrophy and flabbiness of the heart.

5. **Skin.**—The eruption seen in typhoid fever is characteristic. The rose-spots

appear at the beginning of the second week, usually on the trunk, and chiefly on the abdomen. The number varies greatly. Rarely they are entirely absent, most often in elderly persons. Sometimes they are very abundant, and extend to the thighs, the arms, and even to the neck and face. Often they vanish after a few days, but they may persist much longer. In the latter case they may become to a very slight degree petechial, so that they will not entirely disappear on pressure. They often occur in successive crops. We have even seen several cases where new rose-spots kept coming for some days after the fever had disappeared.

As to other cutaneous eruptions, we may mention first of all that herpes labialis is so rare in typhoid that in cases of doubtful diagnosis it is a factor in excluding that disease. Miliaria, urticaria, and superficial pustules are sometimes observed. Occsionally little bluish spots appear, especially on the trunk. These used to be called "*taches bleuâtres*" (pelioma typhosum); but later observations show that they are not connected with typhoid fever particularly. They are due to pediculi. We might use the term pelioma typhosum to designate the kind of vesicles which we have repeatedly seen on the abdomen in severe cases. They are about the size of peas, and have sero-hæmorrhagic contents. Boils and superficial abscesses are frequent, especially as disagreeable sequelæ in convalescence from severe cases. There are often abscesses of the sweat-glands in the skin of the axilla during convalescence. All these and similar cases of suppuration in typhoid fever do not depend upon the original cause of the disease, but upon secondary pathogenic germs, such as the staphylococcus or streptococcus, for whose entrance the typhoid process has merely prepared the way. Extensive ecchymoses are very rare, and are symptomatic of a general hæmorrhagic diathesis. Petechiæ are frequent during recovery. They are generally seen in the follicles of the skin below the knee. There have been a few cases of gangrene in the lower extremities, especially in the toes. We saw in one patient an extensive gangrene of the skin of the abdomen. Its cause could not be determined.

Finally, we must mention that bed-sores are prone to develop in severe or neglected cases. The localities most often attacked are the nates, the furrow between them, and the heels. A bed-sore may be so extensive, and accompanied by such undermining of the skin, as to be a dangerous or even fatal complication.

The epidermis often scales off to a considerable extent during convalescence after a severe attack of typhoid. Everybody knows how the hair falls out after the fever, but it is sure to grow again. The nails also are not infrequently affected, becoming rough and brittle, or even falling off.

6. **Muscles, Bones, Joints.**—Zenker has discovered a degeneration of the voluntary muscles which occurs in typhoid as well as in other severe diseases. It is called the "granular" or "waxy" degeneration. Whether it has clinical symptoms can not be determined. Perhaps it may explain the great muscular hyperæsthesia which is often observed, and the muscular pains, which may be very trying. Severe cases sometimes have hæmorrhages into the muscles, particularly the rectus abdominis.

Lesions of the bones and joints occur but seldom. We have seen periostitis of the tibia, and of a rib, during convalescence. Swelling of the joints is equally rare. If there is a purulent arthritis, it is always due to some secondary infection (*vide supra*).

7. **Genito-urinary Apparatus.**—Genuine, acute, hæmorrhagic nephritis is a very rare complication. It does occur, however, and has even given rise to the establishment of a special "renal form of typhoid fever" (nephro-typhoid). This name applies especially to those cases in which a severe acute nephritis is the predominant symptom at the start, while at a later period the course of the fever, the intestinal symptoms, the rose-spots, etc., show the disease to be typhoid fever.

Nephro-typhoid is analogous to pneumo-typhoid and tonsillo-typhoid. A simple so-called febrile albuminuria occurs very frequently at the acme of typhoid, and is not to be interpreted unfavorably. It is probably the result of that slight parenchymatous degeneration of the kidneys which occurs in typhoid with the same frequency as in most of the other severe infectious diseases. There does not seem to be a direct relation between the albuminuria and the fever, although some authors assume it to exist. It is more likely that the renal epithelium is injured by the noxious products which have been formed in the body and excreted by the kidneys. In other respects the urine presents the same peculiarities as in most other severe febrile diseases : its amount is diminished ; its color dark ; its specific gravity increased ; the excretion of urea greater than normal. It should be added that the urine at the height of the disease presents Ehrlich's "diazo-reaction" in almost all cases.* Cystitis is not a rare development toward the end of the illness. It is probably always secondary.

In men, orchitis is sometimes observed. Women often have their catamenia at the beginning of typhoid. Later in the course of the disease, and in convalescence from severe attacks, the menses are often absent for several periods. In pregnant women there is considerable danger of abortion or miscarriage.

Peculiarities in the Course of the Disease.

The above statements show an almost inexhaustible variety in the possible complications of typhoid. The course of the disease as a whole may likewise present many diverse forms and peculiarities. We shall attempt merely to cite the most essential.

The numerous light and rudimentary attacks (*typhus levissimus*) are first to be mentioned. It was not recognized till lately that they belonged to typhoid fever at all (Griesinger). They used to have all sorts of names applied to them, the favorite term being "gastric fever." This light form lasts eight to fourteen days. The fever is moderate and often decidedly remittent. There is almost no proper fastigium. The typhoid symptoms are but slightly developed. There are no severe pulmonary or cerebral symptoms. There is generally a moderate diarrhœa, the spleen is plainly enlarged, and often rose-spots can be found. The diagnosis of these cases is of course difficult in proportion to the scanty development of typhoid symptoms. It is best established by demonstrating an ætiological relation between these cases and others which are plainly typhoid fever.

Abortive typhoid is justly distinguished by Liebermeister from *typhus levis.* The name belongs to cases which begin with severe symptoms and high fever, as if they were going to be grave, but in which these violent symptoms disappear after a few days and give place to a rapid convalescence.

On the other hand, there are cases which for a long time cause so little subjective discomfort that the patient does not even go to bed (walking typhoid). It is not till quite late that there occurs a sudden change for the worse, or some severe complication. Thus it has happened that people who were apparently healthy have suddenly had all the symptoms of a severe peritonitis due to perforation and have died, the autopsy disclosing the lesions of the third week of typhoid fever.

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* This reaction consists in the red coloration of the urine upon the addition of Ehrlich's reagent (chiefly sulphanilic acid) and ammonia. For particulars, see treatises upon chemical analysis. [Ehrlich's test is carried out as follows: Solution 1 consists of strong hydrochloric acid, one part, mixed with twenty parts of a saturated aqueous solution of sulphanilic acid. Solution 2 is a one-half-per-cent. solution of sodic nitrite in water.

A test-tube is one-eighth part filled with solution 1, and five to ten drops of solution 2 are added to this; a quarter of the test-tube of urine is then added, and, lastly, enough ammonia to make it strongly alkaline. The deep-red color, which appears promptly, is not so characteristic as a green precipitate of phosphates, which appears at the bottom of the tube after standing for twenty-four hours.]

The individual circumstances are very important in weighing each case, for they may modify the disease in many ways.

In children it is a remarkable fact that typhoid ulcers are much less frequent than in adults. This explains why intestinal hæmorrhage and peritonitis are much rarer in children. Severe cerebral symptoms are, on the other hand, very frequent. In severe cases children sometimes exhibit the peculiar symptom of a continuous penetrating screaming. In other, mild, cases the children are soporose.

In the aged the diagnosis of typhoid is often very difficult, since the course of the disease is frequently irregular. Generally the fever is not very high, and it very seldom exhibits distinctly the type described above. The pulmonary or cerebral symptoms predominate as a rule.

In the corpulent, typhoid fever is often very severe, so that our prognosis must always be rather grave, especially if pulmonary symptoms arise.

Hard drinkers are also in especial peril in this as in all other acute diseases. Dangerous cardiac weakness is prone to appear. Severe cerebral symptoms are frequent. It is, however, surprising that true delirium tremens is relatively infrequent, although so common in pneumonia.

The influence of previous strong mental excitement and of certain already existing diseases (cardiac disease, emphysema, kyphoskoliosis, etc.) has been already mentioned. Finally, we repeat that often the different epidemics present certain peculiarities. For instance, in one the type of the disease will be severe, in another mild. In one epidemic relapses are comparatively frequent, in another exceptional. The same is true with regard to the frequency of the appearance of certain symptoms, such as intestinal hæmorrhage, pneumonia, or nephritis. Indeed, it has even been observed that those cases which occur during a given epidemic in the same family or house or block sometimes present striking resemblances to one another ("group typhoid" of E. Wagner and others).

RELAPSES OF TYPHOID FEVER.

Typhoid fever exhibits in many cases the peculiarity of repeating itself completely after having run its entire course and disappeared. This process is called a relapse. It is in all probability the result, not of a fresh infection of the system from without, but of a renewed development, or possibly of a second generation, of the infectious germs already present. A typical relapse is like a first attack in all clinical and anatomical particulars, with this difference, that everything is more condensed, and lasts a shorter time than in the first attack. The interval between the two, during which there is no fever, lasts seven to ten days. It may be longer, and is often shorter. Sometimes the relapse follows immediately upon recovery. Indeed, it may even happen that, before the patient has completely recovered, his temperature begins to rise again in the characteristic step-like way. To such cases as this last the term recrudescence is applied. Except in the time of its beginning, it may be just the same as a genuine relapse. In the interval between the two attacks many persons are perfectly comfortable, and appear to be fully convalescent. There is often, however, a slight evening rise of temperature. It is noticeable that the splenic tumor does not completely disappear after the first attack in many cases which are followed by a relapse.

The relapse is generally briefer, as we have said, than the first attack, seldom lasting more than fifteen to eighteen days. The temperature rises more rapidly, perhaps in two or three days. The fastigium is shorter, the decline more abrupt.

The absolute height of the temperature may be very considerable, even exceeding that in the first attack. Rose-spots appear as soon as the third or fourth day. The stools become liquid, the spleen enlarges again, and all sorts of complications may arise. The danger occasioned by a relapse may, however, be overestimated. On the whole, a relapse is not so very dangerous, and it is especially noticeable that the subjective symptoms, such as headache, are often slight. A severe relapse may follow a mild case. In other instances the relapse may prove merely rudimentary.

The frequency of relapses varies considerably in different epidemics. In Leipsic, we had relapses in about nine per cent. of all cases, but in separate years the percentage varied between four and sixteen. Out of about five hundred cases we have seen three in which there were two successive and typical relapses.

Diagnosis.—The diagnosis of typhoid fever may be perfectly easy, but, if the case be anomalous, or come under observation at a late period, it may be extremely obscure. Inasmuch as a search for the specific bacilli is too difficult and troublesome, for the practicing physician, the diagnosis of the disease must be made from its course and symptoms. Important factors are the gradual onset, then the height and course of the fever, with no demonstrable localized disease, and the rose-spots. Less characteristic, but still of value, are the stools, the tympanites, and the swelling of the spleen. Ætiological factors, such as the occurrence of undoubted cases of typhoid in the neighborhood, are of great diagnostic value in obscure cases. Sometimes the diagnosis can not be established till the appearance of certain symptoms, like intestinal hæmorrhage, a characteristic mode of convalescence—viz., by lysis—or a relapse. It is an important rule not to make a diagnosis of typhoid after a single examination. It is generally necessary to observe the case accurately for several days before the diagnosis can be established. The differential diagnosis from other acute diseases, such as miliary tuberculosis, acute endocarditis, meningitis, etc., will be considered in discussing these diseases.

Prognosis.—A perfectly favorable prognosis should never be made. Cases which seem the mildest may become dangerous. Yet, if there are good nursing and good treatment, typhoid fever is not a particularly dangerous disease, and we may hope for recovery even in very severe attacks. The danger lies, first, in the severity of the infection, as shown chiefly (though not wholly) by the height of the fever and the intensity of the general symptoms. A further danger is the appearance of the complications already enumerated and discussed. Thirdly, the constitution and condition of the individual are important. The circumstances coming under this head have likewise been repeatedly mentioned above. All these factors must be carefully estimated before we decide as to the danger in each case and make our prognosis.

The mortality in typhoid varies greatly in the separate epidemics. The severe cases are undoubtedly more frequent at some times than at others. This renders it difficult to give statistics which are universally applicable. We may in general reckon on an average mortality of about ten per cent., and measure the severity of separate epidemics by this standard. Numerous observers agree that the treatment now in vogue has decidedly diminished the mortality. It was formerly not rare for it to reach twenty or twenty-five per cent.

Treatment.—A specific cure for typhoid—i. e., some remedy to destroy the specific cause of the disease within the system, or to render it harmless—is as yet unknown. Antiseptic and antizymotic drugs, such as quinine and antipyrine (*vide infra*), do have a certain influence upon the fever, but they are not capable of essentially modifying the course of the disease as a whole, at least not in such doses as we dare admininister. The continued internal use of carbolic acid (grains five to ten, gramme 0·30–0·50, or more, in the course of twenty-four hours) is the means chiefly recommended lately for this purpose; but we doubt if it is of much benefit. Liebermeister ascribes to iodine a demonstrable, although slight, beneficial influence. Other physicians had previously recommended it. Four to five drops of the following solution are given every two hours in a wineglass of water: Iodine, one part; iodide of potassium, two parts; distilled water, ten parts. We have had no personal experience with this remedy.

Calomel is also said to have a specific effect on typhoid. Wunderlich and others have noticed that if a few rather large doses of calomel be given at the beginning of the disease, it will on the average run a lighter and more favorable course than otherwise would have been the case. Wunderlich believed that typhoid fever may sometimes be aborted by this method. Although we can hardly expect this, it is really an efficient means of procedure, which we have often found satisfactory, to give two or three powders, of five grains (0·30 gramme) each, of calomel, as the first prescription, to patients who come under treatment in the first week or the beginning of the second. As there is generally constipation, the laxative effect is also beneficial. Moreover, it often lowers the temperature somewhat. A moderate diarrhœa is not a contra-indication, but, if the bowels be very loose, the calomel should be omitted.

Ergotine may be mentioned as another drug which is said to act specifically. It has been lately used, chiefly by French physicians, in doses of twenty to forty-five grains (1·50–3 grammes) in twenty-four hours. Again, subnitrate of bismuth is used (by the French to the amount of four to eight drachms—grammes 15–30 *pro die*), and naphthaline in single doses of three to eight grains (0·1–0·5) or in the course of the day seventy-five grains (5·0). We doubt if these remedies will be popular long.

In the present state of our knowledge the treatment of typhoid must still be chiefly general and symptomatic, and in one sense prophylactic. We must fight the symptoms already present, and further seek, as far as possible, to defend the patient from the attack of certain dangerous secondary disorders. Starting out with this view, the proper treatment of typhoid fever is a task of the highest importance, and by no means a thankless one.

We will begin by considering the general treatment. The sick-room must not be too warm, and must be frequently and thoroughly aired. The sick-bed must be well cared for. If pains be taken to prevent bed-sores, we shall obviate one source of pain and danger (*vide supra*), and save ourselves and the nurse much trouble. Those who are very sick should therefore be laid on an air-cushion, or, if possible, a water-bed. The patient should be told not to lie always upon his back, but to change now and then upon his side. The back, the region of the sacrum, and the heels are to be often bathed with spirits of camphor or brandy. The minutest bed-sore is to be treated carefully. It should be cleansed twice a day (rinsed off with a weak solution of salicylic or carbolic acid), and done up with an ointment containing Peruvian balsam, 1–30.* If the bed-sore be extensive, dusting with iodoform is very efficient treatment. We should be particularly careful not to let the skin be undermined. If this has already occurred, we must be prompt in the use of the knife or drainage-tube.

We can not recommend too strongly that the mouth should be kept clean. In a light case the patient can see to this himself, but otherwise the mouth and tongue must be frequently cleansed with a linen cloth wet in cold water or a solution of borax (1 to 30). Perhaps we need hardly repeat the reason for this excessive cleanliness. It lies in the causative relation between stomatitis and inflammation of the parotid gland, and of the middle ear. If the tongue and lips be dry, they may be touched with glycerine.

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* The unguentum balsami peruviani is made by mixing one part of balsam very exactly with thirty parts of the glycerite of starch (B. P.). It is not officinal in Germany.—TRANS.

The diet must be at once liquid and nourishing. Milk is excellent, and should always be ordered, but will, unfortunately, be taken by very few patients continuously. It is often better borne if coffee or a little brandy be added to it. Cocoa made with milk may also be given for a change. In severe cases Nestlé's food (Kindermehl) has been often employed by us with benefit. Broth and soup, thickened with sago or rice, are also good. They may be made more nourishing by adding an egg to them. If the patient is very anxious to have more solid food, as often happens, we need not hesitate to give him a roll or rusk (Zwieback) that has been softened by soaking. If a patient becomes exceedingly enfeebled we should give him fine shavings of raw beef, regardless of the fever. A little dilute hydrochloric acid might be given with the meat. Beef-tea would be still better than the raw meat, and is to be strongly recommended. The various preparations of meat which are now made (meat-solution, meat-peptones, etc.) may be sometimes useful. Where the fever takes a sluggish course, we must often begin to give stronger nourishment before the fever has ended. The best drink is cold water, which the patient would often not think of using unless we offered it to him. Lemonade and similar preparations generally become distasteful in time. Drinks containing carbonic dioxide are to be avoided, because they increase the meteorism. Cold tea with milk is good. In severer cases we should give some good strong wine, such as port, Malaga, or Hungarian wine. If the patient desires beer, we need not hesitate to give it in moderate amount. During convalescence we should be very careful about diet, since errors often have disagreeable consequences. We must wait till there has been no fever at all for seven to ten days before we allow a solid, animal diet, and return by degrees to common sorts of food.

The general and dietetic treatment which we have thus far discussed is very important. Outside of this, it is our opinion that there is only one method of treatment to be chiefly considered—at least under the present limitations of our therapeutic ability. This method consists in the persistent use of cool baths, as first practiced by Brand in Stettin. We do not indeed believe that the indications for this method of treatment are exactly what its original promoter held them to be, and we think some of the minutiæ of the treatment should be changed. Yet there is at present no other single method of treating typhoid fever which has so numerous and evident advantages for the patient. To carry it out in private practice may often be more difficult than in a well-appointed hospital. However, even in private houses it will generally be possible to manage it, and we regard it as the duty of every physician who undertakes to treat a severe case of typhoid to try his best to have the baths employed.

The great advantages of the treatment by baths are: 1. The baths diminish the fever, if their temperature be only sufficiently low, by direct absorption of heat. The baths thus obviate, as far as possible, all the bad effects which might result from a rise of temperature. 2. The direct influence of the baths upon the nervous system is still more important. The intellect becomes clearer, the apathy and dullness diminish. In fact, if baths be used, we do not see nearly so often as formerly the grave "typhoid condition." It is thus evident that bathing not only causes an improvement in the subjective sensations of the patient, but brings in its train many other beneficial effects. The patient takes his nourishment better, does not so often swallow the wrong way, coughs more effectively, is easier to move, and his body and his mouth can be better cleansed. 3. The influence of the baths upon the respiratory organs is of the greatest importance. We refer especially to the stimulation to deeper inspirations, and the promotion of expectoration. The best proof of the benefit of this influence is the circumstance that, if patients are subjected to baths from the start, it is comparatively a rare thing for severe bronchitis, atelectasis, and catarrhal pneumonia to develop. 4. The good care of the skin, which the bathing makes possible, is not to be despised. Since this treatment has been introduced, bed-sores are much rarer in typhoid than before. 5. Lastly, the baths are sometimes observed to have a diuretic effect.

What has been said shows that the height of the fever is by no means the sole indication for the employment of baths, at least in our opinion. The condition of the nervous system and of the respiratory organs is also to be considered. It is true that numerous mild cases run a favorable course without a single bath; but we should always remember that this treatment is not only directed against the symptoms already existing, but has also a prophylactic importance, since it tends to prevent any severe cerebral or pulmonary manifestations.

We will pass on to the special method of carrying out balneo-therapeutics in typhoid. Full baths are generally employed, immersing the patient to his neck. The tub must stand, if possible, by the bedside. In hospitals, where there are beds on rollers, it is a better way to wheel the patients into the bathroom. All who are severely ill should be lifted into the bath and there held and supported, to avoid any bodily fatigue. During the bath the skin should be gently rubbed. This averts unpleasant sensations of chilliness. The temperature of the water should not be set too low, especially for the first baths. We begin at 85° to 90° (24° to 26° R.), or, if the individual be elderly or sensitive, and timid, at even warmer temperatures. When the patient has become accustomed to the temperature of the water, we can cool off the bath still further. Baths below 73° (18° to 20° R.) have scarcely ever been used by us, and we believe that they are seldom needed. A very satisfactory average temperature is 80° to 85° (20° to 24° R.). A bath lasts on the average ten minutes. If the patient feels very cold or very uneasy in the bath, it must be cut short. After the bath the patient is at once lifted into bed, wrapped up in a sheet previously made ready, and wiped dry, with rather vigorous rubbing of the extremities and back. The moist sheet is then removed. The patient is covered up rather warmly, and is given some hot broth or a sip of good strong wine. The effect of the bath upon the temperature is measured about half an hour later by the rectum. If the temperature be 2° to 3° (1° to 2° C.) lower than before, the result is deemed satisfactory. Often the difference is greater, but in severe cases the fever may be so obstinate that the temperature remits only a small fraction of a degree. In such cases it is sometimes permissible to lower the temperature of the bath still more, or continue it a little longer. If cool baths are ill-borne, protracted baths of lukewarm water are sometimes very efficient (Riess, and other).

In so far as the height of the fever furnishes an indication for baths, we may accept, say 103·6° (39·8° C.) in the rectum, as the temperature calling for a bath. As a rule, baths should not be given oftener than every three hours, as otherwise they exhaust the patient. In many cases three or four baths a day are enough. At night we have given baths very seldom, when forced to by extremely high temperatures or other bad symptoms. It must be a mistake to wake a patient who is quietly sleeping, and put him into cold water, even if his temperature is above 104° (40° C.). Likewise, in cases where the temperature shows considerable spontaneous remissions, there may be no use in inflicting a cold bath upon a patient who has high fever only temporarily. On the other hand, even if the temperature be not excessive, or even if it be normal, there is, as we have said, no better remedy than the baths for severe pulmonary or cerebral symptoms. In such cases we often raise the temperature of the baths a little, and during them we have colder water poured upon the head and back. If we do this, the ears must be stopped with cotton-wool, lest the cold water find its way into them.

It is not always advisable to use baths, however advantageous this treatment may be, in typhoid fever. There are a number of contra-indications which can not be disregarded. First, the occurrence of intestinal hæmorrhage, however slight, and likewise the suspicion that peritonitis is developing, prohibit bathing. In these cases quiet is the very first requirement of the patient, and the baths must be at once discontinued. Further contra-indications are great weakness or great sensitiveness, such that the excitement caused by the bath might do harm. Sometimes baths are followed by severe rheumatic ("rheumatoid") pains in the limbs, and often they seem to promote the occurrence of furunculosis. In such cases it is often necessary to omit the baths, or at any rate to employ them less often and at a warmer temperature. The same is true if a severe laryngeal affection develops, or otitis or nephritis. Nothing seems to us a greater mistake than to attempt to establish a scheme for the treatment of typhoid by baths that shall be always applicable. Here, if anywhere, the only correct way is to treat each individual case according to its special symptoms and circumstances.

We shall now pass on to the consideration of the further symptomatic treatment of typhoid. The first question is whether the fever—that is, the elevation of bodily temperature, as such—demands special consideration. In general, we are of the opinion that the internal antipyretics are seldom indispensable. It is true that by giving quinine (single doses of fifteen to twenty grains, gramme 1 to 1·50) or salicylate of soda (in amounts of a drachm or a drachm and a half, grammes 4 to 6), the elevated temperature may often be considerably diminished; but whether the patient is thereby benefited is at least doubtful. Certainly the unpleasant accessory symptoms caused by the drugs mentioned—viz., vomiting, ringing in the ears, vertigo, and profuse perspiration—make the patient feel decidedly more uncomfortable than he was before. There is also some danger that these medicines may have an unfavorable influence upon the cardiac activity. Decidedly better than quinine and salicylic acid is antipyrine. This substance was synthetically produced by L. Knorr from phenylhydrazin and acetacetic ether, and was first recommended by Filehne. In the dose of fifteen to thirty grains (grammes 1 to 2) it is a very efficient antipyretic. It may be given in wafers or dissolved in water, and it is easy to take. Exceptionally there are disagreeable results, such as vomiting, an eruption resembling measles, or a rigor followed by a return of fever. In severe cases the dose must be repeated after two or three hours. More than eighty or ninety grains (grammes 5 to 6) as a maximum should not be given in one day. The general condition often improves under the influence of antypyrine. Especially is this true with regard to headache, restlessness, and other nervous symptoms. Another very efficient antipyretic is acetanilide, which was lately discovered by Cahn and Hepp, and has already been largely introduced into medicine under the name of antifebrin. One advantage is that it is far cheaper than antipyrine. It is given in doses of four to eight or even fifteen grains (gramme 0·25, 0·5, 1·0) either in wafers or in wine. It usually causes a decided lowering of the temperature, and seldom with any unpleasant effect, such as a chill. One disadvantage, however, is the frequent appearance of a peculiar bluish discoloration of the skin, which is probably dependent upon a change in the coloring matter of the blood, similar to that seen in poisoning from aniline. It is therefore probable that caution should be used in giving the drug. Numerous other antipyretics, such as kairin and thallin, have been proposed, but have not become popular. It is also improbable that a continued administration of small doses of thallin (two thirds to one grain, gramme 0·04, 0·06) every hour would have a favorable influence upon the course of the disease, although this has been asserted. The newest antipyretic of all is phenacetine, which was recommended by Kast and Hinsberg. Its dose is about fifteen grains (gramme 1).

After all, it should be borne in mind that the only benefit from the internal antipyretics consists in the lowering of the temperature, while baths not only abate the fever, but possess numerous other advantages, as has been already shown. If we had to choose whether to treat typhoid fever exclusively with baths or with antipyrine, and the like, we should certainly choose the baths. We do not by any means desire to banish the use of internal antipyretics from the treatment of typhoid, but only to make their employment more limited than has often been the case. We consider that they are actually indicated only where the fever is high and the employment of baths is for some reason impossible or contra-indicated, or where the fever remains continuously high, despite bathing. In such cases it is often advantageous to combine the bath-treatment and the internal antipyretics, especially in the evening. If patients with a moderately high fever are made to take large doses of quinine and the like, without any satisfactory reason for it, we regard such treatment as at any rate useless and often really injudicious. This is, unfortunately, a common practice, and frequently its only permanent result is a disordered stomach.

[Water or an acid drink should be given frequently by the nurse without waiting for the patient to ask for it, unless the mind is unusually clear. Phenacetine seems to have proved itself less depressing to the heart than its predecessors. The antipyretic dose usual in this country is five grains. A strong protest should be entered against the routine or frequent use of any of these internal antipyretics. If the temperature seems, in itself, to cause restlessness and discomfort, an occasional dose may be given. When used early in the course of the disease, antipyretics may seriously embarrass the diagnosis in doubtful cases. The method of Brand has been slowly working its way in America of late years, and perhaps would have spread more widely and rapidly were it not that we have long been in the habit of frequently sponging our typhoid patients with alcohol and water.]

Another important symptom which needs special treatment is intestinal hæmorrhage. It has been already mentioned that if this occurs, the baths should cease at once. Further than this, the chief remedies are ice and opium. Flat ice-bags are laid upon the abdomen. They should not be too heavy, and should, if possible, be suspended from a hoop. Internally, the patient is given every two hours a powder of one-half grain or one grain (gramme 0·03 to 0·05) of opium, either pure or combined with acetate of lead (opii, gr. ss., grm. 0·03; plumbi acetatis, gr. j, grm. 0·05; sacchari albi, gr. j, grm. 0·05). The object of the opium is to check peristalsis, and thus promote the formation of a clot in the bleeding vessel. Liquor ferri chloridi (five to ten drops in water every hour) is often employed, but is of extremely doubtful value. The baths can not be resumed till there has been no bleeding for at least three or four days—and then only cautiously.

If peritonitis occurs, the treatment is much the same. Above all, opium must be used in still larger doses, but, unfortunately, as a rule, in vain. Perhaps surgical treatment is destined eventually to be useful, viz., incision, cleansing, and drainage of the peritoneum. Its results thus far are not very encouraging.

If there is considerable diarrhœa, we can give mistura gummosa [P. G., gum arabic and sugar, each 15 parts; water, 170 parts], tannin, subnitrate of bismuth, or small doses of opium. Constipation at the beginning of the disease is overcome by calomel (*vide supra*). In later stages we always try enemata first, to produce an operation. If this does not succeed, then we must employ rhubarb or castor-oil. Great tympanites may be diminished by laying cold wet cloths or ice-bags upon the belly. Considerable amounts of gas may often be removed by introducing a long rectal tube. As to puncturing the greatly inflated intestines, a method practiced by some physicians, we have no personal experience.

If there are severe pulmonary symptoms, baths and pouring on cool water are, as we have said, the best remedies. Internally we may try liquor ammonii anisatus [P. G., olei anisi, 1 part; aquæ destillatæ, 24 parts; aquæ ammoniæ, 5 parts] and benzoic acid (grains ij to iij, gramme 0·1 to 0·2, in powder). If the pulse be very rapid, we can put an ice-bag over the heart. If at the same time the pulse is small and weak, we give stimulants, of which the best is camphor (*vide infra*). Digitalis (one-half grain of the leaves, gramme 0·03, two or three times daily) may also be employed if the pulse be rapid; but it should be used with great caution.

For nervous symptoms the baths and douching are the most effective remedies. The head is meanwhile covered by an ice-bag. If there be great excitement, as shown by excessive restlessness or delirium, small doses of morphine are often very useful.

The conditions of collapse and cardiac failure, which sometimes appear rather suddenly, demand prompt and energetic treatment. Stimulants to be given internally are some stronger kind of wine, camphor (two to five grains, gramme 0·10 to 0·30, in the form of a powder), musk (one half grain to one grain, gramme 0·03 to 0·05 at each dose), or spiritus ætheris [P. G., æther, one part; alcohol, three parts]. Subcutaneous injections act quicker and are much more convenient. We may use either ether or camphor (one part camphor to four parts olive-oil, seven to fifteen minims, c. c. 0·5 to 1·0, every one to two hours). To start up respiration, the best means is to pour cold water on the back of the neck. Artificial respiration also succeeds, in many cases, in reviving the breathing when it is about to stop.

The numerous other complications and sequelæ which may occur, but which can not all be mentioned here, should be treated on general principles.

The prophylactic measures to avoid the spreading of the disease can be only briefly referred to. Of chief importance is careful disinfection of the excreta, which can be accomplished by pouring upon the stools a not too small amount of five-per-cent. carbolic solution. We should also take care that bed-pans, bed-clothes, linen, etc., are handled by other persons as little as possible. If there seems reason to suspect that the disease arose from bad water, of course the source of such suspected water must be cut off.

[Recent experiments tend to show that the above solution of carbolic acid does not kill spores except after prolonged contact.

The following are the measures of disinfection recommended by the American Public Health Association. It will be observed that they apply to all infectious diseases, and it seems well to give them here nearly *in extenso*, as the directions for disinfection in most text-books are far too vague.

Disinfection of Excreta, etc.—The infectious character of the dejections of patients suffering from cholera and from typhoid fever is well established; and this is true of mild cases and of the earlier stages of these diseases as well as of severe and fatal cases. It is probable that epidemic dysentery, tuberculosis, and perhaps diphtheria, yellow fever, scarlet fever, and typhus fever, may also be transmitted by means of the alvine discharges of the sick. In cholera, diphtheria, yellow fever, and scarlet fever, all vomited material should also be looked upon as infectious; and in tuberculosis, diphtheria, scarlet fever, and infectious pneumonia, the sputa of the sick should be disinfected or destroyed by fire. It seems advisable also to treat the urine of patients sick with an infectious disease with one of the disinfecting solutions below recommended.

Chloride of lime, or bleaching powder, is, perhaps, entitled to the first place for disinfecting excreta, on account of the rapidity of its action. The following standard solution is recommended :

STANDARD SOLUTION NO. 1.

Dissolve chloride of lime, of the best quality,* in pure water, in the proportion of four ounces to the gallon. Use one quart of this solution for the disinfection of each discharge in cholera, typhoid fever, etc. Mix well and leave in vessel for at least one hour before throwing into privy-vault or water-closet. The same directions apply for the disinfection of vomited matters.

STANDARD SOLUTION NO. 2.

Dissolve corrosive sublimate and permanganate of potash in pure water, in the proportion of two drachms of each salt to the gallon. This is to be used for the same purposes and in the same manner as Standard Solution No. 1. It is equally effective, but must be left a longer time in contact with the material to be disinfected—at least four hours. The only advantage this solution has over No. 1 consists in the fact that it is odorless. It costs about two cents a gallon. It is very poisonous, and will injure lead pipes if passed through them in considerable quantities. Solutions of corrosive sublimate should not be placed in metal receptacles.

Disinfection of the Person.—The surface of the body of a sick person, or of his attendants, when soiled with infectious discharges, should be at once cleansed with a suitable disinfecting agent. For this purpose solution of chlorinated soda, diluted with three parts of water, or Standard Solution No. 1, diluted with three parts of water, may be used. A two-per-cent. solution of carbolic acid is also suitable for this purpose, and, under proper supervision, the use of a solution of corrosive sublimate (1–1,000) is to be recommended.

In diseases like small-pox and scarlet fever, in which the infectious agent is given off from the entire surface of the body, occasional ablutions with solution of chlorinated soda, diluted with twenty parts of water, will be more suitable than the stronger solution above recommended.

In all infectious diseases the body of the dead should be enveloped in a sheet saturated with Standard Solution No. 1, or with a five-per-cent. solution of carbolic acid, or a 1–500 solution of corrosive sublimate.

Disinfection of Clothing.—Boiling for half an hour will destroy the vitality of all known disease-germs, and there is no better way of disinfecting clothing or bedding which can be washed than to put it through the ordinary operations of the laundry. No delay should occur, however, between the time of removing soiled clothing from the person or bed of the sick and its immersion in boiling water, or in one of the following solutions; and no article should be permitted to leave the infected room until so treated.

STANDARD SOLUTION NO. 3.

Dissolve four ounces of corrosive sublimate and one pound of sulphate of copper in a gallon of water. Two fluid ounces of this standard solution to the gallon of water will make a suitable solution for the disinfection of clothing.

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* Good chloride of lime should contain at least twenty-five per cent. of available chlorine. The cost of the solution is less than one cent a gallon. The sediment does no harm.

The articles to be disinfected must be thoroughly soaked with the disinfecting solution, and left in it for at least two hours, after which they may be wrung out and sent to the wash.

Clothing may also be disinfected by immersing it for four hours in a two-per-cent. solution of carbolic acid. Soiled mattresses, pillows, feather beds, and articles of this nature can not be effectually disinfected by sulphur fumigation, owing to the fact that the gas does not penetrate to their interior in sufficient amount. For articles of this kind, and in general for articles of little value, which have been soiled by the discharges of the sick, destruction by fire will be advisable.

Disinfection of the Sick-Room.—No disinfectant can take the place of free ventilation and cleanliness, and it is impracticable to disinfect an occupied apartment. Neutralizing bad odors is not disinfection.

All surfaces should be thoroughly washed with Standard Solution No. 1, diluted with three parts of water, or with a 1–1,000 solution of corrosive sublimate. Standard Solution No. 3, diluted in the proportion of four ounces to the gallon of water, may be used.

The walls and ceiling, if plastered, should be brushed over with one of these solutions, and subsequently washed over with a lime-wash.

Especial care must be taken to wash away all dust from window ledges and other places where it may have settled, and thoroughly to cleanse crevices and out-of-the-way places. After this application of the disinfecting solution, and an interval of twenty-four hours or longer for free ventilation, the floors and wood-work should be well scrubbed with soap and hot water, and this should be followed by a second more prolonged exposure to fresh air, admitted through open doors and windows.

As an additional precaution, fumigation with sulphurous-acid gas is to be recommended, especially for rooms which have been occupied by patients with small-pox, scarlet fever, diphtheria, typhus fever, and yellow fever. All apertures into the room should be carefully closed, and not less than three pounds of sulphur for each thousand feet of air-space should be burned. To secure complete combustion, the sulphur, in powder or small fragments, and moistened with alcohol, should be placed in a shallow iron pan, and this should be placed on bricks in a tub partly filled with water to guard against fire.]

CHAPTER II.

TYPHUS FEVER.

(*Spotted Fever. Ship Fever.*)

Typhus fever is an acute infectious disease, perfectly distinct from typhoid fever, but formerly often confounded with it. The similarity of the two diseases, which led to their similar names, consists only in the grave general condition, with fever, and in a number of complications, which may appear in both. There is, however, an essential difference in the whole course of the diseases, and especially in the circumstance that the intestinal lesion which is characteristic of typhoid is never seen in typhus. The chief distinction between the two affections, which must undoubtedly lie in the difference in their causes, can not yet be demonstrated. We do not yet know the organized pathogenic agents of typhus fever, although it must be presupposed that they exist.

Ætiology.—As to the way in which infection occurs, we have much less information even than in relation to typhoid. We regard it as an established fact that the disease never arises spontaneously, and that its appearance in a place previously free from the disease is always to be referred to an importation of the pathogenic poison. It is likewise determined, through numerous observations, that typhus is one of the contagious diseases—that is, that the specific poison can be directly transferred from the patient to others around him. How it is transferred we have no certain knowledge. Perhaps the poison is contained in the expired air; or, as is still more probable, in the scales of epidermis; or, perhaps, in the other excretions and secretions of the patient. We are equally ignorant through what channel the infectious agent enters the system—whether it is inspired or swallowed. It is certain that the poison may be transferred in the clothes, etc., of the patient (fomites).

Favorable hygienic surroundings decidedly diminish the contagiousness of typhus fever. For example, in the well-ventilated pavilions of the Leipsic hospital there have rarely been cases of transfer of the disease to physicians, nurses, or other patients. On the other hand, if the hygienic influences be unfavorable, typhus fever may appear in very widespread epidemics. Those terrible epidemics which have been described under the names of "famine fever," "camp fever" (Hungertyphus, Kriegstyphus), etc., were for the most part typhus fever. In the smaller epidemics it is often possible to trace the disease to some wretched, overfilled tenement-house.

At present typhus fever appears constantly in Great Britain. Ireland has been notorious for many years as a breeding-place of the disease. It is also frequent in the eastern part of Germany (Posen, East Prussia and West Prussia, Silesia), in Poland, Galicia, Russia, and in parts of southern Europe. The isolated cases which occur every year here and there in central Germany, though more or less numerous, are, almost without exception, to be referred to an importation of the disease.

Typhus fever attacks by preference young adults of twenty to forty years; but it occurs in children, and is comparatively frequent in elderly persons. There is no marked dependence of the epidemics upon any particular season of the year. As in the case of typhoid fever, a person who has once had the disease seems to enjoy immunity from any fresh attack.

[The practical acquaintance of American physicians with typhus fever is, fortunately, limited. Many of the outbreaks which have occurred were traceable to immigrants, especially from Ireland.

During our civil war the disease broke out neither among the armies in the field nor among the prisoners of war. A number of cases were reported at the time, but great doubt has since been thrown upon the correctness of the diagnosis.]

Course and Symptoms of the Disease.—If we try to sketch the characteristic behavior of typhus fever, especially as contrasted with typhoid, we may say that the disease begins much more abruptly and rapidly, and that the fever quickly becomes very high and the general disturbance very severe, but the illness lasts a shorter time, seldom more than two weeks, and generally passes by crisis into recovery.

The length of incubation seems to vary. Murchison thinks it is usually more than nine days. Sometimes, though not invariably, slight prodromata precede by some days the actual outbreak of the disease. These are languor, anorexia, headache, and pain in the limbs. Then the regular illness begins, as a rule, rather suddenly, and often with a pronounced rigor. With this the temperature rises quickly, and may on the very first evening reach 104° or 105° (40°–40·5° C.). Vomiting is not rare, and may be repeated. A grave general condition, with fever, is developed in a few days. The patient feels exhausted.

There is often violent pain in the loins and extremities. Nervous symptoms soon appear: persistent and intense headache, vertigo, spots before the eyes, ringing in the ears, and in many cases quickly increasing stupor and delirium. In severe cases the fever often reaches 106° (41° C.), and may be even higher, and it is almost constant, with but slight morning remissions. The skin is hot and dry, the tongue dry and thickly coated, the respiration moderate, the pulse very rapid. We very frequently find in the chest the signs of an extensive bronchitis. Nasal catarrah and conjunctivitis also occur. Serious intestinal symptoms are generally absent, although there may be slight tympanites or diarrhœa. The spleen is almost always greatly enlarged. Only in a few epidemics is the splenic tumor said to have been wanting (?). The urine is concentrated and scanty, and sometimes has a trace of albumen.

On the third to the seventh day of the disease the characteristic eruption appears. To this the disease owes its name of "spotted fever." The eruption consists of rose-spots, generally very numerous and widespread, upon the trunk and extremities, often also on the face. Sometimes the spots are larger, and may then bear great resemblance to a fresh eruption of measles. The skin between the separate rose-spots is not infrequently diffusely reddened. After two or three days the roseolæ become hæmorrhagic, and change into lighter or darker petechiæ. It is commonly only in the lighter cases that the rose-spots fade away without first becoming petechial. In rare though well-substantiated cases the eruption has been scanty, or even wholly wanting. Herpes does occur, but only seldom.

The fever begins to abate in light cases as early as the second week, coincidently with an improvement in the general symptoms. Often this change is indicated about the seventh day by a considerable remission in the temperature. On the other hand, in severe cases, all the symptoms grow worse. The weakness increases. The nervous derangement reaches the extreme of a severe "typhoid state," expressed either by marked stupor, which sometimes passes into complete coma, or by violent delirium. Lobular pneumonia attacks the lungs, and the fever continues with unabated violence. These symptoms may end with death, but in favorable cases they decline rapidly. Sometimes this decline is preceded by a great rise in temperature (*perturbatio critica*) on the fourteenth to seventeenth day, rarely a few days earlier or later. In such cases the temperature is apt to fall by crisis, sinking in a day or two, with but slight interruption, down to the normal level. Even in those cases in which the descent is by gradations it is always decidedly more abrupt than in typhoid. The eruption quickly fades, the patients gradually improve, and, as a rule, become completely and permanently convalescent. It is true that some observers have seen relapses, but they are, at least in our present epidemics, extremely rare.

Complications and Varieties in the Course of the Disease.—From what we have said of its course, it is evident that the symptoms are essentially those of an intense general infection of the system. The sole demonstrable local lesion which is almost invariably present is the characteristic eruption, and this has evidently no causal relation to the severe symptoms of the disease. It is likewise extremely probable that most of the complications, which not infrequently arise in severe cases, are secondary, and occur in the way already described with considerable detail in the preceding chapter. They are just such complications as are possible in every severe general disease, and embrace otitis, parotitis, extensive lobular pneumonia, more rarely gangrene of the lungs, and pleurisy; also furunculosis, purulent cellulitis, bed-sores, dysentery, icterus, etc. Whether some of the local lesions which are observed may not be direct results of the pathogenic poison, we can not at present decide. Among these would come, first of all, the rare cases of lobar pneumonia and nephritis. Sequelæ are, on the whole, rare, though sometimes there is a tedious anæmic condition, or neuralgia, paralysis, etc.

The separate epidemics of typhus present considerable variety, not only as regards the occurrence of individual complications, but more especially in the general course and character of the cases. For instance, some epidemics are distinguished by the greater frequency of light attacks (*typhus exanthematicus levissimus*, unsuitably termed by some "febricula"). Here the entire attack runs its course in five to eight days. The fever is generally comparatively moderate; there are no severe general symptoms, and complications are exceptional.

Diagnosis.—It may be very difficult for a time to distinguish typhus from typhoid. The following factors are of chief importance: 1. The onset is much more abrupt in typhus than in typhoid, and is often accompanied by a pronounced rigor. 2. In typhus, the nervous disturbances usually appear earlier and are more severe than in typhoid. 3. The rash is seldom so extensive in typhoid as in typhus, and in typhoid it hardly ever becomes petechial. 4. In typhus the pains in the loins and limbs are generally much more pronounced. 5. If we still find it hard to decide, the manner of recovery will almost always settle the question. Recovery in severe cases of typhoid is, on the average, much more tardy and gradual, by lysis. In typhus it occurs generally by the seventeenth day, and by crisis.

The **prognosis** is chiefly determined by the severity of the fever and of the nervous symptoms. Extensive lobular pneumonia is the most frequent dangerous complication. The mortality varies greatly in the separate epidemics. It is sometimes only six or seven per cent., but may rise to twenty per cent.

Treatment is based on the same principles as in typhoid fever. There is no specific remedy. Besides good nursing, a judicious employment of baths is certainly our chief reliance for lessening the severity of many of the symptoms, such as febrile, nervous, and pulmonary disturbances, as well as for averting many dangerous complications. For all details of treatment we may refer to the preceding chapter.

CHAPTER III.

RELAPSING FEVER.

(*Relapsing Typhus—Febris recurrens.*)

Ætiology.—This disease was first named by English pathologists relapsing fever, and by Griesinger *febris recurrens*. It has a peculiar course, made up of separate attacks, and is further of great interest because it is one of the first infectious diseases in which the specific pathogenic organisms became known, and, being easily demonstrable in each separate case, were utilized for the speedy and certain diagnosis of the disease. Obermeier discovered in Berlin, in the year 1873, that in relapsing fever the blood, at certain times, invariably contains peculiar, thread-like micro-organisms. This discovery has since been universally confirmed; and it may be maintained that if once the presence of these "spirilli" be demonstrated in the blood, we are justified in making an absolute diagnosis of relapsing fever.

In Germany the disease did not become epidemic till the year 1868. In 1872 and 1873 there were considerable epidemics in Breslau and Berlin. Its last extensive appearance was in 1879 and 1880, when it spread over most of northern and central Germany, and was accurately studied by numerous observers. People of the poorer classes were almost exclusively attacked, and especially the "tramps." The uncleanly dens where these people lodge were found everywhere to be the chief centers of infection.

The precise manner of infection is as yet almost wholly unknown. All observers agree that the disease is directly contagious; but it can not be very contagious if the hygienic influences be good. At least the results of our late epidemics would imply this. In the Leipsic hospital, where over two hundred and fifty cases were treated, and isolation could not be at all perfectly carried out, not one case of infection occurred. It is certain that the disease can be transmitted by direct inoculation with the blood of patients. This has been established by a Russian physician by the experimental inoculation of healthy persons. Doctors have been repeatedly inoculated at the autopsy of those who have died of relapsing fever. The disease may likewise be transferred by inoculation to monkeys, while other mammals seem to enjoy an immunity from it.

[The first cases of relapsing fever observed in this country were in Irish immigrants coming over in the same vessel in the year 1844. At several periods since then more or less limited outbreaks traceable to immigration have occurred, but the disease has never acquired any foothold with us, and comparatively few physicians have ever seen it. So far as I can learn, only one case has ever been seen in Boston, and that was in the person of a physician from another city, who brought the disease with him and passed through it in the Massachusetts General Hospital.]

Clinical History.—The stage of incubation lasts about five to eight days. It is only exceptionally that some slight prodromal symptoms present themselves just before the outbreak of the disease proper. As a rule, it begins suddenly, with a more or less pronounced chill and intense constitutional symptoms. There are violent headache, great languor, anorexia, and especially marked pains in the loins and extremities. The temperature rises rapidly, reaching generally 106° (41° C.) or higher as early as the first or second day. The skin is hot and dry, and usually quickly assumes a very characteristic dirty-yellowish color. In Leipsic, we often saw herpes labialis, which seems, however, to have been rarer in epidemics elsewhere. The tongue becomes dry and thickly coated. Sometimes there is vomiting. The bowels are constipated, or there is a slight diarrhœa. The spleen becomes rapidly enlarged, being, as a rule, even larger than in typhoid or typhus. The liver is slightly enlarged. The chest presents the signs of a bronchitis, generally moderate, but in exceptional instances severe. The pulse is much quickened. It is seldom that there are severe cerebral symptoms beyond a certain apathy and stupor. We have seen delirium tremens sometimes, in drunkards. A very characteristic symptom, already mentioned, is the marked hyperæsthesia of the muscles, especially in the calves.

After these symptoms, accompanied by persistent and generally very high fever, have lasted five days to a week, there is a critical decline of temperature, with profuse sweating. The patient now improves so rapidly and decidedly that he thinks himself completely cured, and generally gives little credence to the physician's prophecy of a relapse. In rare but well-attested cases there has been really but one attack. The rule is that, after about a week, a second attack occurs, often a third after that, and, infrequently, even a fourth and fifth. In each of these, the above-mentioned symptoms are repeated more or less completely and violently. As the only certain and constant sign of the recurring attacks (the so-called relapses) is a fresh rise of temperature, it will be well to consider their peculiarities at the same time that we describe the course of the fever. During the intervals of normal temperature the other objective symptoms of disease are usually absent, except an evident splenic tumor, and, not infrequently, the peculiar pale-yellow hue.

COURSE OF THE FEVER (see Fig. 3).—The beginning of the fever in the first attack is, as we have said. almost always sudden, so that it may even in a few hours reach a considerable height. The fever lasts, as a rule, five to seven days, but not infrequently as short a time as three or four days. or as long as ten or twelve days. During this time it may keep a tolerably uniform height, but oftener there are considerable remissions, which may even come to deserve the name of pseudo-crises. In such cases the temperature sinks in the morning to normal or even lower, so that we might believe the fever ended; but in the evening the temperature rises again to its former level. These pseudo-crises are most frequent toward the end of the attack, but do occur sometimes in the very first days. The absolute height of the fever is, as a rule, very considerable. Temperatures between 105·5° and 106·5° (41° and 41·5° C.) are very often observed, and in themselves are not especially ominous in relapsing fever. The highest temperature we have observed was 107·9° (42·2° C.). Sometimes the temperature is more moderate (between 102° and 104°, 39° and 40° C.). The fever almost always ends at the close of the attack by crisis, only rarely by a rapid, gradual decline. The crisis is often preceded by an especially great rise the evening before (*perturbatio critica*); so that the subsequent fall of temperature is very considerable. It generally occurs at night, and is accompanied by profuse perspiration. The fall may amount to 9° or 10° (5° to 6° C.). The temperature sinks almost always below normal, often as low as 95° (35° C.) or thereabouts. Once we saw it fall to 92·1° (33·4° C.).

To the first attack succeeds an interval during which there is no fever (apyrexia), which lasts on the average about a week, sometimes a less time, and often a greater. The longest interval we have ever observed lasted seventeen days.

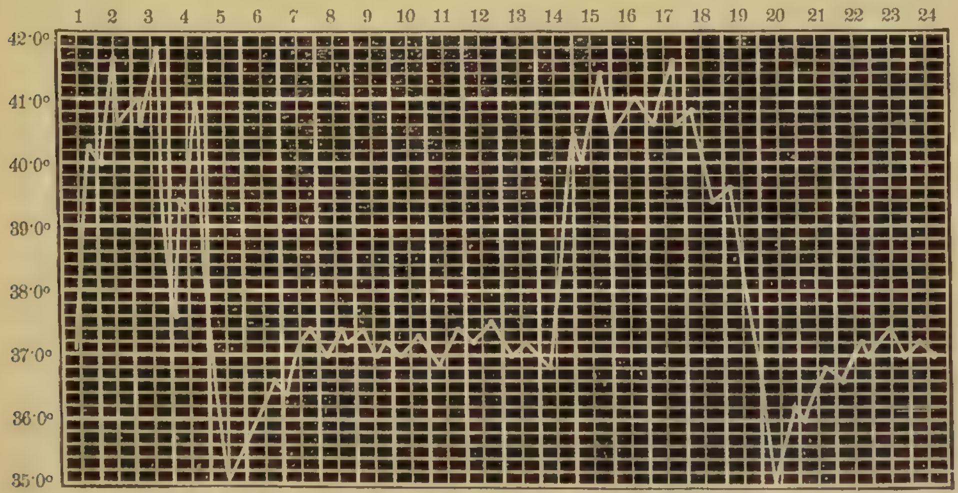

FIG. 3.—Example of the temperature curve in relapsing fever.

During this interval the temperature, which, as a rule, is at first subnormal, rises to normal, and then generally remains there. Exceptionally there are slight evening exacerbations to above 100·5° (38° C.). These may have no demonstrable cause, or may result from some complication, such as otitis, or a furuncle. Then comes another change, and generally a sudden one, ushered in with a chill, and a new rise of temperature, the beginning of the second attack or first relapse. During this attack the fever has the same general peculiarities as in the first attack. Generally the first relapse is briefer by a day or two than the first attack; but the reverse is sometimes true. We will add that we have observed not infrequently a rather high evening temperature (say 101·5°, 38·5° C.) for one or two days before the second attack began, as also before the third.

Relapsing fever seems in many epidemics to have been made up of two attacks, so that not more than one tenth of the cases had a third attack. On the other hand, the majority of the cases in the last epidemic had two relapses. And in these cases the rule was for the interval between the second and third attacks to be one or two days longer than the first apyrexia; but earlier epidemics seem to have had the second apyrexia, if there was one at all, briefer than the first. The third attack is set down in all reported cases as decidedly shorter than either of its predecessors. It lasts generally two or three days. Exceptionally we have seen it persist for four or even six days.

A fourth and even a fifth attack may occur, but only exceptionally. If they do happen, they are usually imperfectly developed, and often are limited to a fever of one day's duration. The more accurately and persistently we take the temperature during convalescence, the oftener do we find slight rises of temperature occurring at intervals late in the history of the case. These are probably to be interpreted as final, rudimentary attacks.

THE SPIRILLI.—The number of cases of relapsing fever in which no spirilli can be demonstrated in the blood, if the examination be accurate, has become so small that it can be disregarded when we compare it with the much greater number of cases where such demonstration is made with ease and certainty. The best way is to get a drop of blood by pricking the skin, and examine it as it is, without mixing anything with it. There is no need of an immersion lens. With a good Hartnack No. 8 [about 330 to 440 diameters, according to eyepiece] the spirilli are seen with perfect distinctness. We have often seen them plainly with a No. 7 [250 to 340 diameters]. It requires a little practice to make them out; but this is easily obtained. Often the attention is first caught by little jogglings and motions of the red blood-corpuscles, and then we see the delicate, narrow threads. Their length equals about three to six times the diameter of the red globules (Fig. 4). They exhibit an active and almost continuous motion, like snakes. Often the whole thread bends upon itself, and then stretches out again. They are partly separate and partly tied up in knots composed of four to twenty individuals. The whole number visible in one field varies greatly in individual cases, and has no direct relation to the severity of the case. Often it requires long searching to find a few, while in other cases there may be twenty or more in the field at once. A very interesting fact is that their appearance in the blood depends upon the attacks of fever. On the first day of the attack we rarely find spirilli, and then only one or two. Upon succeeding days their number increases. Shortly before the end of the attack—that is, before the crisis—they generally disappear entirely; but even after the crisis they have been found, exceptionally and in very small numbers. They have very often been found by the author as well as other observers during the pseudo-crisis described above, so that, after the temperature has become normal, the presence of spirilli makes it very probable that another rise of temperature is impending. The spirilli have thus far been found in the blood only, in the catamenia, in bloody urine, or in blood coughed up from the lungs, and

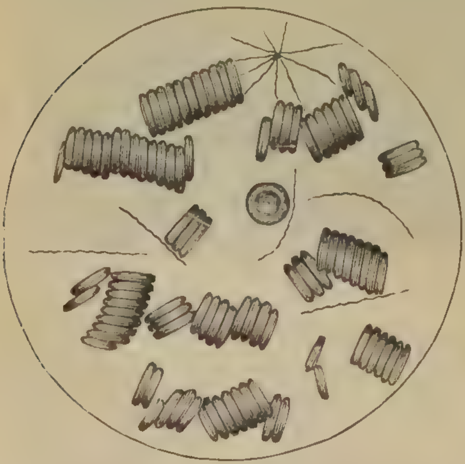

FIG. 4.—Spirilli of relapsing fever in the blood.

never in the organs or secretions (urine, milk, sweat, contents of herpetic vesicles). There can hardly be any doubt that the spirilli which appear in the separate attacks are to be regarded as separate generations. As to their manner and place of development we have as yet no knowledge. In the final, rudimentary attacks, we find few if any. If the patient dies during an attack, they are to be found in the blood after death. Artificial cultivations have not been very successful; nor have pure cultures of them, to our knowledge, ever succeeded. Albrecht states that they will subsequently develop in blood taken from a patient during the interval when he has no fever.

The blood is otherwise modified during relapsing fever. We very often find a slight increase in the white corpuscles. There is often a noticeable abundance of very small bodies, so-called granular elements (Körnchenbildungen). The significance of these (the remains of white corpuscles ?) is still doubtful. There are, finally, peculiar cells, rather large, with fat-granules. They were demonstrated by Ponfick in the venous blood, and are said to come from the spleen. We also find fatty-degenerated endothelium in the blood.

Complications are on the whole rare, and mostly secondary. Important among these are troublesome ophthalmic disturbances, especially iritis and iridochoroiditis. Sometimes parotitis, laryngitis, and pneumonia occur. Epistaxis is a not infrequent complication. It is usually profuse and persistent, and may even be dangerous. Sometimes there has been rather severe dysenteric trouble. In one case which ended fatally we observed a very peculiar intestinal lesion, consisting of hæmorrhagic-necrotic foci in the colon and lower ileum. In severe cases acute hæmorrhagic nephritis occurs with comparative frequency. At the autopsy an important and characteristic phenomenon is the wedge-shaped white spots which occur in the spleen, like infarctions. They have a clinical interest, as they may become the starting-point of pyæmic conditions or of peritonitis. Splenic abscesses have been observed in a few cases.

Variations in the course of the disease occur in this, as in all other acute infectious diseases. First there are mild, abortive cases, in which the attacks are few and brief. Then cases have been described resembling intermittent fever. Of chief importance is that severe variety of relapsing fever first observed in Egypt by Griesinger, and described as "bilious typhoid." There is no longer any doubt about the proper classification of this disease, for spirilli have been proved to appear in it, and it has even been shown that its inoculation upon another person (!) produces a common case of relapsing fever. Bilious typhoid fever occasions successive attacks, like those of relapsing fever. The type is much more severe. As a rule, there appear marked icterus, grave nervous symptoms, hæmorrhages into the skin and mucous membranes, and the termination is frequently fatal. The autopsy shows a greatly enlarged spleen, often containing infarctions and abscesses, and in some cases hepatic abscesses, septic nephritis, and similar lesions.

The **prognosis** of ordinary relapsing fever is on the whole very favorable. In the last epidemics the usual mortality was only two to four per cent. The fatal cases could some of them be laid to wretched nursing In the remaining portion death resulted from complications, such as pneumonia and nephritis.

The **treatment** must as yet be purely symptomatic. Antipyretic treatment is generally needless, since the fever is relatively brief and often quite intermittent. Moreover, most patients can not well endure cold baths, because the muscles are so painful. As a rule, good nursing and proper food amply suffice. If the muscular pains are very violent, we may order chloroform liniment as an embrocation. Complications are to be treated on general principles.

We are not acquainted with any remedy that can influence the disease itself or avert the relapses. Large doses of quinine, salicylic acid, etc., have been frequently employed for this purpose, but never with success. Lately there has been ascribed to calomel a favorable influence upon the general course of the disease, and its use is said to diminish the number of attacks. We must await further evidence in support of this statement.

CHAPTER IV.

SCARLET FEVER.

(*Scarlatina.*)

We now begin the consideration of those acute infectious diseases which are usually embraced under the name of the "acute exanthemata." In this group are reckoned, besides scarlet fever, measles, rötheln, small-pox, varicella, and sometimes also facial erysipelas. The point which these diseases have in common is that in all of them is developed a characteristic eruption, of slight clinical significance in itself, in most cases, but of thoroughly characteristic appearance in each disease, and hence of essential importance in diagnosis. A number of the acute exanthemata have this further point of mutual resemblance that they appear chiefly in children. These diseases are scarlet fever, measles, rötheln, and varicella.

Ætiology.—Infection with the specific scarlatinal poison occurs almost always by contagion, which takes place very readily. A single approach to a patient ill with scarlet fever may suffice to communicate the disease. There is no doubt that the disease may be transferred by objects which the patient has touched, such as linen, clothing, furniture, or toys. Persons who have been with the sick may be the means of transmitting the disease, although themselves unaffected. In England it has been thought that the contagium may be carried by milk.

Numerous observations show that the scarlatinal poison is with great difficulty destroyed, and may keep its contagious powers for months ("tenacity"). We can thus see how difficult, how impossible, it may be in an individual case to point out the source of contagion. Scarlet-fever patients can communicate the disease certainly as late as the end of the desquamative period.

Details as to the manner of contagion, or as to the specific poison itself, are as yet unknown. There have been repeated statements about the presence of bacteria in the blood and in the tissues of scarlatinal patients; but it is very improbable that the specific poison of scarlet fever has been observed. This poison must, however, be contained in the blood, and in the contents of the miliary vesicles, of scarlet-fever patients, for the disease has repeatedly been artificially produced in healthy persons by inoculation with these fluids.

Predisposition to scarlet fever is far less universal than is predisposition to measles or small-pox. In families with several children often only one or two fall sick, while the rest escape, although equally exposed. As age increases, liability to the disease greatly diminishes, although there are cases of scarlet fever among adults. The majority of patients are between two and ten years of age. Scarlet fever is rare during the first year of life. It is an interesting fact that children with fresh wounds, either accidental or surgical, are especially liable to scarlet fever. An analogous and familiar fact is that women after delivery have a strong tendency to the disease.* With very few exceptions a person is attacked but once, so that, after the disease is over, an immunity from contagion is enjoyed; but there are exceptions to this rule.

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* In puerperal cases genuine scarlet fever and septic diseases were formerly often confounded. (See Chapter XVIII.)

Scarlet fever is now spread over the entire globe. Here in Germany there are almost always some sporadic cases in the larger towns, while from time to time, especially in autumn, there are more or less extensive epidemics in one place or another. There is considerable variation in the different epidemics of scarlet fever, as in many other infectious diseases, in the general character of the disease, and above all in the prevailing mildness or severity of the cases.

Clinical History.—The period of incubation is about four to seven days, or is sometimes apparently shorter. There are hardly ever any decided prodromata The disease begins rather suddenly, with fever, often introduced by chilliness, and sometimes by a well-marked rigor. There is almost invariably a painful, scarlatinal sore throat. A further symptom, in all cases of any severity, is cerebral disturbance, generally rather intense. There may be headache, dullness, uneasy sleep, delirium, and, in smaller children, sometimes even convulsions. A very frequent and characteristic early symptom is vomiting, which may be repeated.

The characteristic rash usually appears as early as the close of the first day, or on the second, and begins on the neck and on the chest and face, soon becoming almost universal. The eruption consists at first of numberless small red points, crowded thickly together and soon united into a diffuse, intense, scarlet-colored erythema. The small and somewhat elevated points almost always correspond to the swollen hair-follicles. The diffuse redness is the result of an excessive hyperæmia of the skin, and vanishes completely on pressure. The back usually presents the most vivid tint. In the face there is generally pallor of the lips and chin, presenting a very striking and characteristic contrast to the bright-red cheeks. If some object like the end of a pen-holder be drawn over the red skin, there soon arise corresponding white lines, due to contraction of the blood-vessels. It is possible thus to make letters or pictures upon the back of the patient. We should add, however, that this is not a peculiarity of the scarlatinal eruption, being seen in other erythematous eruptions.

The rash persists for some three or four days, at first even increasing somewhat in vividness. It often appears more intense by artificial light than in the daytime. Meanwhile the severe general symptoms continue—the fever, the usually excessively rapid pulse, the cerebral symptoms, and the throat trouble. The spleen is often somewhat swollen, though seldom very large. Then the eruption begins to fade, the fever gradually ceases by lysis, the general condition and the difficulty in swallowing improve. With the end of the first week or the beginning of the second, the cases which run the typical course become fully convalescent. When the rash disappears, the epidermis usually begins to peel off, in a very characteristic way, in pieces of considerable size. The exfoliation upon the hands and feet is especially pronounced, and the little convalescents often amuse themselves by peeling off the epidermis in strips. Cases which are apparently the mildest and most benign may have their convalescence interrupted by the occurrence of a secondary scarlatinal nephritis. There is no certain prophylaxis against this.

We will now pass on from this general summary to a more complete consideration in detail of general and local symptoms. And we shall see how manifold are the clinical phenomena presented by scarlet fever.

1. *Fever* (see Fig. 5).—Although in a few undeveloped cases there is no fever, or scarcely any, almost all cases of any importance have high fever. It is only exceptionally that severe cases are observed where the bodily temperature is little if at all elevated. As a rule, the fever rises rapidly upon the very first day, corresponding to the sudden onset of all the symptoms, to about 104° or 105° (40°–40·5° C.). The next day it often becomes a little higher still, and then persists

with but slight variations, as a rule, so long as the eruption is at its height. During this period a temperature of 105° or more (40·5°–41° C.) is not infrequently observed. When the eruption fades, and the other symptoms decline, defervescence occurs. This happens but rarely by crisis, and that in the slight attacks. It is almost always by prolonged lysis, as in typhoid, only more irregularly and more rapidly. If the fever lasts into the second week of the disease, it is almost always (though not without exceptions) caused by demonstrable complications. The most frequent causes are the persistence of a severe sore throat, the occurrence of inflammatory changes in the cervical glands, or a purulent otitis media. In closing what we have to say about the fever in this disease, we would emphasize the fact that the pulse is often very rapid, even considering the height of the temperature.

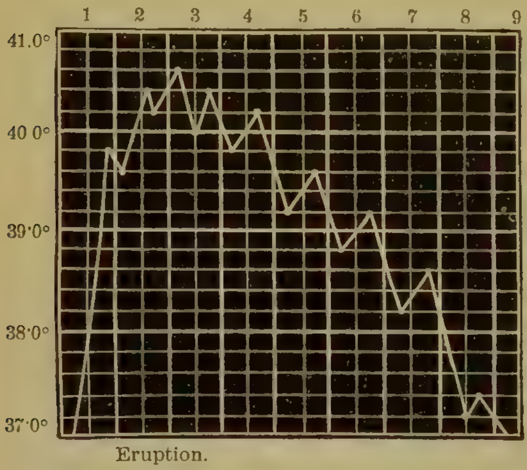

Fig. 5.—Example of a normal scarlet-fever curve.

2. *The Throat.*—The throat presents the most constant local lesion of scarlet fever. Sore throat is only in the rarest cases wholly absent; but its form and intensity may vary extremely. The mildest variety is a simple, erythematous catarrh, without much swelling, but exhibiting a more or less intense reddening of the soft palate and tonsils, frequently associated with enlargement of the little mucous follicles. Sometimes minute hæmorrhages take place into the mucous membrane. In other cases the scarlatinal affection of the throat is from the start associated with considerable swelling of the parts, and especially of the tonsils, justifying the term "parenchymatous sore throat." Not infrequently small abscesses form in the lacunæ of the tonsils or superficial spots of necrosis develop which leave behind them larger or smaller ulcers, and sometimes occasion considerable hæmorrhage. There may even be a circumscribed gangrene of the tonsils.

By far the most important, because it is also the most dangerous, of the affections of the throat resulting from scarlet fever is the so-called scarlatinal diphtheria—that is, a diphtheritic inflammation of the tonsils and soft palate. This usually develops on the third, fourth, or fifth day of the disease, replacing a simple inflammatory condition of the parts. Whitish, dirty-colored spots develop on the tonsils, the arches of the palate, and the uvula. These rapidly increase in size, and cause a dry necrosis of the mucous membrane and subsequent ulceration. The process is a truly diphtheritic one—that is, there is an inflammation combined with an extension into the diseased tissues of a fibrinous exudation.

It is especially characteristic of scarlatinal diphtheria that there is almost invariably a considerable swelling of the cervical lymph-glands, except in those cases which die very quickly. It is true that the glands are usually somewhat enlarged in the milder forms of pharyngitis accompanying scarlet fever, but they seldom attain the size observed in the true diphtheritic process. In this there is an inflammatory and œdematous infiltration affecting often not only the glands themselves, but also the surrounding connective tissue, so that in severe cases the whole cervical region and the floor of the buccal cavity presents a firm and usually a very painful enlargement. It should be added that the severity of the throat symptoms and the extent of the glandular swelling are not always commensurate. Almost always the scarlatinal diphtheria is associated with a marked stomatitis, and very often also with a severe purulent or even diphtheritic rhinitis. At the alæ of the nose and the corners of the mouth there are often superficial ulcers from this cause. Otitis is also a frequent complication of scarlatina (*vide infra*). The swelling of the lymph glands in the throat very often terminates in suppuration. The influence upon the general condition of the patient of the scarlatinal diphtheria is apt to be considerable, apart from the marked local discomfort. The constitutional symptoms are often as severe as in septicæmia or pyæmia. Many cases end fatally in a few days, while others pursue a more tedious course, lasting perhaps several weeks before death comes. These are often associated with pyæmic processes in other parts of the body.

With regard to the pathogenesis of the throat troubles seen in scarlet fever, the more simple forms are in all probability directly associated with the scarlatinal process—that is, they are direct sequences of the affection. As relates, however, to the severer forms, and in especial the diphtheritic variety, it is at least very probable that these are not a direct result of the scarlatinal poison, but are due to some secondary infection which takes place at the spot of the primary scarlatinal angina. But we are almost obliged to suppose that there is some intimate connection between the two varieties of infection, else why should this peculiar secondary disease, the scarlatinal diphtheria, be so often conjoined with scarlet fever? Yet the fact deserves emphasis that the diphtheria seen in scarlet fever has no ætiological connection with genuine primary diphtheria (see the chapter on this latter disease), although the pathological anatomy is almost the same in both diseases. From a clinical standpoint the two present several important points of difference. In particular the scarlatinal diphtheria, in contrast with the primary form of the disease, seldom spreads to the larynx. Laryngeal croup is therefore but rarely seen in scarlet fever, and perhaps when it does occur it is to be explained by an actual complication of the scarlatina with genuine diphtheria. The severe dyspnœa which sometimes develops in the course of scarlet fever is probably caused by an inflammatory œdema of the glottis. Another important clinical distinction between genuine and scarlatinal diphtheria is that paralysis of the soft palate, the ocular muscles, and other parts is scarcely ever a sequel of scarlatinal diphtheria.

The definitive answer to the question of the primary and the secondary varieties of infection in scarlet fever must be postponed until the individual germs corresponding to the same are accurately known. At present we have but a single noteworthy item of information, which is that there is present in scarlatinal diphtheria a peculiar "chain-forming micrococcus," discovered by Löffler in the necrotic mucous membrane, the lymph-glands, and even in the blood. This, perhaps, is the occasion of the secondary disease.

It may be added that, in puerperal scarlet fever, affections of the throat are noticeably infrequent or trivial, but the same question arises whether here scarlet fever and other diseases have not been confounded.

In addition to the redness there is often a variable degree of swelling. These latter cases form a connecting link between the ordinary variety and those cases of parenchymatous sore throat where the tonsils are intensely swollen and the soft palate and uvula are more or less œdematous. There may be small suppurating spots in the crypts of the tonsils, or these organs may suffer from larger abscesses, necrosis, or even gangrene. When the necrosed portions slough off, there may in rare cases occur a considerable hæmorrhage from the tonsils. Often chronic hypertrophy of the tonsils remains after these severer forms of inflammation.

These graver varieties are almost always accompanied by swelling of the submaxillary lymph-glands. The neighboring connective tissue will then often present diffuse infiltration and œdematous swelling. This swelling may in severe cases involve the floor of the mouth and the entire neighborhood of the throat. The severity of the trouble in the throat does not always correspond to that in the lymph-glands. Very frequently the swelling of the lymph-glands and the neighboring structures ends in the formation of abscesses.

The croupous and diphtheritic inflammations of the throat are the most important and justly the most feared. We believe that it is a mistake to speak of a "complication of scarlatina with diphtheria." The diphtheria of scarlet fever has, from an ætiological point of view, no relation to the common genuine diphtheria. It is a throat trouble having a direct connection with the scarlatinal process as such. It can, indeed, from its outward appearance,* be distinguished with difficulty, if at all, from the primary, genuine variety (*q. v.*); and therefore, from an anatomical point of view, it must be termed a croupous or diphtheritic inflammation.

Scarlatinal diphtheria may be united with any of the above varieties of angina, either appearing at the very beginning of the illness, especially in very severe cases, or not till later, at the end of the first or even in the beginning of the second week. It is almost always the sign of a severe attack, and is therefore generally associated with high fever and grave general symptoms. The secondary swelling of the cervical lymphatic glands and the surrounding connective tissue generally attains a severe grade, and it is often very painful. Here, as in the other varieties of severe angina, there is almost always a simultaneous, intense stomatitis, and frequently a purulent coryza. There are often superficial ulcers on the alæ nasi and at the corners of the mouth. This form of diphtheria has one peculiarity of great interest and clinical importance. Unlike primary diphtheria, it seldom extends to the larynx, so that it is only in rare cases of scarlet fever that there are symptoms of laryngeal croup. A further important clinical difference between these two forms of diphtheria is that in scarlet fever there is scarcely ever any subsequent paralysis of the soft palate or of the muscles of the eye. A dangerous, but fortunately a rare, complication is œdema of the glottis. This may quickly cause death from suffocation. We may finally mention that puerperal scarlatina is said to have in many cases extremely slight throat complications, or none whatever.

3. We proceed by a natural sequence to the consideration of the affections of certain parts adjacent to the throat, troubles which must be regarded as chiefly the result of direct extension, or of a conveyance of the inflammatory process from the throat.

The stomatitis we have already mentioned, as well as the disturbance in the neighboring lymph-glands † and the surrounding tissue. Parotitis is not rare in severe cases. Of especial importance is the scarlatinal inflammation of the middle ear, which often results in permanent deafness.

This inflammation usually arises at the time of desquamation, but it may occur earlier. It is either a simple catarrh of the middle ear, or, in severe cases, an actual diphtheritic process. The deafness and earache are easily overlooked, as the other symptoms of the patient occupy the attention, so that the ear trouble is first recognized by the occurrence of perforation of the tympanum and subsequent purulent otorrhœa. After this declines there very often remains behind, as we have said, permanent deafness. Statistics have shown that four or five per cent. of all cases of deafness are referable to an attack of scarlet fever in childhood. The otitis is seldom immediately dangerous, but yet cases of purulent meningitis have been observed to follow it.

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* In microscopic preparations, however, according to Heubner's investigations, there are evident differences between genuine primary diphtheria and that of scarlet fever.

† It should be remarked that not infrequently there is also in scarlet fever a slight universal swelling of the lymph-glands (back of the neck, axilla, groins, etc.).

We have already spoken of the purulent or even diphtheritic rhinitis which almost always accompanies the scarlatinal sore throat. In rare cases there may also occur a purulent conjunctivitis, which is most probably the result of a direct conveyance of inflammatory secretions.

The tongue in scarlet fever deserves special mention. The first coating cleans off, and then the tongue usually presents a very characteristic appearance. It is diffusely reddened and covered with little elevations corresponding to swollen papillæ (strawberry tongue, scarlatinal tongue).

4. The characteristic eruption, as developed in the great majority of cases, has been described above. It remains to describe certain variations from the usual appearances.

First, the eruption may be rudimentary. It is then not pronounced, and visible only on a limited portion of the body (face, trunk, or extremities).

Variations from the type are not rare; sometimes the papules are more strongly developed (*scarlatina papulosa*); very frequently there are little vesicles (*scarlatina miliaris*). This latter form of the eruption appears by preference upon the trunk, but it also may come upon the extremities, and is often brought out by excessive perspiration, or by wrapping up the patient too warmly. Many epidemics are noticeable from the frequent appearance of this miliary form. More rarely the rash has a spotted look, resembling the eruption of measles (*scarlatina variegata*). There may be minute ecchymoses, which are not ominous. Well-developed cases of hæmorrhagic scarlatina are, however, very dangerous, because here the general infection of the system is almost always exceedingly severe; and there is besides, as a rule, a general hæmorrhagic diathesis. Other cutaneous lesions, especially herpes and urticaria, are not so very unusual in connection with the scarlatinal eruption. Furunculosis has been repeatedly observed after the rash fades.

Desquamation generally begins as soon as the rash has completely disappeared, but may not occur till a few days or even one or two weeks later. Its extent corresponds in general to the severity of the eruption, although extensive desquamation may follow a rudimentary eruption. It is seldom bran-like or furfuraceous, as in measles. The rule is for it to be in lamellæ, so that, as we have stated, quite large strips of epidermis may be detached entire.

In rare cases an œdema of the skin appears after scarlet fever, which can not be shown to depend upon nephritis (*vide infra*), but may perhaps be due to an abnormal permeability of the walls of the cutaneous blood-vessels following the eruption (*hydrops scarlatinosus sine nephritide*).

Kidneys.—Next to the severer forms of throat trouble, the most important and dangerous complications are located in the kidneys. They may appear as early as the acme of the disease, as in many other infectious diseases. The urine has a trace of albumen. In rare cases the amount of albumen may be considerable. The appearance of the urine is generally not much changed, and the microscope reveals but few abnormal constituents. There are some white and red blood-globules, a few hyaline casts, sometimes one or two renal epithelial cells. This initial albuminuria very rarely gives cause for alarm.

The genuine scarlatinal nephritis scarcely ever develops much before the end of the second or the beginning of the third week. Sometimes it comes even later. In one case under our own observation it did not begin till the thirty-third day of the disease. It may therefore be regarded to a certain degree as a localized relapse. It may be so mild as to cause no subjective symptoms whatever, and would be unnoticed if the urine were not carefully examined. On the other hand, it may be accompanied by the gravest symptoms, and soon terminate fatally. It may follow either severe cases or the mildest, so that the rule should be to examine the urine in every case of convalescence from scarlet fever as often and as accurately as possible. No exact statement can be made as to the frequency of this complication, for it is much more common in some epidemics than in others.

The development of nephritis is often marked by a fresh rise of temperature. The elevation may be slight or it may reach 104° (40° C.). According to our own experience, the fever often comes a day or two earlier than the changes in the urine. As the nephritis goes on, it is very often accompanied by a moderate fever with remissions. This fever may be almost wholly absent, especially in mild cases. The pulse generally becomes harder, and is sometimes quickened; but in many cases it will be slow, and it is sometimes irregular. Among other objective symptoms, the first to excite notice is generally a slight puffiness of the face, which is usually pale. The eyelids, particularly, present an evident œdema. In the milder cases this œdema remains limited, while in others it gradually increases in extent and degree, involving first, as a rule, the dependent parts of the trunk, and later the extremities. Severe cases develop a pronounced anasarca. There are then, usually, effusions into the serous cavities, especially ascites and hydrothorax. The hydrothorax is frequently combined with severe bronchitis, and then may occasion extreme dyspnœa.

The urine exhibits the most important changes. These may be insignificant in the milder cases, but are very pronounced in the severe ones. The amount is much diminished. Sometimes there will be for several days almost complete anuria. In cases of any severity the urine is turbid, dark, often evidently bloody, with increased specific gravity (about 1015 to 1025), and containing a large amount of albumen. The sediment is generally abundant, and exhibits numerous hyaline casts of various lengths and diameters. To these may be attached red or white blood-corpuscles, detritus, granules of hæmatoidin, or bacteria. In cases of some duration the casts are often moderately fatty. Very frequently there are found noticeably long and broad waxy casts, which are opaque and yellow. In many cases of scarlatinal nephritis the urine is peculiar in having very many white blood-corpuscles, either isolated or adhering to the casts. These undoubtedly originate for the most part in the kidneys. Red globules, some of them in the form of colorless rings, are found. They are usually present in small numbers, but may become more abundant, especially for a day at a time. Renal epithelium is frequently seen, but not invariably nor in very large amount. It must be mentioned, in conclusion, that in some rare instances the autopsy discloses quite a marked nephritis, although the urine was apparently normal during life, or at least was not very abnormal.

Uræmic symptoms are not infrequent. They may be of all degrees of severity. They will be described in detail under diseases of the kidney (*vide infra*). The uræmia may be so severe as to cause convulsions, coma, and death; but it is remarkable with what frequency children recover from what seems to be the most pronounced uræmia.

The duration of scarlatinal nephritis varies greatly according to its severity. In cases which run a favorable course, the urine is generally abnormal for two to four weeks, or even longer. Death may be due either to uræmia or to dyspnœa. The latter cause is the more frequent one, and may depend upon the ascites and hydrothorax, or upon pneumonia (*vide infra*). Sometimes death comes from cardiac failure, which may now and then be very suddenly developed. The nephritis may go on into chronic renal disease, but this is rare.

Pathologically, the kidneys present, in a more or less pronounced degree, the lesions of ordinary acute hæmorrhagic nephritis (*vide infra*). It is sometimes astonishing to see how apparently insignificant the lesions are, in spite of the grave clinical symptoms. In such cases there is usually a so-called glomerulonephritis (Klebs), in which the lesions are chiefly confined to the walls of the capillaries and to the epithelium of the glomeruli. If the nephritis has been of some weeks' duration, we generally find that well-marked hypertrophy of the left ventricle has already developed, as was first pointed out by Friedländer. We have ourselves observed it, and have even been able to demonstrate it repeatedly during life.

6. *Joints.*—When desquamation begins, or even earlier, pain and swelling may attack a certain number of the joints. This trouble was formerly called scarlatinal rheumatism, but now is usually known as scarlatinal synovitis. It is generally mild and quite temporary. The articular inflammation may, however, be severe and even purulent. This is usually a part of a general pyæmia, as evinced by such other lesions as empyema and subcutaneous abscesses; and they all seem to be caused by the above-mentioned "chain-forming" micrococcus, which swarms in the pus found in all the affected parts.

We have seen a few instances of excessive pain in the muscles of the thighs, accompanied by a moderate, diffuse swelling.

7. Another important complication of scarlet fever is pneumonia. In severe cases lobular pneumonia sometimes appears as early as the first stage of the disease; but it occurs more frequently in connection with the nephritis. The respiration may be very seriously interfered with by it. Inflammations of serous membranes in the chest—viz., endocarditis, pericarditis, and pleurisy—are more rare. They may or may not be accompanied by disturbances in the joints (*vide supra*). Quite severe intestinal symptoms, such as diarrhœa, may appear. These are generally the result of a catarrhal inflammation of the intestinal follicles. Dysentery is less frequent. The enlargement of the spleen has been already mentioned. The liver is also sometimes found to be considerably enlarged.

Variations in the Course of the Disease.—The diversities of the clinical picture in different cases of scarlatina will be understood when we consider the variety and number of the disturbances thus far cited. It is to be added that the general course of the disease may exhibit numerous peculiarities, of which it is hardly possible to give an exhaustive presentation. We will content ourselves with a cursory statement of the most important deviations from the typical course.

1. *Rudimentary Forms.*—To this class, in which the disease does not reach a perfect development, belong first the cases of simple sore throat with no eruption, or at most an extremely faint and partial one (*scarlatina sine exanthemate*). Sometimes even the sore throat is hardly to be seen, and there is nothing but a brief and slight fever with mild symptoms of general disturbance. The recognition of these cases as scarlatinal is possible only when we consider their ætiological relation to other undoubted cases of scarlet fever. We had an excellent opportunity to observe them when the disease broke out in the children's wards of the hospital at Leipsic. The diagnosis is sometimes confirmed by a slight though evident desquamation, affecting the hands, feet, legs, and back, or by an acute nephritis, which may follow the mildest attacks of this sort. Many cases of acute nephritis, though apparently wholly spontaneous and primary, must be regarded as ætiologically scarlatinal.

2. *Rudimentary but Pernicious Forms.*—Under this head belong those attacks of scarlet fever where the eruption is scanty or absent, while from the very start the most violent general symptoms appear. There is a very high fever, enormously

rapid pulse, and delirium. Such cases must be the result of an uncommonly severe general infection. They usually end in speedy death. Other cases, ending fatally in a few days, have a well-developed rash without other localized disturbances.

3. *Severe Forms with a more Protracted Course.*—In these cases the long duration is not the exclusive result of especial complications, but is likewise due to the severity of the intoxication. One variety is the so-called typhoid form of scarlatina, with persistent high fever and severe constitutional symptoms. Another variety is the hæmorrhagic form briefly mentioned above, in which there are extensive hæmorrhages into the skin and into the mucous and serous membranes. This form may run an extremely acute course. Further, in all pernicious forms, there may be severe local complications, particularly diphtheritic or gangrenous sore throat, inflammations of serous membranes, etc. Attacks of this sort are often not produced by the poison of scarlet fever alone, but by secondary complicating processes. While speaking of scarlatinal diphtheria it has already been pointed out that secondary infection of the body may result, principally originating from the diseased throat, and occasioning sometimes a grave septic condition of the whole body, sometimes local disease of distinct parts.

4. In extremely rare cases relapses do occur. After the first illness a fresh eruption breaks out with all the other symptoms of scarlet fever. In anomalous cases, running a severe course, there is sometimes, at an advanced stage, a fresh, imperfect eruption (generally in spots), which Thomas has termed a pseudo-relapse.

Diagnosis.—The diagnosis of scarlet fever is made in most cases from the characteristic eruption taken in connection with the other symptoms. We should, however, bear in mind that exceptionally other eruptions appear which exhibit the closest resemblance to that of scarlet fever. 1. After the use of certain drugs, especially atropine (belladonna), quinine, antipyrine, morphine, chloral ; and likewise after the ingestion of crabs, fish, etc. 2. As a symptom of other infectious diseases, such as typhoid fever, small-pox; and, above all, in septic diseases (*vide infra*). In an anomalous case factors of importance for diagnosis are the ætiology and the occurrence of desquamation or of a secondary nephritis.

The **prognosis** must in every case be guarded. From what has been said of the course of the disease, it is evident that, even in cases which are at first apparently the most favorable, dangerous complications may appear later, particularly nephritis.

Treatment.—The majority of those cases of scarlet fever which take a typical course will recover completely without our aid. In these the task of the physician, so far as treatment is concerned, consists in arranging the details of hygiene and the general care of the patient. The sick-room should be cool, and the diet rather strict, consisting mainly of milk. Broths and eggs may also be allowed. We should see that the skin and the mouth are kept clean. To change the linen frequently, if done with proper precaution, is not only permissible, but very desirable. The favorite practice of rubbing the skin with fat pork has some merit, and is especially to be recommended if the skin be harsh and dry after the eruption has faded.

[From the moment that the disease is declared the patient should be thoroughly anointed daily with carbolized vaseline, lard, or the like; and this should be kept up until desquamation has ceased. Not only is the comfort of the patient promoted, but the danger of the spread of the infection is thereby greatly lessened.]

The scarlatinal disease of the throat must be treated with the greatest attention, the main duty of the physician in this regard being to prevent, if possible, the ingress of the above-mentioned secondary infection. It is therefore our opinion that in every case of scarlet fever the greatest pains should be taken from the very commencement of the disease to maintain complete disinfection of the mouth and throat. Larger children may use a gargle (two-per-cent. solution of chlorate of potash, one- or two-per-cent. solution of carbolic acid). Inhalation of carbolic-acid spray is also to be recommended where practicable. If there is prostration, or if the child be young or willful, we must cleanse the mouth and throat at short intervals, by means of a spray-apparatus, with disinfectants, such as carbolic acid or permanganate of potash in solution. Sometimes it is a good plan to let the patient swallow slowly a half-teaspoonful of a solution of potassic chlorate (about 1 to 40) every half-hour or oftener, with the object of contributing to the local disinfection of the throat. If scarlatinal diphtheria nevertheless develops and the cervical lymph-glands begin to increase further in size, there is reason to hope, according to the experience of Taube and Heubner, that parenchymatous injections into the tissue of the tonsils or the palatine arches may yet check the spread of the secondary infection. About 6 minims (a Pravaz syringe half full) of a three-per-cent. solution of carbolic acid may be injected twice daily upon each side by means of a long hollow needle and a subcutaneous syringe.

If the nose be likewise affected, the chief thing to do is frequent cleansing and syringing while the head is bent forward. We should be on the watch for the possible occurrence of otitis. In this particular the physician is often guilty of sins of omission. Much harm may be averted by a prompt cleansing of the ears, or, if need be, by insufflation of air into the middle ear, or paracentesis of the membrana tympani.

Inflammation of the glands in the neck, if severe, is prone to pass on to suppuration, and must then be treated surgically. When the swelling has just begun, or is still moderate, we may try to cure it by rubbing in iodoform ointment (1 to 15) two or three times a day. Ice is generally not so well borne as warm applications (poultices or warm bran-cushions).

If there be continuous high fever, accompanied by rather severe constitutional symptoms, a moderate employment of the cold-water treatment is strongly to be recommended. The baths seldom need to be cooler than 81° to 88° (22°–25° R.), and are to be employed two or three times daily, or oftener in severe cases. If the nervous disturbance be serious, or if the respiration be impaired, the patient should be douched with cold water during the bath. At the same time wine or strong coffee is to be given as a stimulant, or, if cardiac failure and signs of collapse appear, the best remedy is subcutaneous injections of camphor. We are convinced that internal antipyretics, such as antipyrine, may usually be dispensed with, although in private practice we may be obliged to employ them.

If the pulse is abnormally rapid, and there is danger of cardiac failure, we can employ, besides stimulants, an ice-bag placed over the heart. Digitalis may also be tried cautiously.

The scarlatinal inflammation of the joints is sometimes improved by salicylate of soda (forty-five to sixty grains, grm. 3 to 4, in one dose [!]). Sometimes, however, this remedy has failed us.

We know of no means to avert the nephritis. In justice to himself, the physician must always at the start point out the possibility of its occurrence, and must avoid as far as possible errors in diet or exposure to cold on the part of his patient. He may thus escape blame. For the treatment of the nephritis and its results, see the section on renal diseases. We must likewise refer the reader to the appropriate chapters for the treatment of other possible complications of scarlet fever.

The patient must, as a rule, keep his bed three or four weeks, even if convalescence be uninterrupted.

[This injunction is rather extreme. Nephritis is as likely to follow a mild as a severe case, and occurs sometimes in spite of every precaution. The physician should use his discretion as to the length of time the patient is kept in bed, carefully guarding against exposure to cold and imprudence in diet.]

The disease is so dangerous that, whenever a case occurs in a family, isolation is absolutely demanded, and, if possible, all the other children should be sent away. If this advice be disregarded, we can reject all responsibility for any further cases and their results.

[Scarlet fever is a disease at once so highly contagious and so common that it may be taken as the type of its class. Its hygienic treatment and the measures needful to prevent its spread consequently deserve more minute detail.

The sick-room should be at the top of the house, if possible, and exposed to the south; every unnecessary article of furniture and all ornaments should be removed beforehand, carpets, curtains, and stuffed or upholstered furniture being included. A window should be kept open constantly, top and bottom; in cool weather a fire should be burning; in warm weather ventilation is furthered by placing a gas-burner or large kerosene lamp near the throat of the chimney. Outside the door of the sick-room a sheet moistened with a disinfectant solution should be carefully hung. Only those whose presence is absolutely necessary are to be allowed in the sick-room, and the physician, when his visit is completed, should pass directly out of the house.

A convalescent should be kept away from all who are liable to contract or convey the disease until desquamation has entirely ceased. Several warm soap-baths should be given before the child emerges into every-day life, and it should finally be dressed in uncontaminated clothing.

For further directions as to the disinfection of the room, the clothing, and the excreta, see pages 26, 27.]

CHAPTER V.

MEASLES.

(*Morbilli.*)

Ætiology.—In contrast with the malignancy of scarlet fever is the comparatively benign nature of measles, a disease of childhood which is but little feared even by mothers. It is so wide-spread, and the susceptibility to it is so universal, that measles passes for an almost unavoidable but comparatively insignificant annoyance. Indeed, few escape it; and probably the reason that adults have measles so much less frequently than children is simply that most adults have already suffered from it in childhood. A second attack of measles in the same individual may occur, but it is certainly extremely rare.

[In highly civilized countries measles has prevailed so long that it would seem that a relative resistance against the poison has been acquired. The frightful ravages of the disease when it was planted in virgin soil, as among the Fiji Islanders not many years ago, apparently bear out this view. The susceptibility to measles is greater and more widespread than is that to scarlet fever—that is to say, fewer individuals reach adult life without having experienced an attack of the former than of the latter.]

Measles generally comes in epidemics. Sporadic cases are exceptional. In this respect measles differs decidedly from scarlet fever. The rapid spread of the disease when it has once broken out is a result of its great contagiousness. If one child in a family is attacked, the others almost always take the disease. The infection may be transferred even by well people and by means of articles with which the sick have come in contact. We are not yet acquainted with the specific poison of measles, although its existence is to be taken for granted, nor with the details of its transmission. Still it seems most probable that the poison is inhaled through the mouth and nose, and that this is the reason why its effects are usually first developed in the respiratory passages (*vide infra*). The disease can be artificially produced by inoculation of healthy children with the blood or liquid secretions of those suffering from it.

Clinical History.—The length of the stage of incubation is tolerably uniform. It is ten days to the beginning of the first symptoms, and thirteen or fourteen days to the breaking out of the eruption. These figures have been established by the observations of Panum, the opportunity having been afforded upon the first introduction of the disease into the Faroe Islands. As a rule, there are no especial prodromata during the period of incubation except some slight elevations of temperature. At the end of ten days the initial stage* begins, generally suddenly, and with an abrupt rise of temperature to 102° or 104° (39°–40° C.). At the same time the characteristic catarrhal symptoms appear: nasal catarrh (coryza), to be recognized by the abundant nasal secretion, the frequent sneezing, sometimes also by nose-bleed; more or less severe conjunctivitis, recognizable by the photophobia, the reddening of the eyes, and the increased flow of tears; and, lastly, symptoms of a catarrh of the upper part of the respiratory tract, usually moderate, but nevertheless causing hoarseness and a slight cough. With all this the general condition is disturbed, the children are restless, have headache, and eat little. Symptoms of a mild sore throat are not infrequent, but are very far from being so prominent as in scarlet fever.

These initial symptoms last, as we have said, three or four days. Then the eruption begins (stage of eruption). This is very often preceded for a day or two by a peculiar, usually spotted, reddening of the hard and soft palates, termed "eruption upon the mucous membrane." The true eruption of measles begins almost always in the face, on the cheeks, forehead, and around the mouth (contrasting with the characteristic pallor of the chin in scarlet fever), and spreads from there rapidly downward over all the rest of the body. The eruption consists at first of little papillæ, corresponding to the follicles. These are soon surrounded by a pale-red, slightly elevated border, and in many cases become confluent. Perfectly flat elevations, of various sizes and of extremely irregular, dentated, roundish, or angular shape, develop. These are often so thickly crowded together as to touch one another, but usually limited portions of normal skin intervene between them. Within each raised spot the little follicular papillæ remain visible.

With the beginning of the eruption the fever rises, having been, as a rule, slight during the last days of the initial stage. It attains about 104° or 105° (40°–40·5° C.). In thirty-six to forty-eight hours the eruption reaches its full development and its greatest extent. The fever and the catarrhal symptoms also persist for the same length of time. Sometimes we find a slight swelling of all the lymph-glands. Then follows a decline of the fever, usually rapid, and indeed almost by crisis, while the eruption after a short period of full development begins gradually to fade during the two or three days following. At the same time the catarrhal symptoms diminish. A more or less extensive desquamation of the epidermis begins, scarcely ever in large pieces as in scarlet fever, but in little scales, "like bran." After eight or ten days, if the disease runs a normal course, the patient is fully convalescent.

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* We consider the term "initial stage" more correct than "prodromal stage." The "prodromal symptoms" are the first slight symptoms which occur during the time of incubation of an infectious disease, while the symptoms presented by measles before the breaking out of the eruption are a part of the already developed disease.

After this brief description of the usual course, we must consider more closely some of the symptoms and possible complications.

The fever (see Fig. 6) of measles exhibits, as has been already implied, a tolerably typical course. It begins with a rather marked and rapid rise upon the commencement of the disease. On the morning of the second day there is usually a marked remission, often to normal. In the last two days of the initial stage the fever is moderate, very rarely being so high as at the beginning. With the eruption there is a new, rapid rise, usually higher than the initial one, so that we may well divide the fever into two periods—the prodromal fever and the eruptive fever. This latter is but brief and does not persist, as in scarlet fever, during the entire duration of the eruption. It falls by crisis when the rash has attained full development. There may, to be sure, be slight elevations of temperature during the next day or two; but, if the fever is considerable and persistent, it is always a sign that complications have arisen, probably in the respiratory apparatus.

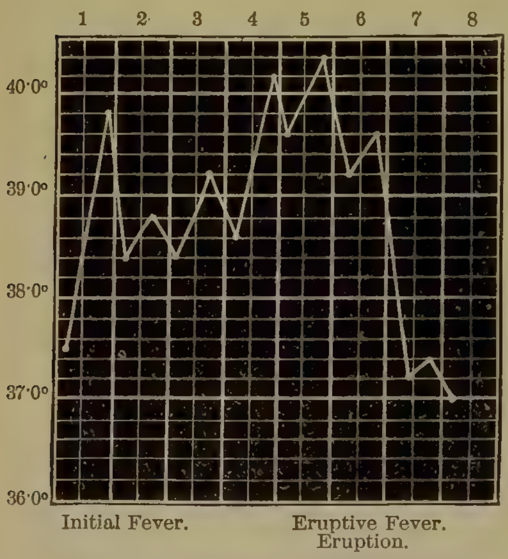

Fig. 6.—Example of the temperature curve in measles.

The eruption usually assumes the form described above, but may present manifold varieties. Sometimes its development is rudimentary. Sometimes it does not begin in the face, but on some other part of the body. This is generally regarded as a sign that the case will be anomalous in other ways as well. The individual spots may be smaller than usual, and may remain entirely separate from each other (*morbilli papulosi*). In other cases the eruption is so confluent (*morbilli confluentes*) that it resembles the eruption of scarlatina. The formation of vesicles (*morbilli vesiculosi*) also occurs, but much more rarely than in scarlet fever. Hæmorrhagic measles are also observed, but usually only in the form of small, capillary bleeding, and in cases that otherwise run a perfectly favorable course. Very rare cases have indeed been described, with a general hæmorrhagic diathesis and bad symptoms, resembling hæmorrhagic scarlatina. It is doubtful whether the "black measles" of the old writers was actually measles at all. In addition to the proper eruption of measles, other eruptions sometimes develop—among others, vesicles, wheals, and pustules.

The **complications of measles** are for the most part exaggerations, or abnormal varieties and extensions, of those troubles which are observed during the usual mild course of the fever. As in scarlet fever (*vide supra*), we often have to deal with the effects not of the original, but of secondary infection. Compared with the great majority of mild attacks taking the typical course, cases presenting complications of any severity are rare, and much less frequent than in scarlet fever. Epidemics are only now and then distinguished by unusual severity.

Often quite grave eye diseases are developed, particularly blennorrhagic conjunctivitis, keratitis, and iritis.

Marked inflammation of the mucous membrane of the nose, throat, and larynx may prolong the course of the disease. These are often merely exaggerations of the usual catarrh. Otitis media likewise sometimes occurs. A laryngitis of marked intensity, with considerable swelling of the parts involved, may produce much discomfort, or even symptoms of stenosis ("false croup"). Actual croupous and diphtheritic lesions of the throat and larynx also occur (diphtheria of measles). This last is indeed much rarer than scarlatinal diphtheria, but may have the same unhappy termination. It is worth mentioning that sometimes genuine laryngeal croup is observed in measles, unaccompanied by lesions of the pharynx.

It is, however, in the lungs that the most frequent and important of all complications in measles occur. The usual mild bronchitis becomes very intense, extends into the bronchioles (capillary bronchitis), and then results, for the most part, in a more or less extensive, lobular, catarrhal pneumonia (*q. v.*). This is almost always to be suspected when moist râles are heard in abundance over a large part of the chest, and when there is at the same time persistent fever and pronounced difficulty in respiration, with cough or dyspnœa. We get decided dullness on percussion only when the separate centers of infiltration are more than usually confluent. Genuine lobar, croupous pneumonia appears much less often than the lobular variety. It attacks one lobe, or several, is attended by high fever, and may end with a well-marked crisis.

The foregoing pulmonary symptoms usually appear at the height of the disease, and persist after the eruption fades. They may delay convalescence for weeks. In other cases measles will seem at the start to run a normal course, the temperature will have already fallen, and then come new fever and the appearance of decided pulmonary disturbance. This is always to be regarded as a grave complication; and especially in feeble children it may lead to death, with the symptoms of impaired respiration, or of constitutional exhaustion.

Marked intestinal symptoms sometimes appear, particularly an excessive diarrhœa, due to intestinal catarrh. It is characteristic of measles that in severe cases such a diarrhœa may assume a pronounced dysenteric character, indicated by blood and slime in the dejections, symptoms which usually depend upon the development of follicular colitis with ulcerations.

Now and then still other complications may present themselves, of which a full enumeration is impossible. Nephritis does occur, but far less often than in scarlet fever. A simple albuminuria during the acme of the disease is not infrequent, but as a rule has no especial clinical significance. We should mention gangrene of the cheek, the so-called noma, as a complication, which is very rare but apparently characteristic.

Peculiarities in the course of the disease are much rarer in measles than in scarlet fever. Yet we see, on the one hand, unusually mild or rudimentary cases, in which either the rash or the other local symptoms are remarkably slight, and on the other hand, abnormally severe cases. These latter are distinguished by the unusual height or persistence of the fever, by the severe constitutional and nervous symptoms, and further by the early appearance of complications. Such cases have been termed "typhoid measles." We have already mentioned the severe form of hæmorrhagic measles.

We should notice the clinical relation which measles bears to some other infectious diseases—to whooping-cough and tuberculosis. Measles and pertussis (*q. v.*) may follow each other in the same individual at a short interval, sometimes one and sometimes the other taking the initiative; epidemics of the two diseases prevail with comparative frequency at the same time. Tuberculosis is likewise to be mentioned as an important sequela of measles. Its frequent appearance at the close of measles is of course to be explained by supposing either that, in children who are already the victims of tubercle, the further extension of the tuberculosis is favored by measles, or that the catarrhal inflammation due to measles leaves behind it an especial predisposition to infection with the tubercular poison.

The **diagnosis** of measles, as of the other acute exanthematous diseases, is based chiefly upon the eruption. Personal experience does more to sharpen the perception than can the fullest descriptions. We can merely suspect the disease during the initial stage unless an epidemic prevails. If, beside the characteristic catarrhal symptoms, the above-mentioned eruption on the mucous membrane of the palate exists, the diagnosis becomes tolerably certain. We should consider that eruptions similar to that of measles appear in other diseases, more especially in rötheln, scarlet fever, typhus fever, in the beginning of small-pox, and in syphilis. Furthermore, we need to exclude eruptions due to such drugs as antipyrine, turpentine, and balsam of copaiba. In doubtful cases we shall be enabled to form a decided opinion by the other symptoms, and, above all, by the further course of the disease.

Prognosis.—We have already remarked how favorable in general the prognosis is, but we must here repeat that all epidemics do not exhibit the same benign character, and that in every case the physician must bear in mind the possibility of complications, and particularly the danger of severe pulmonary disturbances.

Treatment.—The patient should in general be kept somewhat warmer than in scarlet fever. Even in what seem to be the mildest cases the child should be kept in bed till desquamation is over. The sick-chamber is to be somewhat darkened, on account of the photophobia which usually exists at first. In this way, normal cases run on favorably without any especial therapeutic interposition. The catarrhal symptoms, however, should always be heeded, since to disregard them may lead to their becoming aggravated. The chief requisite is cleanliness. At regular intervals the eyes, the nasal cavity, and the mouth should be washed out with lukewarm water.

If, despite all this, certain disturbances appear in a worse form than usual, or if complications develop, these must receive especial attention. Severe eye troubles should be treated according to the usual ophthalmological practice; and here unguentum hydrargyri oxidi flavi (1 to 100) [U. S. P. is 40 to 420] and atropine are chiefly employed. The treatment of croupous trouble in the throat or larynx will be fully described in a later chapter. For the pulmonary troubles, lukewarm baths, combined if need be with rather cool douches, constitute the most effectual remedy, which we should employ if it is in any way possible. We thus evoke deeper inspirations and promote expectoration, and thereby contribute largely to preventing the development, or the aggravation, of severe lung trouble. Inhalations of steam or of medicated fluids are often advantageously combined with the baths. To substitute the cold pack for the baths is in general justifiable only when the baths are not practicable. Still, the pack does good. If employed, it should be kept up for three hours at a time, two or three times a day. The breathing will be improved, and usually the child will go quietly to sleep. We are not acquainted with any internal remedies for the lung troubles which are at all reliable. In rare instances the excessive accumulation of mucus in the bronchi requires the administration of an emetic. As expectorants we may try ipecac, liquor ammonii anisatus, or benzoin. If considerable intestinal disturbance arises, we must employ small doses of opium, or calomel, or subnitrate of bismuth. We hardly need to say that, whatever else is done, the strength of the patient should be kept up as much as possible by giving wine, broths, milk, eggs, etc. For at least two or three weeks after the disease has ended, the child must be very carefully watched.

As the disease is usually so mild, prophylaxis is not very strenuously attempted. If one child in a family is attacked, it is probably already too late to isolate the others, and it is even an advantage to the family to have all the children finish at once what they will hardly be able eventually to avoid. We would make an exception in favor of isolation if the disease prevailed in a severe form.

[It is not customary with us to insist so strongly upon isolation and thorough disinfection as in scarlet fever. But the tendency of the present day is toward a wide application of the principles of preventive medicine. It is certainly of no advantage to a child to contract the measles. Delicate children, especially those with tubercular predisposition, should be carefully guarded against it; and, even if it is decided that it is not worth while to attempt to confine the disease to one member of a family, every precaution should be taken against infecting other families. Under suspicious circumstances, consequently, children are to be kept away from school and from contact with others.

If there is any reason to fear the development of tuberculosis, every possible hygienic means should be employed in order that full vigor may be regained.]

CHAPTER VI.

RÖTHELN.

(*German Measles.*)

Rötheln is a disease similar to measles, but distinct from it, although formerly often confounded with it, and perhaps with scarlet fever as well. The observations of Steiner, Thomas, and others leave now no room to doubt that these diseases are distinct, for epidemics occur in which all cases present the characteristic peculiarities ascribed to rötheln. But the best proof is that children who have had rötheln are not infrequently attacked by genuine measles later. It may indeed be very difficult in an individual case to decide which disease is present; but that rötheln does exist, as an independent form of disease, can be denied by those alone who have never seen it.

After an incubation of about two or three weeks the disease begins with the appearance of the eruption. Initial symptoms preceding the eruption are either wholly absent or at most last for half a day. The eruption is decidedly like that of measles, but its individual spots are smaller. They are seldom larger than small peas and circular, being only exceptionally as dentated and irregular in outline as are the maculæ of measles. They appear on the whole face, the head, the trunk, and the extremities, are pale red (sometimes deep red), but slightly elevated, and are not apt to become confluent. In rare instances, small vesicles develop upon the macules. The soft palate sometimes exhibits, as in measles, a faint macular eruption at the beginning of the disease. After two to four days the eruption fades. There is usually no decided desquamation.

Other symptoms of disease than this eruption are slight. Fever in many cases appears to be entirely absent. As a rule, there is for a day or two a slight elevation of temperature, reaching 102° (39° C.) at most. Tokens of a moderate catarrh of the conjunctiva, the nasal mucous membrane, the throat, and the larynx are also observed—viz., photophobia, nasal discharge, and cough. Often, the cervical lymph-glands are more or less swollen. The constitutional disturbance is generally so slight that the child can hardly be kept in bed. Important complications hardly ever occur.

The prognosis of rötheln is therefore perfectly favorable, and the employment of any special treatment is needless.

CHAPTER VII.

SMALL-POX.

(*Variola. Varioloid.*)

Ætiology.—Small-pox has been known for centuries, although formerly often confounded with other diseases.* It is one of the most dreaded acute infectious diseases, and in earlier times it has destroyed thousands in its pestilential progress. It was the discovery of the possibility of prophylactic inoculation, and the ever-increasing spread of this precautionary measure, which first robbed the disease of some portion of its terrors.

Numerous statements have been made about the occurrence of micro-organisms in the variolous eruptions on the skin and mucous membranes, but we are compelled to say that we are not yet acquainted with the specific, organized poison of small-pox, however strongly justified we may be in assuming its existence. Bacteria can in fact easily be demonstrated in the eruption of variola, but most of them come from the surrounding atmosphere, and have no relation to the specific variolous processes. The foci of bacteria found in the internal organs (liver, spleen, kidneys) are also due, as their discoverer, Weigert, himself supposed, to the secondary ingress of other varieties of micro-organisms, and are not directly associated with the variolous process, the diseased condition of the skin furnishing a ready entrance for infectious matter.

Predisposition to variola, except as diminished by vaccination (*vide infra*), is universal. The disease may appear at any age, even *in utero*. Women are believed to be especially liable to it during pregnancy and child-bed. It is said that persons ill with another acute infectious disease, such as scarlet fever, measles, or typhoid fever, are, for the time being, tolerably secure from infection with small-pox; but this rule has exceptions. The same individual rarely takes the disease a second time.

A case of variola is always the result of transmission of the poison to a healthy person from one who is already ill with it. The specific poison certainly is most abundant in the diseased portions of the body and in the pus of the suppurating pocks, as well as the crusts and scales which are left when these have dried up; but the disease is also contagious in its earlier stages, before the pustules develop, and even, according to a few observations, during the stage of incubation. Certainly the variolous poison is very volatile—that is, it is prone to disseminate itself through the air in the neighborhood of the patient. In order to catch the disease it is not necessary to touch the patient, but merely to remain in his vicinity. In many cases we can not, however, determine with exactness the mode of transmission, since the contagion may either be direct or by means of objects and utensils with which a patient has come in contact—for example, the soiled linen. The dead body is capable also of transmitting the disease. In general, numerous instances point to a considerable "tenacity" in the poison. The precise manner of infection is not yet known. It is most probable that the poison is drawn into the lungs with the inspired air.

It has been demonstrated that the disease can be transmitted to healthy persons by direct inoculation of the contents of the variolous pustules. It is stated that monkeys and other animals may be successfully inoculated in the same way. Whether inoculations with the blood of the sick will reproduce the disease is not yet settled. The secretions (saliva, sweat, urine, and milk) do not apparently contain the infectious matter.

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* The very name small-pox (*petite vérole*) is significant of its confusion with syphilis, which was termed the "great pox."

Course of the Disease. Variola and Varioloid.—The stage of incubation lasts some ten to fourteen days, often a somewhat shorter time, seldom longer. During this period prodromal symptoms are absent or insignificant.

The disease itself begins suddenly with what are usually very characteristic initial symptoms—rigor, fever, headache, and intense pain in the loins. It is only in comparatively few cases that one or another of these symptoms is slight or wanting. The constitutional symptoms may be very severe—a dry tongue, stupor, wakefulness, delirium. The fever continues intense for some days. The pulse is much quickened. There is almost total anorexia, and often there is vomiting. There is constipation, or, more rarely, diarrhœa. Frequently there is a slight sore throat, and sometimes a slight bronchitis. The spleen is enlarged in most of the severe cases, and the urine often has a trace of albumen. In women, menstruation occurs in a remarkably large number of cases. The proper variolous eruption does not at once appear, but from the second day other characteristic efflorescences are not rare. These are termed the initial rash of small-pox. We may find either a diffuse or macular erythema, extending in varying degree over the trunk and extremities, or a hæmorrhagic eruption with small spots appearing by preference upon the hypogastrium and the inner surface of the thighs (in the so-called femoral triangle of Simon). It is noteworthy that this particular region is said often to remain free from the proper variolous eruption. The erythema soon vanishes, but the petechiæ remain visible for some time.

The initial stage, just pictured, lasts usually three days. Severe symptoms occurring at this time do not exclude the possibility that the further course of the disease may prove favorable, while mild symptoms are of good omen.

At the end of the third or on the fourth day the temperature makes a decided fall, and the regular variolous eruption begins to be developed upon the skin—the *stadium eruptionis*. During this period an evident difference among the separate cases becomes manifest. This distinction can not indeed be always drawn with a narrow line, but it is noticeable enough to justify the establishment of two types of variolous disease. We refer to the division into a severe form (variola vera), and another, mild form (varioloid). The variola proper has a well-developed eruption with many pustules, and, as a result of this, a second stage of fever (*stadium suppurationis*). Varioloid has a much more scanty eruption, and little or no suppurative fever. We must now discuss these two forms separately.

VARIOLA VERA.

The eruption almost always begins in the face and upon the hairy scalp, appearing somewhat later on the trunk and arms, and last of all upon the legs. It begins in the form of little red dots and spots, which develop in about two days to small papules (*stadium floritionis*). If the hand be passed over thickly set and well-developed papules of variola, a peculiar soft, satin-like feeling is perceived. On the points of these papillæ a little vesicle forms. This keeps growing larger and larger, its contents become turbid and purulent, till at last, on the sixth day of the eruption and the ninth of the disease, the development of the genuine pustule of variola is complete (*stadium suppurationis*). The pustule usually presents upon its summit a little dimple ("Pocken-nabel"), and is surrounded by a red border or "halo." Where the pocks are especially close together, as in the face, the skin between them is diffusely swollen, and the consequent burning and pain are excessive. The countenance becomes much disfigured. Often the eyes can not be opened because of the œdema. The hands also are apt to be intensely affected, especially the back of the hands, and also all parts which have previously been injured in any way (pressure or friction of clothing, etc.). The immunity of the skin in the so-called femoral triangle has been already mentioned.

At the same time with the eruption upon the skin, or even somewhat earlier, a perfectly analogous efflorescence develops upon the mucous membranes. The chief places for its appearance are the mouth and throat, the tongue, the soft palate, the nasal cavity, also the larynx, the trachea, and the upper part of the œsophagus. In the vagina and rectum it is rare and scanty. In this mucous efflorescence, however, there are no proper pustules, but small, superficial ulcers. These result from the maceration of the uppermost layers of the mucous membrane. They sometimes become confluent. The annoyance produced by this eruption in the mouth and throat is, of course, very great. The pocks in the larynx manifest themselves by hoarseness, and occasionally by symptoms of stenosis.

As we have said, the beginning of the eruption is the signal for a noticeable fall in the temperature. But in true variola the fall does not reach the normal, or only temporarily. The other symptoms likewise remit, especially the headache and lumbar pain. When, however, the suppuration begins, the fever rises once more, and there are fresh symptoms of constitutional disturbance. This is the time for the dreaded attacks of delirium, during which the patient must be vigilantly watched, lest some untoward event happen. Now, too, complications may arise (*vide infra*).

On the twelfth or thirteenth day of the disease the pustules begin to dry up (*stadium exsiccationis*). The purulent contents of the pustules, part of which have burst, form yellow crusts, the swelling of the skin subsides, and, a few days later, the crusts and scabs begin to fall off. With the beginning of desiccation, the fever declines ; the local as well as the constitutional symptoms become daily slighter, and convalescence follows. The healing of the pustules is frequently accompanied by an extremely troublesome itching. After the scabs have been cast off, the skin presents pigmented spots, which persist for months. Wherever the cutis vera has itself been destroyed by the suppuration, a scar is inevitable. Thus arise the familiar scars of small-pox, which continue visible through life. Very often, after the end of the disease, there is almost complete alopecia. The hair often grows again, but not always.

VARIOLOID.

The distinction between varioloid and variola vera is not in kind, but in degree. Varioloid is only a milder form of variola. There is, as we have already said, no sharp boundary-line between the two. Varioloid is most often observed in those whose susceptibility to the variolous poison has been diminished by vaccination (*vide infra*).

As above mentioned, the behavior of the disease during its initial stage will not permit us to decide positively whether variola or varioloid will be developed. It is true that if the symptoms be especially mild, we may guess that it will be varioloid ; and, likewise, the appearance of the initial erythema already spoken of is regarded as a favorable omen.

Shortly after the pocks begin to appear, the decision can almost always be made with certainty. In varioloid the eruption is rather scanty. It is often irregular, and does not by any means always begin, like that of variola, in the face, but often on the trunk. The individual pocks are in no way different from those of variola ; but it often happens that they do not pass through all the regular stages to full suppuration, but undergo resolution before this occurs. Such cases, where there is nothing beyond papillæ or vesicles, are sometimes spoken of as *variolois verrucosa seu miliaris.* The scantiness of the eruption and the limited amount of suppuration have for their corollary an absence, or at least a very slight development, of the suppurative fever.

When the eruption appears the temperature usually falls by crisis to the normal level and remains there. The desiccation may begin as early as the eighth or tenth day of the disease, so that the whole duration of varioloid is considerably shorter than that of variola. Grave complications are very exceptional. The pocks may develop upon the mucous membranes, but here, too, they are scanty and not very vigorous.

Course of the Fever, Symptoms presented by Separate Organs, and Complications.

1. *Fever* (*vide* Fig. 7).—In the initial stage, as we have said, the temperature rises rapidly as a rule, with a pronounced rigor; and during the first days it very often reaches 104° to 106° (40°–41° C.). It sinks on the third to the sixth day, when the first papillæ develop, and now, in the case of varioloid, falls rapidly to normal, and remains there. In variola the decline is slower and less complete; and with the beginning of suppuration the temperature begins to rise again. The violence of this suppurative fever is usually in direct proportion to the severity of the eruption. It has manifold fluctuations, but seldom lasts, in severe cases, less than a week. Temperatures of 104° (40° C.) and higher are common. The fever declines by lysis. In case of approaching death, the temperature may be extremely high, even reaching 108° or 109° (42°–43° C.).

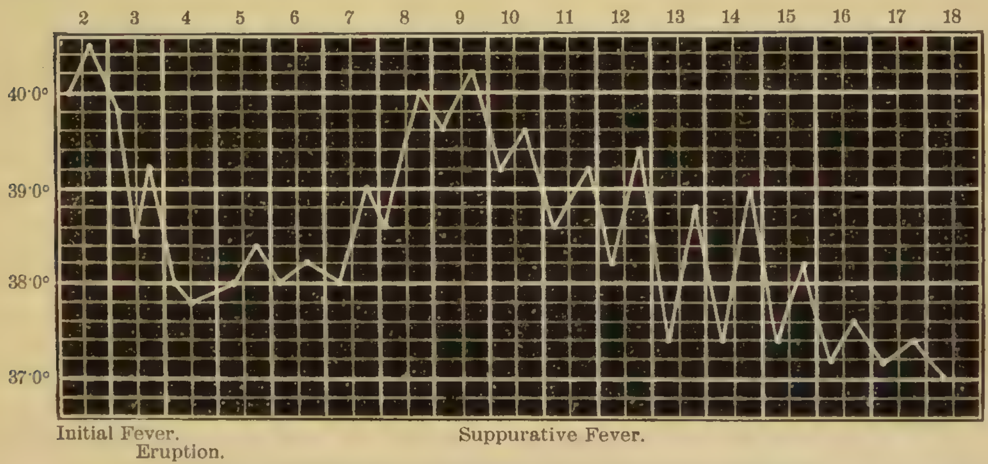

Fig. 7.—Example of the temperature curve in true small-pox.

2. *Skin.*—We have already described the macroscopic appearance of the eruption. It remains to mention briefly the histological phenomena. The first demonstrable changes are in the cells of the deeper layers of the rete Malpighi. As a result of the variolous infection, the cells perish, are swollen by the lymph which escapes from the papillary blood-vessels, and are transformed into flaky, homogeneous masses without nuclei ("coagulation necrosis" of Weigert). The lymph becomes more and more abundant, and crowds the cells farther and farther apart. These are thereby finally changed into threads and membranes, forming a distinct net-work in the vesicle. This explains why, if such a vesicle be pricked, its entire contents are never discharged at once. Great numbers of white corpuscles escape, along with the lymph, from the blood-vessels, and finally render the contents of the original vesicle purulent. Proliferative processes occur

in the surrounding epithelial cells, which are still intact, and thus the margin of the vesicle becomes elevated, while the dead portion in the center sinks in. Thus the pock becomes umbilicated. If a portion of the papilla itself suppurates, a scar must be left on healing. If the process remains limited to the epithelium, complete regeneration takes place, and the skin reassumes its normal appearance.

Certain secondary complications, which sometimes attack the skin, remain to be mentioned : abscess, phlegmon, erysipelas, gangrene, and bed-sores. None of these are directly due to the specific variolous intoxication.

3. *Respiratory Organs.*—The disturbances here are in part symptoms of the specific process of the disease, and in part secondary. The frequent occurrence of secondary symptoms in small-pox is easy to understand (compare the chapter on lobular pneumonia). Of the primary symptoms, we should mention genuine pocks in the larynx, the trachea, and the larger bronchi. As sequels to these, more or less severe secondary disorders are very frequent : laryngeal ulcerations, which may even lead to laryngeal perichondritis and œdema of the glottis ; diffuse bronchitis ; lobular pneumonia, often of great extent, due to the inhalation of solid matter into the lungs, and frequently accompanied by pleurisy. It should be especially noticed that lobar, croupous pneumonia is not rare. Whether this be likewise secondary or a direct result of the variolous poison is not yet known.

4. *Digestive System.*—The genuine pocks often develop, as stated, in the mouth and pharynx, and likewise in the upper part of the œsophagus. They are not observed in the mucous membrane of the stomach or intestines. The active diarrhœa sometimes seen depends upon catarrh of the intestine. Dysentery is rare. The eruption in the mouth and throat may result in severe secondary troubles, purulent otitis, parotitis, pharyngeal diphtheria, etc. The spleen is almost always considerably enlarged, and often the liver, but in a less degree.

5. *Circulatory System.*—Pathological changes in the heart are rare, if we except the slight parenchymatous degeneration of its muscular fibers, common to almost all severe infectious diseases. Sometimes there is a slight endocarditis (*q. v.*), which is probably secondary. Pericarditis is rather more frequent.

6. *Organs of Special Sense.*—Genuine variolous pustules occur upon the eyelids and the conjunctiva. Later in the disease there may be keratitis, iritis, or choroiditis.

We have already mentioned the relative frequency of aural disturbances, particularly purulent otitis media.

7. Articular swelling may appear in the suppurative stage. The shoulders and knees are most apt to be attacked. Periostitis also occurs.

8. *Nervous System.*—We find no pathological changes corresponding to the severe nervous derangements manifested during the disease. After the small-pox is over, spinal diseases sometimes occur, with either paralysis or ataxia. Westphal has demonstrated as their cause, in some cases, numerous disseminated centers of inflammation in the spinal cord.

9. Albuminuria is quite frequent in severe attacks, but genuine nephritis is a very rare complication.

Anomalies in the course of the disease are manifold. We do not speak of the two typical forms already considered. There are abnormally mild cases, with scarcely any initial symptoms, or with an obscure eruption, or with no eruption at all (*febris variolosa sine exanthemate*). In such cases a correct diagnosis is possible only at the time an epidemic prevails, and by the aid of the attendant ætiological circumstances. There are also abortive cases in which the first symptoms are severe, but which recover with remarkable rapidity.

The abnormally severe cases are more important. First, there is the confluent variety. This is merely the typical process in its completest development. The initial symptoms are themselves generally very severe, and are followed, without any considerable remission of the fever, by the eruption of hundreds of pustules. The skin of the face and hands is one continuous area of suppuration. The local discomfort is extreme, as is also the intensity of the fever and of the constitutional symptoms. The nervous system suffers most. There is at the same time an unusually abundant eruption upon the mucous membranes. The occurrence of the above-mentioned complications affecting the various organs of the body is frequent. Death is a common result; or, if recovery takes place, it may be delayed by tedious sequelæ.

Hæmorrhagic small-pox is the worst anomalous form. The name is applied to several different varieties. In the first place, any variolous eruption may become more or less hæmorrhagic, and yet the general course of the disease not be essentially altered. Such cases are more common among elderly people, cachectic persons, and drunkards. Secondly, there is a very severe form of small-pox, which is generally quickly fatal. The initial stage is marked by the unusual severity of the symptoms. The abundant eruption soon becomes hæmorrhagic, and there are also ecchymoses in the mucous membranes and the internal organs. This has been called black small-pox, and by Curschmann *variola hœmorrhagica pustulosa.*

There is another form of hæmorrhagic variola, different from these but linked to them by transitional varieties. In it the acute hæmorrhagic diathesis develops during the initial stage. Death almost always occurs before the regular variolous eruption. This most frightful form is usually termed *purpura variolosa.* That it is small-pox is proved by its ætiological relations alone. Otherwise it would be impossible to distinguish it from certain other acute septic disorders. It is prone to attack the youthful and vigorous. Chills, headache, and pain in the loins are the first symptoms, just as in ordinary cases. Cutaneous ecchymoses appear as early as the second or third day. They increase in area so rapidly that one can almost see them grow. They are most extensive in the hypogastric region. There are also ecchymoses in the eyelids, the conjunctiva, the mouth and pharynx, and, as the autopsy discloses, many in the internal viscera. The constitutional symptoms are most severe, and the patient seldom survives the fifth or sixth day of the disease.

Diagnosis.—The certainty with which we can make the diagnosis of small-pox in any well-developed case is equaled by the difficulty of deciding about it during the beginning of the disease, or even during the beginning of the eruption. At this period diagnosis may be impossible. When the variolous eruption is in process of development, it may be confounded with typhus fever, with that form of measles in which the papillæ are prominent, with syphilitic eruptions, and with certain forms of erythema exsudativum, just breaking out. We can not here fully discuss all the factors which should be considered in making this diagnosis. It is important not to regard the cutaneous appearances alone, but to note all the other symptoms besides. But it is often necessary to watch a doubtful case for some time before a diagnosis can be established.

Prognosis.—The facts which are of greatest weight in prognosis have already been emphasized. We may repeat that during the initial stage the prognosis of any individual case can seldom be determined. If the first symptoms are mild, or if the initial erythema appears, the case is regarded hopefully. The abundance of the eruption has an influence upon the severity of the disease. Circumstances peculiar to the individual are also important—e. g., age, constitution, or alcoholic habits. We have already called attention to the danger of confluent small-pox, and to the almost absolutely fatal prognosis in the genuine hæmorrhagic variety. The mortality varies greatly in different epidemics; on the average it may be taken at about fifteen to thirty per cent. Beyond doubt, the introduction of vaccination has decidedly lessened the fatality of the disease by diminishing the frequency of the severe forms.

Treatment.—1. *Prophylaxis—Vaccination.*—As in all contagious diseases, isolation is of little avail unless complete. This fact has led to the erection in late years of small-pox hospitals. All utensils used by the patient, and his clothing, bedding, and the like, should be most carefully disinfected. The best method is to employ a high degree of heat—viz., 240° to 250° (115°–120° C.).

These precautionary measures are employed in many other diseases as well, but for small-pox we are acquainted with a peculiar method of prophylaxis. It is founded upon a fact which is at once the most remarkable and inexplicable, and the most beneficent, within the domain of the infectious diseases. We refer to vaccination. It must long ago have been remarked that a person who has had the disease once, enjoys, to a large degree, immunity from any fresh infection. This suggested the idea of exposing children purposely to contagion, so as to insure them from small-pox for the rest of their lives. The actual inoculation of small-pox is said to have been long practiced in India and China. In the year 1717 it was employed by Lady Montague, of England, upon her own son, and with success. Unfortunately, however, the inoculated small-pox proved fatal in many instances; and, being itself contagious, it served to spread the disease still further. Then appeared an article written by the English surgeon, Edward Jenner, in 1798. This informed the medical profession of a fact already known to the rural population of his native place, but which Jenner first established scientifically, and recognized in all its importance. There sometimes occurs a disease similar to small-pox upon the teats and udder of the cow, called *variola vaccina*. It is apparently a local trouble, and can easily be inoculated upon the skin of human beings. Vaccine pustules will be developed upon the spot inoculated. These almost invariably heal without any great constitutional disturbance; but the person vaccinated possesses the same immunity from small-pox as if he had had small-pox itself. This statement of Jenner's was soon confirmed upon every side. The result is the continually spreading custom of prophylactic vaccination. In some countries it is enforced by law, and it can be opposed only by ignorance or by lamentable prejudice.

To explain how vaccination can protect against small-pox in this way is still utterly beyond our powers. We have lately gained this much help in understanding it, that it is no longer an isolated fact; for analogies have been discovered in the case of other acute infectious diseases (cf. the chapters on hydrophobia and malignant pustule). We are likewise in the dark as to the relation between small-pox and vaccinia. Many authors regard the virus of vaccinia as merely a modification of the variolous poison, while others assume that there is a specific difference between the two. As yet, the infectious material of neither has been exhibited in a pure state; and we must for the present therefore leave this question undecided. A statement in support of the essential unity of the two infectious agents can be adduced. The inoculation of cows with small-pox is said to produce vaccinia, which, inoculated in its turn upon children, will result in vaccinia and not in small-pox. This statement rests on doubtful evidence. Careful experiments made in 1865 in Lyons had a different result, showing that the inoculation of cows with the contents of variolous pustules produces an eruption different from that of vaccinia. Children inoculated from these cows had true variola; while no case of small-pox has ever resulted from genuine vaccine lymph.

We can mention only the most important of the details relating to vaccination and the method of its performance. The inoculation is made either with animal virus, direct from the cow, or with humanized virus, obtained from persons previously vaccinated. The lymph taken from a vaccine-pustule can be kept a long time, either pure or mixed with glycerine, without deteriorating. It is kept in small glass tubes, hermetically sealed, or in a dried form between glass plates which have been thoroughly disinfected and cemented together, [or else upon little "points" made of bone]. The most common mode of vaccination now in use is to make three shallow incisions, 3 or 4 ctm. apart, in the skin of the upper arm, and to introduce the vaccine-lymph into them. The surrounding tissue becomes swollen in three or four days. In seven or eight days the vaccine vesicles are well developed, if the disease takes its normal course. Next they become purulent, and then dry up, and finally, on healing leave the familiar scar behind. The whole process occupies about three weeks. If the vaccination fails, or is but partially successful, it must be repeated after a few months. The protective power of vaccination does not last indefinitely, and therefore re-vaccination is necessary every five or six years. The first vaccination of children usually takes place when they are three or four months old. If they are feeble we wait longer, unless small-pox is prevalent.

It must be confessed that vaccination is not without its dangers. The little cutaneous wound made by it may lead, like any other, to sepsis or to erysipelas. The latter has been called vaccination-erysipelas. But such misfortunes are extremely rare. The "vaccine roseola" deserves especial mention. It appears first upon the arm vaccinated, and spreads over the rest of the body; but it is not a serious matter. It is of course possible that other diseases, among which syphilis is of chief importance, may be inoculated along with vaccinia; but this is a very rare occurrence—much more so than the enemies of vaccination pretend. If the physician exercise proper care in the selection of the person from whom to take humanized virus, it can be entirely avoided. The exclusive employment of animal virus in vaccination does away with a number of dangerous possibilities, and for this reason it is constantly growing in popularity.

[The incubation stage of vaccinia being shorter than that of small-pox, the prompt vaccination of an unprotected individual who has been exposed to infection should always be practiced, if possible; oftentimes the severe disease may thus be prevented.]

2. The treatment of small-pox is purely symptomatic. When the disease has once begun it is too late for vaccination to have any influence upon its further course. During the initial period we may advantageously employ cool baths to diminish the fever and alleviate the constitutional symptoms. An ice-bag will relieve the headache. We must not let the lumbar pains lead us to any but a cautious use of local irritants, for the pocks come out in greater abundance upon such portions of the skin as have been in any way irritated. If the disease proves, during the stage of eruption, to be varioloid, there will be no further need of special treatment. Good nursing and proper food will suffice.

The true small-pox, on the other hand, demands the interposition of the physician. He must strive to guard the regular course of the disease in the skin and in those portions of the mucous membrane which are accessible from being disturbed by secondary inflammations. For we have no doubt that the ruptured pustules furnish a most easy ingress to septic impurities from the surrounding atmosphere, so that later, when there is extensive suppuration of the skin, or analogous and severe disturbance in the mucous membrane, it is impossible to discriminate between the effects of the small-pox itself and those due to the secondary suppuration. If we were able to have the whole process go on "antiseptically" we should certainly have made an important advance in therapeutics. Indeed, the methods of treatment which have been up to this time recommended fulfill this indication up to a certain point, e. g., painting the skin with tincture of iodine, or with a strong solution of nitrate of silver—methods formerly much in vogue. Schwimmer's suggestion seems still better. He recommends, from the beginning of the eruption, the use of a paste made as follows: Acid. carbol., parts 4 to 10; ol. olivæ, 40; cretæ præparat., 60. M. et ft. pasta mollis. This is spread on pieces of old linen and laid upon those parts where the eruption is apt to be worst—viz., the forearm, hand, and leg. The face is covered with a mask, having holes corresponding to the mouth, nose, and eyes. The applications are changed every twelve hours. Under this treatment the local distress is said to be diminished, suppuration slight, and healing comparatively rapid. The pain and sense of tension in the skin are often relieved by cold applications, or by simple ointment or oil. Under Hebra, in Vienna, continuous warm baths were employed in severe cases with great success.

The treatment of the affected mucous membrane in small-pox must also meet the indication above mentioned. The most thorough disinfection of the mouth and pharynx must be aimed at. The means to be used are careful washing and gargling with solutions of chlorate of potash (1 to 30), carbolic acid, borax, permanganate of potash, or liquor ferri chloridi. The eyes, if they need it, must also be appropriately treated. As to all other complications, cool baths are relatively the most useful remedy. They can be given without difficulty. The chief indications for them are severe pulmonary or nervous symptoms, or continuous high fever. Internal antipyretics, such as quinine or antipyrine, are also employed. Violent nervous disturbances, such as delirium, sometimes require the cautious use of narcotics. There is nothing to add as to the treatment of malignant hæmorrhagic small-pox, for, as we have said, such cases are unfortunately almost hopeless.

CHAPTER VIII.

VARICELLA.

(Chicken-pox.)

Varicella is truly one of the children's diseases. Adults very rarely have it. It is contagious, and often comes in epidemics.

The stage of incubation does not last over thirteen to seventeen days. The disease begins with the appearance of vesicles, the size of a pea or a little larger, usually having a small red areola, and varying in number from ten to one hundred or more. The trunk usually bears the greater part of the vesicles, while the extremities have few. The face is frequently the seat of a considerable number, and sometimes there are a few upon the hairy scalp. There may be a vesicle here and there upon the mucous membrane of the mouth or palate. There are seldom any prodromata. Slight symptoms of fever may attend the eruption itself. The eruption is usually over in a few days, although there may be repeated crops, so that we often see fresh vesicles by the side of others which are drying up. Each separate vesicle heals quickly, and the pustulation seen in small-pox is here exceptional. The course of the disease is completed in a week or ten days. Most children feel perfectly well the whole time, although there may be in rare cases pain in the limbs, anorexia, and a slight coryza. A severe complication is hardly ever seen. An unusual event is a mild nephritis.

Exceptionally, the disease may be rudimentary, with a varicelloid roseola and no formation of vesicles. On the other hand, some cases present quite severe constitutional symptoms and a high fever, even reaching 105° (41° C.) temporarily.

In most cases, however, as we have said, the child is so slightly disturbed that a physician is hardly thought necessary.

The **diagnosis** is almost always easy. Formerly varicella was often confounded with small-pox, and to this day the followers of Hebra, in Vienna, for some inconceivable reason, maintain the identity of the two. That they are essentially distinct is shown (1) by the epidemics of the two appearing entirely separate from each other, (2) by the fact that having one does not give immunity from the other, and (3) by the uniform failure of attempts to produce variola by inoculating varicella, or *vice versa*. Still, we must bear in mind, in order to avoid mistakes, that many dermatologists class the mildest cases of small-pox as varicella. Those who devote themselves to general diseases are probably all now convinced that varicella is a separate disease.

The **prognosis** is perfectly good. There is usually no special treatment necessary, but young children should be kept in bed till the eruption has dried up.

CHAPTER IX.

ERYSIPELAS.

(St. Anthony's Fire.)

Ætiology.—Erysipelas is an inflammation of the skin, excited by the presence of a specific, pathogenic micrococcus (*vide infra*), and recognized by redness. swelling, and pain. It has the peculiarity of spreading gradually, by direct extension, from its point of origin over a larger or smaller portion of the skin. There are two varieties commonly recognized—an idiopathic, or exanthematic, and a traumatic. The latter may follow any cutaneous wound if it be infected with the specific virus of erysipelas. Traumatic erysipelas is therefore a surgical disease, and will not be further considered here; nor shall we treat of puerperal erysipelas, a possible sequence to injuries inflicted upon the female genital organs during parturition; nor of the erysipelas of the new-born, which usually has its origin in the navel or in small fissures of the anus.

The so-called idiopathic erysipelas appears almost exclusively in the face, or at least it starts there. As it goes on it very frequently spreads to the hairy scalp, and not infrequently it also extends down upon the trunk. The clinical manifestations are perfectly characteristic. It is, however, a question whether idiopathic erysipelas is essentially different from the traumatic variety. There is good reason to suppose that facial erysipelas is really traumatic in every case, having its origin in injuries of the skin or mucous membrane, which are so small as to be overlooked. This view not only seems *a priori* very probable, but is supported by numerous cases. We see, for example, erysipelas taking its origin in excoriations of the nose or the borders of the nostrils, or in excoriations or fissures of the lobe of the ear. Quite often coryza precedes the erysipelas, and, in that case, the first inflammatory swelling of the skin is at the nose. The probable explanation of this fact is that the nasal catarrh is apt to cause slight erosions of the mucous membrane, and that these furnish an opportunity for infection with erysipelas. On the other hand, it can not be denied that there are cases of facial erysipelas where it is absolutely impossible to make out any cutaneous excoriation, and where there is an initial stage with feverish symptoms preceding the localized trouble in the skin (*vide infra*). Such cases suggest the thought that erysipelas is like the acute eruptive diseases, and that it is at least possible that infection may take place in some other way than the one mentioned.

The specific virus of erysipelas has been brought to our knowledge chiefly through the researches of Fehleisen. He has demonstrated a characteristic "chain-forming micrococcus" (streptococcus erysipelatos, *vide* Fig. 8) in the lymphatic vessels and serous canaliculi of the diseased skin. This micrococcus is distinguished by its peculiar behavior in pure gelatine cultures, and invariably causes erysipelas in the rabbits and the human beings that are inoculated with it.

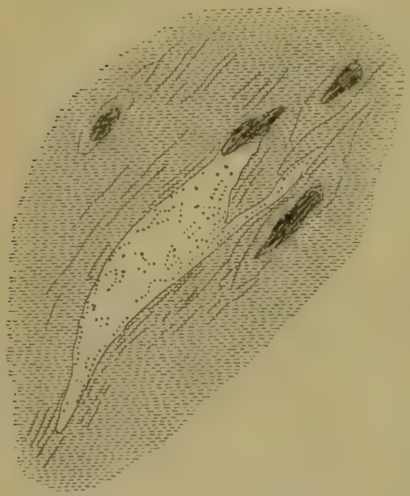
FIG. 8.—The cocci of erysipelas. ×700. Section through a lymph-vessel in the skin.

Facial erysipelas is most apt to attack the young, and seems to be somewhat more frequent in women than in men. The laity erroneously regard catching cold and getting frightened as frequent causes of the disease. If we except the predisposing causes above mentioned —viz., coryza, slight scratches, cuts, etc.—we usually find no cause of which we can feel certain. Often endemic influences are important. It has been long known that traumatic erysipelas can get so secure a footing in particular hospitals or wards that every wounded person treated in them is in danger of this disease. But the apparently idiopathic variety is sometimes remarkably frequent in particular places. Likewise several members of one family may have facial erysipelas simultaneously. In nearly all such cases the sufferers are infected from some common source, for direct contagion is certainly exceptional. Direct inoculation can, however, as has been proved, convey the disease from a patient to other persons or to animals.

In contrast with the behavior of the acute eruptive diseases, erysipelas is peculiarly apt to attack the same individual over and over again. There are persons who have facial erysipelas about every one or two years. Often the explanation of this apparently lies in some chronic disease—e. g., chronic ozæna—which makes infection easy, but in other cases no cause can be discovered. Marasmus seems to predispose to erysipelas. At least we have observed that erysipelas occurred with relative frequency, in the Leipsic hospital, in patients suffering from the last stages of phthisis or cancer, or similar diseases.

Clinical History.—In many cases the first subjective symptoms are simultaneous with the cutaneous swelling, and these are chiefly local. There is pain and a sense of tension in the skin. Soon subjective symptoms of fever also appear, such as general malaise, anorexia, and headache. In other cases the disease starts with more violent constitutional symptoms: there is an initial rigor, with violent headache and great languor. Almost at the same time, or sometimes two or three days later, the patient notices that the face is swollen. In rare instances the disease begins with sore throat. We saw three almost simultaneous cases of facial erysipelas in one family, where a severe sore throat lasted for four or five days preceding the appearance of the cutaneous disorder.

The erysipelatous process in the skin is almost always circumscribed at first. It usually starts on the nose, less often upon the cheek, the ears, or the hairy scalp. The skin becomes considerably swollen, grows red, smooth, and shiny, and feels hot. The redness and swelling keep spreading. There is usually a sharp, elevated ridge, perceptible to sight and touch, separating the diseased from the still healthy portion of the skin. As long as the erysipelas is spreading, we see stretching out from its border, or somewhat removed from it, small red streaks and spots which gradually increase in area and intensity, and finally coalesce. Any decided fold in the skin may hinder for a time the extension of the disease. The naso-labial folds are particularly apt to limit it. The border of the hairy scalp frequently forms a terminal line ; but the whole scalp may be attacked, the inflammation stopping only when it reaches the nape of the neck. It is only in a relatively small number of cases that it spreads farther yet, attacking the back, the arms, and the anterior surface of the trunk, or even extending to the feet. This is known as *erysipelas migrans.* The facial erysipelas may be healed long before the disease ceases to extend over the other parts of the body. When the spreading process is about to cease, the inflammation usually becomes decidedly milder, appears only in isolated spots, and finally stops completely. In most cases, only the face, the ears, and a part of the scalp are attacked.

It is not a rare thing for vesicles or bullæ to form in the portions of skin attacked. Such cases are called *erysipelas vesiculosum* or *erysipelas bullosum.* The serum may change to pus in these blisters, and then we have *erysipelas pustulosum.* Exceptionally the infiltration of the skin becomes so intense as to result in a localized necrosis or gangrene—*erysipelas gangrœnosum.* The parts most apt to be attacked by this are the eyelids.

Microscopic examination of the skin shows a marked hyperæmia of all the blood-vessels and a very considerable infiltration of both the skin and the subcutaneous connective tissue with serum and cells. In those parts where vesicles are formed there are many dead and disintegrated epithelial cells in the rete Malpighii. The presence of great numbers of the specific chains of micrococci has been already mentioned.

The inflammation in any one spot usually ends four or five days after it has made its appearance there. There is usually much attendant desquamation. The face is often left with a finer complexion than it had before.

The other symptoms, of which the constitutional disturbance and the fever are chief, correspond pretty closely to the severity and extent of the cutaneous lesion. It is comparatively seldom that this correspondence does not exist.

The fever in facial erysipelas usually rises rapidly at first, and to a considerable height. We have seen but few cases where the high fever was delayed till a day or two after the skin was attacked. The temperatures observed in erysipelas are often extreme : 106° (41° C.) is not at all rare. The highest we ever saw was 107·2° (41·8° C.). While the erysipelas continues or is spreading, the fever is seldom continuous, nor are the remissions insignificant. Pronounced intermissions, even down to normal, are very frequent, but are followed again by a rapid and great rise of temperature. The fever may terminate with a genuine crisis. In intense cases of considerable extent, or in erysipelas migrans, the termination is more apt to be by a more or less gradual lysis. We have seen the cutaneous inflammation in erysipelas migrans still extend itself a little, in a rudimentary form, after the fever had completely ceased.

The headache is often intense, and seems to result not merely from the inflammation of the scalp, but from disturbances of the circulation in the underlying parts. Other severe cerebral symptoms are also relatively frequent. The patient may be very restless, excited, and wakeful. At night there may be mild or even violent delirium ; or there may be decided stupor. All these symptoms are in chief part due to the constitutional infection, or, to speak more accurately, to the intoxication caused by the infection ; but they also justify a surmise, as we have said, that there is a circulatory derangement in the meninges and the brain itself, resulting from the inflammation of the scalp. In drunkards, delirium tremens is not infrequent.

One of the most constant symptoms in facial erysipelas is gastric and intestinal disturbance. There is usually complete anorexia. The tongue is thickly coated. Vomiting is frequent, not only at the beginning but during the course of the disease. There is constipation; or there may be quite severe diarrhœa. There is no pathological lesion known corresponding to these clinical symptoms.

The entire duration of the disease varies greatly in different cases. A very light case may get well in a few days. Most cases of average severity last a week or ten days. Erysipelas migrans may continue for many weeks. We have several times seen a relapse come on after a number of days of complete apyrexia. Either the face would be once more attacked, or some portion of the skin which had previously escaped.

Local **complications** are comparatively rare in erysipelas. The lymphatic glands of the throat and back of the neck are very frequently somewhat swollen, but seldom attain great size. Bronchitis and lobular pneumonia may develop in severe cases, but are not at all characteristic. Some observers call attention to the occurrence of pleurisy, endocarditis, and pericarditis; but these complications also are probably secondary. The spleen is usually slightly swollen. Sometimes there is an icteroid hue. The urine frequently contains a small amount of albumen, and in severe cases of erysipelas acute hæmorrhagic nephritis is not so very rare. Swelling of the joints has been repeatedly observed. It is more frequent in the severe surgical forms of erysipelas, which are combined with universal septic and pyæmic conditions of the system. Purulent meningitis may complicate an erysipelas located in the head, but it is very rare. We should be exceedingly cautious about asserting its existence even when the cerebral symptoms are very pronounced.

Cutaneous complications are relatively frequent. We have seen herpes labialis quite often, and a number of cases of urticaria. Of much greater importance are the cutaneous abscesses which occur in severe cases. These are due to a phlegmonous or even gangrenous inflammation of the connective tissue. Their most frequent seat in the face is the eyelids, as already stated; and in that case the eye may itself be endangered. At the close of severe cases of erysipelas migrans, numerous abscesses may develop in the skin of the trunk and extremities, delaying convalescence.

The **diagnosis** of erysipelas is almost always easy when once the cutaneous lesion has developed. Phlegmonous inflammation of the skin and lymphangitis are to be eliminated; but this is always possible, with proper care. After a single examination, we may confound it with acute facial eczema of great severity, or even with a marked urticaria. Chief attention should be paid to the characteristic border of erysipelas and to its manner of extension.

The **prognosis** of facial erysipelas, when it attacks a healthy person, is very favorable. In drunkards a severe case may be complicated by delirium tremens, and the issue be unfavorable. We saw one case end fatally because of gangrene in the eyelids, followed by purulent inflammation of the orbital connective tissue. Erysipelas migrans may so exhaust the powers of feeble patients as to become dangerous. The prognosis of surgical erysipelas is relatively more unfavorable, but can not be considered here.

Treatment.—In a case of not more than average severity no special treatment is needed. To lessen the local discomfort, we usually cover the skin with powdered starch, or anoint it with olive-oil, carbolized oil, or vaseline. An ice-bag on the head is agreeable to most patients. If we wish to prescribe something, we may choose an acid mixture, as follows: Acid. muriat. dil., parts 8; syrup, 15; aquæ, 120. M.

In severe cases, however, the high fever and the nervous symptoms may demand our interference. The remedy to be chiefly recommended is cold baths, of which two or three may be given in a day, and which most patients bear very well. The exhibition of quinine, antipyrine, or antifebrin is seldom called for, as there is a tendency to great spontaneous intermissions in the fever. If the facial inflammation proves to be part of an erysipelas migrans, the chief indication for treatment would be to check the unceasing advance of the disease; but, unfortunately, the means recommended for this purpose too often fail. It used to be customary to cauterize the skin along the border of the erysipelas with nitrate of silver, but this has been almost entirely abandoned as useless. Hueter recommends the injection of a two-per-cent. solution of carbolic acid beneath the skin at a little distance from the border of the inflammation. Although this is certainly a rational method of treatment, we have seldom seen any brilliant results from it. Of more benefit seems to be the newly recommended scarification of the diseased surface, followed by the application of a solution of corrosive sublimate. For internal use we mention first Pirogoff's camphor treatment. The patient takes every hour or two three grains (0·15 grm.) of powdered camphor and drinks large quantities of hot tea, to promote perspiration. In severe cases this method deserves a trial. Numerous other internal remedies have been recommended; but we need not enumerate them. We have not seen any influence upon the extension of the disease exerted by large doses of salicylic acid or benzoate of soda. In England a prominent remedy is liquor ferri chloridi (in the form of tr. ferri chlorid.), given to the amount of drachms jss.–ijss. in the course of the day (6–10 grm.). In the severe cases the main point, after all, is to maintain the patient's strength by nursing and food. If cutaneous abscesses form, they should be opened promptly, when they usually soon heal.

CHAPTER X.

DIPHTHERIA.

(*Diphtheritis. Croup. Cynanche contagiosa.*)

Ætiology and General Pathology.—Clinically, "diphtheria" means a certain well-characterized, specific, acute, infectious disease, the chief visible lesion of which is a croupous-diphtheritic inflammation of the pharynx and upper air-passages. In a purely pathological sense, however, the terms "croupous" and "diphtheritic" have a broader meaning. They denote a certain form of inflammation which may occur in the mucous membrane of almost any part of the body. It is frequent in the intestine and bladder. There is great diversity in the causes which may produce it.

The pathological characteristic of croupous-diphtheritic inflammation consists in the formation of a fibrinous exudation. This may either be a croupous membrane, which is grayish white, rather firm, elastic, and can be lifted off with comparative ease from the mucous membrane upon which it rests, or it may be a diphtheritic infiltration with necrosis of the tissues. Here the exudation is more or less deeply imbedded within the proper structure of the mucous membrane itself. There is no essential difference between croup and diphtheria; diphtheritic inflammation is the severer form of the disease, croupous inflammation the milder. In diphtheria the fibrinous exudation is preceded by a necrosis of the epithelium and of the underlying tissues of the mucous membrane as well, while in the case of croupous exudation the necrosis is limited to the epithelium. The croupous membrane never rests upon an intact mucous surface, but replaces the epithelium, which has already been totally or in very large part destroyed. Flaky remnants of the epithelium, no longer nucleated, are sometimes found in the meshes of the fibrin. The preceding destruction of the epithelium is essential to the occurrence of fibrinous, croupous inflammation. The fibrinous exudation can be formed in those places only where the cause which excites the inflammation kills the epithelium at the same time. Apparently the epithelial cells have little if any share in the formation of the exudation. It is more probable that the material for the fibrin comes from the fibrinogen of the inflammatory matter which transudes through the walls of the vessels, and also from the disintegrated migratory white blood-globules. These last are abundant throughout the deposit itself, and still more numerous in the entire tissue of the mucous membrane beneath the croupous or diphtheritic exudation. If recovery takes place in croup, all that is needed after the exudation has been cast off is the renewal of the epithelium, which can be accomplished through the exclusive agency of the remnants of epithelium left along the borders of the diseased spot. In diphtheria, however, the entire necrotic portion of mucous membrane must slough off, a line of demarkation being formed, and cicatricial tissue replaces the necrosed portion.

The above is a bare outline of the present views about croupous and diphtheritic inflammations. They have been reached gradually through the labors of E. Wagner, Weigert, and others. We have not yet touched upon the ætiological factors; but what precedes renders it evident how manifold they may be, for many causes which destroy the epithelial layer of the mucous membrane, and at the same time promote inflammation, may excite croup. We have mechanical causes, such as impacted fæces, gall-stones, renal calculi; chemical irritants, caustics, like ammonia and the acids; and, finally, a number of specific, infectious, disease-producing poisons. Among these is the specific poison of diphtheria proper.

Beyond a doubt the diphtheritic poison is organized. To demonstrate this, however, has been thus far extremely difficult, for there are in the diseased spots a great number of diverse micro-organisms, originating in the mouth and throat, and really secondary to the diphtheritic process; but, although they of course are entirely different from the specific "diphtheritic bacteria," it is very hard to separate them. The latest systematic investigations of Löffler have made us acquainted with a bacillus which can be found in most cases of diphtheria, while at other times it is very rarely found in the mouth. Löffler's bacilli are little cylinders with a peculiar club-like swelling at their ends. In croupous membranes they are found in colonies. Inoculated upon animals, they have a decidedly pathogenic power, and produce a disease similar to diphtheria. That they constitute the long-sought diphtheritic poison is therefore probable, but it is by no means conclusively proved.

Diphtheria is chiefly a disease of childhood. It is much less frequent in those over ten years of age than in earlier life. In the larger towns sporadic cases are occurring all the time, but now and then the cases become so numerous as to be endemic or epidemic. As to the precise way in which a human being becomes infected the opinions of physicians differ. We think it most probable that the poison reaches the pharynx along with the inspired air or in some other way, and here penetrates into the mucous membrane. It is very rare for the larynx to be the point of entrance (*vide infra*). It first excites a local disturbance, to which the general infection of the system (*vide infra*) is only a sequel. At the same time secondary infection plays often an important part in diphtheria, just as in many other infectious diseases. The seat of the primary diphtheritic process promotes and perhaps gives the first opportunity for a colonization of other varieties of micro-organisms; and these change for the worse the original character of the disease. It is especially probable that many cases of so-called "septic diphtheria" (*vide infra*) are due to a complication of the primary simple diphtheria with a septic infection. A confirmatory and remarkable fact has been discovered by Löffler. He has found in the diseased tissues of the throat, and also in the internal viscera, in severe cases of diphtheria, the same "chain-building micrococcus" with which, as the cause of certain severe forms of scarlatinal angina (see p. 39), we have already become acquainted. According to A. Fränkel, this organism is identical with the streptococcus pyogenes.

The original source of the infectious material is probably always some other case of diphtheria. Sometimes a direct, immediate transmission of the poison (contagion) is extremely probable—e. g., due to coughing, or to sucking out bits of membrane after tracheotomy. The latter is a comparatively frequent cause of the disease in physicians and nurses. The infection is often spread by some person who may himself escape the disease, or by fomites, playthings and other objects, to which the poison is adherent. The potency of the infectious matter is not easily destroyed by such external influences as desiccation or changes of temperature. In other words, it possesses great "tenacity." As to what extent the diphtheritic poison may have a power of independent reproduction outside the body—e. g., in the ground, or in the floor of dwellings—we remain in complete ignorance. Finally, we should notice that of late attention has been called to the possibility of catching diphtheria from diseased animals. Poultry, doves, and calves have diseases that are at least similar to diphtheria.

[While it seems in the highest degree probable that the poison is usually purely local at the start, cases occur which suggest that constitutional infection through the pulmonary blood-vessels may precede the local manifestations. Infection through the alimentary canal is not probable, though its occurrence can not be positively denied.

There are still points in the ætiology and pathology of this affection which are involved in obscurity. Much has been said and written in this country and in England about the relations of filth and diphtheria. That filthy surroundings contribute a soil favorable to the development of the poison, and at the same time diminish the resisting power of the human organism, can not be doubted; but, as long as the parasitic theory of infectious diseases prevails, sewer-gas and the like must be classed among the predisposing or accessory causes.

Some of the frightfully virulent epidemics of diphtheria in sparsely settled country districts and on the Western plains are difficult to explain under the theory that each case is mediately or immediately the result of a previous case; these difficulties will, however, doubtless be cleared away in time.]

Clinical History.—The incubation is rather brief, seldom exceeding two to five days. The disease itself almost always begins with general malaise, headache, fever, and pain on swallowing. Little children, however, often do not complain of this last symptom, and in older children the sore throat may not be very troublesome at first. It is therefore a very important rule for the physician to examine the throat carefully in every child who presents ill-defined general symptoms. If diphtheria is beginning, we find redness of the soft palate, and more or less swelling of the tonsils. Upon the inner surface of the latter, and perhaps upon the arch of the palate and the uvula also, are spots covered with a grayish-white coating, which is quite firmly adherent to the mucous membrane. They are less frequent upon the posterior wall of the pharynx and the hard palate. Their extent varies greatly in different cases. In the mildest they are chiefly confined to the tonsils, attacking the soft palate or the tonsillar surface of the uvula but little if at all. In severer attacks the spread of the false membrane during the first days of illness is rapid. Almost invariably there is a very early and considerable swelling of the lymph-glands at the angle of the jaw. The constitutional symptoms persist. The children are restless. There is complete anorexia, and frequently vomiting. The temperature-curve is not typical. It is irregular, but is often rather elevated, reaching 104° (40° C.) or more. On the other hand, fever may be slight or almost absent, even in the worst cases. The pulse is very rapid. The urine may have a trace of albumen.

In mild cases the local and constitutional symptoms remain moderate; and at the end of a week or ten days there is decided improvement, with rapid convalescence. In severe cases, however, dangerous symptoms appear, perhaps early; the croupous inflammation involves neighboring organs, or a severe constitutional infection is developed.

The diphtheria very frequently extends into the nose. This "diphtheritic coryza," though not in itself dangerous, is usually a sign that the case is a severe one. The inflammation of the nasal mucous membrane may be simply mucopurulent, but it may also be croupous. It is betrayed by the abundant purulent discharge. Excoriations and superficial ulcers are usually soon produced at the edge of the nostrils. There may be nose-bleed.

A much more dangerous complication is the extension of the process into the larynx. This creates a mechanical hindrance to respiration, which proves fatal in a great many cases, as the child's larynx is so small. Formerly "croup"—i. e., croupous inflammation of the larynx—was regarded as a different disease from diphtheria. Many specialists in children's diseases still maintain this view; but it is in direct opposition to the teachings of pathological anatomy as well as of the clinical symptoms. We grant that there are cases where the pharynx is slightly affected, while the croupous inflammation of the larynx is extreme; and once in a great while the diphtheritic infection results in croupous laryngitis and tracheitis alone, the pharynx escaping disease. Still the proposition that there are two distinct diseases, "croup" and "diphtheria," is absolutely untenable. In the overwhelming majority of cases the throat is first affected and then the larynx. We should also consider how easily slight lesions in the pharynx might be overlooked, especially if located upon the posterior surface of the soft palate or upon the epiglottis. Cases of what is called "ascending croup," in which the laryngeal affection precedes the appearance of the disease in the pharynx, are, to say the least, very exceptional.

Usually hoarseness is the first indication that the diphtheria has attacked the larynx. Then follows the peculiar, harsh, ringing, "croupy cough," so dreaded by the parents, and, finally, there are signs of beginning laryngeal stenosis. Respiration is not much accelerated, but is labored, and the accessory muscles of respiration are called more and more into action. The child becomes more restless and anxious. Its face grows pale and livid. The chief cause of the dyspnœa is undoubtedly the mechanical stenosis due to the croupous deposit. Paralysis of the laryngeal muscles may perhaps be a factor. If portions of the false membrane become partially detached, they may act like valves, being sucked in at each inspiration, and pushed aside by the current of expired air. If stenosis occurs, respiration becomes noisy, resembling snoring. Inspiration, particularly, is prolonged and "sawing," and is attended by marked depression of the larynx toward the sternum. An important diagnostic point is the drawing in during inspiration of the supra-sternal region, the epigastrium, and the lower part of the sides of the thorax. This is the direct result of the obstructed flow of air into the lungs. As the lungs do not expand enough to correspond to the inspiratory dilatation of the thorax, the parts mentioned are forced in by atmospheric pressure. The degree of dyspnœa may vary at different times. The false membrane may be loosened and coughed up, rendering respiration easier for a time, till fresh exudations or displacements of membrane cause renewed distress. Recovery is still possible. The membrane may be expectorated and no more be formed. Unfortunately, this happy termination rarely occurs. In most cases the symptoms of suffocation increase more and more, respiration grows quicker and more superficial, and the child becomes more and more stupefied by the excess of carbonic dioxide in the blood. The pulse gets very small, rapid, and irregular. There are mild convulsions and then death.

The autopsy in these cases discloses usually that the croupous inflammation has extended into the larger bronchi or even into the smaller. The lumen of the bronchioles may be almost completely occluded by false membrane. There is also sometimes a genuine croupous inflammation of the pulmonary parenchyma. Lobular pneumonia in the lower lobes is much more frequent, however. It is probably secondary and to be regarded as a pneumonia from inhalation. During life the pulmonary complications may be suspected, but can hardly be diagnosticated. If we hear abundant moist râles over the lower lobes we are usually justified in supposing that lobular infiltration exists, even if there be no dullness on percussion. The croupous bronchitis as such gives rise to no especial auscultatory signs. If it is very extensive and reaches into the smaller bronchi, it may prove fatal, even if there be no laryngeal stenosis. This is more apt to occur in adults.

Another danger from diphtheria is through the general infection of the system, which may cause a fatal result. Although diphtheria does seem to start as a local disease, yet infectious matter (or poisonous substances) are certainly absorbed from the primary lesion into the general system. These exert their deleterious influences mainly upon the nervous system. We have already pointed out (p. 66) that the exact interpretation of these phenomena is rendered difficult by our inability as yet to distinguish between the effects of the primary and of the secondary infection of the body. Of especial practical importance are those cases in which we see the child sink into somnolence, and finally into complete sopor; its pulse becomes weaker and weaker, and more and more rapid, reaching 200 or more, and at last there is "paralysis of the heart" and death. All this occurs without any great degree of laryngeal stenosis. Such cases of severe constitutional infection, or "septic diphtheria," are most frequent when the local disorder in the pharynx is of unusual intensity, and the croupous exudation is replaced by deeper-reaching, necrotic, or even gangrenous inflammation within the mucous membrane. This has been called "gangrenous diphtheria." In such cases, also, the cervical lymph-glands are usually intensely inflamed. It must be noted that, exceptionally, a comparatively mild local disorder may co-exist with the worst general symptoms. A question of great interest, but which can not yet be answered, here presents itself. It is whether the constitutional symptoms are in every case directly dependent upon the diphtheria, or whether the diphtheria is not re-enforced by a peculiar, secondary, septic infection, proceeding from the diphtheritic ulcerations. We regard the latter view as probable. In adults the constitutional infection is a prime factor of danger, for in them laryngeal stenosis is less apt to occur, as the parts are so much larger than in children.

As to the part which the other organs play in diphtheria, we should mention that the process may extend not only into the nose and larynx, but into the Eustachian tube and the middle ear. It may also attack the anterior portion of the mouth, the gums, and the lips, or it may travel through the nose and the nasal ducts to the conjunctivæ. Very exceptionally the croupous process extends into the œsophagus. The infectious matter may be transferred, by the finger or in some similar way, to excoriations or accidental lesions of the skin, and excite diphtheritic exudations upon them. The frequent cases of inflammation of the eyes probably originate in the same manner, and also the diphtheria of the external genitals seen in children. With regard to other organs, it should be mentioned that there is not infrequently a splenic tumor. Intestinal complications (diarrhœa) are generally rare; nor is swelling of the joints frequent. The heart and kidneys, however, always need watching. It has already been stated that in severe cases of diphtheria the pulse becomes remarkably small and rapid. It is also often irregular. Further, even in cases that otherwise seem mild, sudden cardiac failure may occur, ushered in by excessive rapidity of the pulse, and often proving quickly fatal. This misfortune often happens at a time when convalescence seems to be fully established. Small islets of myocarditis are frequently found in diphtheria, but it is probable that the collapse is not so much due to them as to derangement of the cardiac nerves, particularly the vagus (*vide infra*). Renal disturbance betrays itself through the greater or less degree of albuminuria existing in most of the severe cases. It usually appears when the disease is at its height, less often at a later period. We often find a few casts in the urine, but seldom much blood. Œdema is rare. At the autopsy there is generally little macroscopic alteration of the kidneys. The microscope reveals the ordinary degenerative changes of acute nephritis (*vide infra*).

The Nervous Sequelæ of Diphtheria.—The convalescent from diphtheria is liable to be attacked by certain nervous sequelæ. Of these, diphtheritic paralysis is the most important. It appears about one or two weeks after the throat trouble ceases, or perhaps earlier, and it is quite as likely to follow mild cases as severe ones. It attacks the soft palate by preference. The tone becomes nasal and deglutition difficult. The naso-pharynx is imperfectly cut off during the act of swallowing, and with each attempt liquid regurgitates through the nose. Usually the pharyngeal mucous membrane is anæsthetic at the same time, and deprived of its reflex excitability. There may also be paralysis of the vocal cords upon one or both sides, and this again is frequently combined with anæsthesia of the mucous membrane of the throat. There may be paralysis of the ocular muscles, of which those controlling accommodation are most apt to be affected, rendering the vision for near objects imperfect. Paralysis of the muscles of the trunk and extremities is least frequent, but it may be very extensive. Sometimes several of these parts are paralyzed simultaneously. Thus we see quite often paralysis of the soft palate and of the muscles of accommodation combined. In some cases there is well-marked ataxia of the lower limbs with or without paresis. This renders the gait very uncertain and tottering, the tendon reflex is almost always abolished, while sensation is affected slightly if at all. Very rarely diphtheria is followed by contracture of the hands or other parts, by difficulty in articulation and paresis of the bladder. A paralysis of the pharynx is sometimes left behind, so that the children have to be fed for weeks through an œsophageal tube. It is a remarkable fact that not only in almost every case of the nervous disorders which we have mentioned, but often also in individuals who have entirely escaped them, there is no patellar reflex after diphtheria for weeks or even months. With regard to the pathological state, it is probably a degenerative condition of the corresponding peripheral nerves, not only in the post-diphtheritic paralysis, but also in the cases of post-diphtheritic ataxia. This harmonizes with the usually favorable termination of the nervous sequelæ of diphtheria. But there is one paralysis which is highly dangerous—that of the heart, as already mentioned. It may occur suddenly during convalescence. Probably it is analogous to the other nervous derangements, and the result of degeneration in the fibers of the pneumogastric.

Diagnosis.—The physician will seldom mistake a case of actual diphtheria if he pays proper attention. The characteristic deposit and the severe general and local symptoms make the diagnosis certain. It is much more common to take other forms of sore throat, particularly in adults, for diphtheria. The most deceptive are follicular and necrotic tonsillitis (*vide infra*). We must not suppose that every white spot upon the tonsils is diphtheritic. The above-mentioned forms of sore throat are, however, frequent during epidemics of diphtheria, and even, as we have often observed of late years, in families where there are simultaneously cases of genuine diphtheria; so that the thought is suggested that they may ætiologically have some relation to true diphtheria. It is at any rate advisable not to omit proper precautionary measures, especially if there are children about.

[When the membranes are confined to the nose, the diagnosis may be more or less difficult; but it is especially in cases in which the nasal mucous membrane is involved that we encounter great swelling of the glands at the angles of the jaw. There is also apt to be a thin, acrid, bloody, or sero-purulent discharge.

Jacobi states that while diffuse pharyngeal injection may or may not point to imminent diphtheria, marked local congestion is either traumatic or diphtheritic. An examination of the urine should never be neglected in doubtful cases: in diphtheria a trace of albumen is very common; in simple or follicular sore throat, albumen is very rare, if indeed, it occurs at all.]

Prognosis.—The unfavorable prognosis of the disease is universally known, even by the laity. The very fact that the best developed and healthiest children so often fall victims to it associates the name diphtheria with the saddest memories. There are indeed many mild cases which recover in a week or two, and severer ones which end happily in three or four weeks; but in most cases, where the process extends into the larynx, or the symptoms of a severe constitutional infection occur, medical interference has, unfortunately, no power to control the unfavorable issue. What the dangers are, and how recognized, can be well enough inferred from the preceding description of the symptoms. We will only remind the reader how carefully the physician should watch the behavior of the heart, since danger is apt to arise from this source, even when the case seems otherwise to be taking a favorable course.

Treatment.—If we take the ground that diphtheria begins as a merely local process, then local treatment of it certainly seems rational, at least at first. Unfortunately, the practical result bears out the theory very imperfectly. An actual and complete destruction of the croupous exudation is but seldom possible; and the attempt to accomplish this in a struggling child is so difficult and disagreeable that to-day most physicians have entirely abandoned the application with a brush of caustics or other substances to the throat. If it does, nevertheless, seem desirable to try energetic local measures at the commencement of the disease, the best agents to choose are a concentrated solution of argentic nitrate (1–10), or a solution of corrosive sublimate (1–1,000), or a mixture containing equal parts of carbolic acid and alcohol. If the disease has already made some progress, we may well spare the patient needless torture and consider that, by destroying the mucous membrane and by wiping off the exudation, we are likely to contribute to a further extension of the diphtheritic process. The author has not used energetic local treatment for years, regarding it as a useless disturbance of the patient.

We do not, therefore, regard active local interference as justifiable except at the very beginning of the disease; but we do believe that both then and at a later period it is extremely desirable to disinfect the mouth and throat as thoroughly as possible. Although this has little effect upon the diphtheria itself, it is at least a factor in preventing secondary septic infection, and thus of great therapeutic importance. Adults and older children should rinse the mouth and gargle frequently, using disinfectant solutions, e. g., of potassic chlorate or carbolic acid. Often, however, gargling proves painful. Inhalations are very appropriate. They are usually well borne when either lime-water (diluted with equal parts of distilled water) or simple salt solution is used, while the stronger disinfectants, such as a one-per-cent. or two-per-cent. solution of carbolic acid, often cause a burning sensation. Nevertheless, it is advisable to keep up a carbolic spray in the neighborhood of the patient. We may sometimes try rinsing out the mouth with a fountain syringe, using for the purpose a weak solution of salicylic acid or some similar antiseptic. The frequent introduction of a few teaspoonfuls of cold water into the nose has also been recommended as an appropriate treatment—"cold nasal bath."

We shall mention only a few of the numerous other remedies which have been recommended. There is another local remedy, papayotin, which is obtained from the milky juice of a certain plant, and has the property of digesting albumen. If a diphtheritic exudation be frequently touched with a five-per-cent. solution of this, it will sometimes disappear rapidly; but the drug can not be shown to have an active influence upon the disease itself. Of internal remedies, we should mention potassic chlorate, which has been much vaunted as a specific, when given internally in rather large doses. We recommend it, but it should be used as follows: a half-teaspoonful of a two- or three-per-cent. solution should be slowly swallowed about every half-hour. The aim is to obtain, not a constitutional, but a local antiseptic action. It should not be given in larger amounts than drachm j-jss. (grm. 5–6) in twenty-four hours, lest it cause hæmoglobinuria or other toxic symptoms. Several physicians have lately recommended spirits of turpentine very highly, one half to one teaspoonful being given several times a day. It has not become popular. The same may be said of the internal use of potassic iodide. Injections of pilocarpine have also been praised. They are said to promote the detachment of the false membrane; but their efficacy is doubtful.

[The tincture of the chloride of iron is much used in this country in the treatment of diphtheria, and appears to be of real service; but it must be given in large doses. The following prescription is recommended by Jacobi, whose experience has been very large, for a child of two years:

℞ Tinct. ferri chloridi	ʒ ij;
Potas. chlorat.	gr. xx;
Glycerin. pur	℥ j;
Aquæ	℥ v.

M. S.: Teaspoonful every fifteen, twenty, or thirty minutes.

Turpentine is better as an inhalation than by the stomach; a teaspoonful or two of the oil can be poured in water kept at the boiling point by an alcohol-lamp. The whole air of the room is thus charged with the remedy. No drug should be used which disorders the stomach. Tablet triturates containing one one thousandth of a grain of corrosive sublimate can be allowed to melt in the mouth with the greatest freedom, and seem sometimes to exert a distinctly beneficial local action. The dose is, however, too small to secure the systemic effects of the drug unless the case is a mild one and free from notable dysphagia.]

If the larynx is attacked, and the consequent laryngeal stenosis threatens to cause suffocation, tracheotomy is our only resort. It is never indicated by the disease itself nor by the severity of the case, but only by persistent obstruction of the larynx. It is therefore not invariably easy to decide whether tracheotomy is called for in any particular case. If the general condition be bad and respiration already impaired, it may be very difficult to determine whether laryngeal stenosis exists. Tracheotomy will be of no avail if the croup has already extended to the bronchi, or if the dangerous condition of the patient is due to the severity of the constitutional infection or to incipient paralysis of the heart. This explains why the results of tracheotomy are not remarkably brilliant. On an average, only about one third or one fourth of the cases operated upon get well; but even this number is enough to make us prize the operation very highly. How it is performed, and in what the after-treatment consists, must be learned in the text-books on surgery.

The attempt to expel the false membrane from the larynx by inducing vomiting is still often made, but seldom succeeds, and tortures and exhausts the child. Warm baths with cold douches may prove very beneficial. They excite deep respiration and more vigorous coughing, and also tone up the whole nervous system. The wet pack is also often employed, and sometimes with great benefit. Outward applications upon the throat are of little use. In general, we prefer the cold, wet compress to the ice-bag and ice-poultice, which are likewise often employed.

[Dr. Geo. W. Gay says ("Phila. Med. Times," 1884): "Not a single case of pseudo-membranous laryngitis has ever recovered in the Boston City Hospital without operation." In twenty years tracheotomy has been done one hundred and eighteen times with thirty-nine recoveries.

Four, if not five, successful cases were practically moribund at the time of operation.

Lovett and Munro bring the Boston City Hospital statistics down to Jan. 1, 1887. Tracheotomy for croup was done three hundred and twenty-seven times, all the cases but thirty dating from 1880. There was recovery in ninety-five, or 29·05 per cent. "The following table shows the recovery rate in cases operated upon within one, two, three, and four days after the beginning of the obstructed respiration":

Day of operation.	Number of cases.	Recovery.	Per cent. of recovery.
1	123	40	32·5
2	86	24	28·0
3	33	8	25·3
4	7	1	14·0

"Since Dr. Gay's article was written there have been two recoveries from moderate dyspnœa without operation. . . . Of forty-two patients under two years of age only three recovered."

Intubation, as devised by Dr. O'Dwyer, is a procedure which makes a distinct advance in the treatment of laryngeal stenosis. In permitting the free access of air to the trachea, intubation may save life without resort to the serious operation of tracheotomy; or it may tide over a time until tracheotomy becomes absolutely necessary or until the consent of the parents can be obtained to the use of the knife. In hopeless cases it may promote euthanasia. Of eight hundred and six cases collected and analyzed by Dillon Brown two hundred and twenty-one recovered, 27·4 per cent.]

In most cases of septic diphtheria, treatment usually proves completely futile. We must seek to avert cardiac paralysis as well as we can by stimulants, such as wine, camphor, and strophanthus, and endeavor to improve respiration and the condition of the nervous system by lukewarm baths combined with douches. Finally, we repeat that the physician should never neglect to maintain the patient's strength, as far as possible, by proper nourishment.

The nervous sequelæ of diphtheria are best treated with the constant current. As an internal remedy, iron is good, and also nux vomica or strychnine. The last may be given subcutaneously, if desired, in doses of gr. $\frac{1}{60}-\frac{1}{30}$ (grm. 0·001–0·002).

[Diphtheria is a disease which involves commonly much exhaustion, and too much stress can hardly be laid on the importance of administering the maximum amount of nourishment in the most assimilable and easily swallowed forms from the start.

It is also important to give stimulants early in most cases, not waiting for signs of exhaustion. Enormous quantities of brandy can often be given to small children without the slightest toxic effect. No general rule can be laid down; the requirements of each case must be studied and met.

When painful deglutition interferes with nutrition, peptonized milk, eggs, brandy, and the like, must be given by the rectum. Rectal alimentation and stimulation are also to be resorted to in cases of post-diphtheritic paralysis of the œsophagus.]

CHAPTER XI.

INFLUENZA.

(*La Grippe.*)

Influenza is a specific, acute, infectious disease which is especially distinguished by the enormous extent of its epidemics. While often years and decades pass without any especial attention being called to the disease, suddenly cases of it will appear with such frequency that the largest part of the population are attacked, and the disease may better be described as pandemic than epidemic. Pandemics of influenza can be traced back with certainty into the sixteenth century. In the present century the influenza during the years 1830–'33 traversed almost all ot Asia and of Europe, then later there appeared numerous smaller epidemics, but these aroused general attention so little that the disease, upon its last pandemic appearance in the winter of 1889–'90 was almost unknown.

Ætiology.—Although we have every reason to suppose that the true cause of influenza is the infection of the body with a specific, organized pathogenic germ, yet this latter has as yet evaded discovery. It is evident that we have to do with some micro-organism which at certain times appears over an immense territory, the spores of which are probably scattered by the wind over large areas and are inspired with the atmospheric air by human beings. Many observations upon the appearance of the disease in isolated institutions (convents and the like) render it quite probable that the poison may also be carried by a person suffering from the influenza to regions previously unaffected. Nevertheless, this contagious manner of spreading plays no great *rôle* in comparison with the direct infection from the outer world, this latter mode being everywhere possible during an epidemic of influenza.

There is scarcely any reason for speaking of especial predisposing causes of influenza, inasmuch as at the time of a well-marked epidemic the overwhelming majority of the population are attacked, both the healthy and the diseased, the vigorous and the feeble. Sex certainly makes no difference, and age only to this extent, that the disease is seen more rarely in little children under one year old than in older children and adults. That catching cold has no special ætiological significance is evident from the fact that influenza often appears in patients who are already sick in bed.

It should finally be mentioned that animals also, and in particular horses, may be attacked by the influenza; but, nevertheless, it is as yet a doubtful question whether all the diseases in animals which are described under this name are actually identical with genuine influenza.

Symptoms and Clinical History.—The best general idea of the extremely manifold symptoms of the disease is to be obtained if we bear in mind that the influenza causes both a marked infectious (or toxic) general constitutional disturbance of the body, and also certain local lesions with local symptoms. The clinical picture therefore varies greatly according to the predominance of one or the other group of symptoms, and also according to the special form of the local disease.

The onset of influenza is generally rather sudden. As a rule the marked cases begin with rather high fever, ushered in with a chill, violent headache, marked constitutional depression, and usually considerable pain in the back and loins. The weakness of the patient may be so great that, even if a vigorous individual, he will at once take to his bed. Severe nervous symptoms, such as stupor and delirium, are exceptional. Sometimes, but not very often, there is initial vomiting. The pains in the back are often associated with pains in the muscles and joints. Oppressive pain in the eyes is quite characteristic also. This is particularly felt upon moving the eyeballs, and therefore is probably located in the external muscles. The spleen is sometimes somewhat swollen, but any great increase in its size is exceptional.

If the clinical symptoms as the case progresses are mainly limited to the above-named constitutional symptoms—fever, languor, headache, pain in the muscles, we may speak of a typhoid form of the disease. Usually, however, certain local symptoms put in an early appearance, and it is especially the respiratory apparatus which is attacked. The precise symptoms vary considerably in different cases. Sometimes the upper portion of the respiratory tract, the nose, larynx, and trachea are involved; sometimes, from the start, the smaller bronchi. In the first instance there is marked coryza or hoarseness, in the other case there is cough, due to a dry bronchitis, which can be easily detected upon auscultation, and which involves especially the lower portion of the lungs. If these local symptoms outweigh the constitutional, the case is described as belonging to the "catarrhal form of influenza."

Sometimes the influenza is localized in the digestive apparatus. This "gastro-intestinal form" is much rarer than the catarrhal. In this case, in addition to the more or less strongly characterized constitutional symptoms, there is marked disturbance of the stomach and intestines, as shown particularly by nausea with persistent vomiting, diarrhœa, abdominal pain, etc. In one case we observed jaundice. We may also mention in this connection the appearance of an initiatory pharyngitis.

The already mentioned pains in the back, loins, and extremities may persist with unusual violence, and this peculiar form of the disease is known as the "rheumatoid." Probably the muscles and the muscular attachments are the chief seat of these pains, which may be so violent that the patient does not know how to lie, and sometimes keeps up a continual moaning. The loins, in particular, may be the seat of most acute pain, also the upper arms, the knees, the thighs, and the eyes. Objective changes in the painful parts, such as swelling of the joints, are scarcely ever seen, nor are the nerve-trunks as a rule especially sensitive to pressure. The painful muscles are usually weaker than normal.

The grouping of the clinical varieties of influenza under the four forms already named renders it easier to obtain a general idea of the manifold symptoms of the disease, but this division into separate forms must not be carried out too strenuously, for in reality numerous cases of the disease occur which present transition forms and combinations of the various groups of symptoms. Moreover, in all the forms a distinction must be made between mild and severe attacks, for in influenza, just as in most other infectious diseases, there are numerous rudimentary and mild cases as well as the fully developed ones, and some could not be properly interpreted but for the presence of the epidemic.

The duration of the disease is best determined by the duration of the fever. In the very mildest cases there may be no fever whatever, or simply a slight evening rise of temperature. As a rule, there is a moderate fever, say between 101·5° and 103° F. (38·5° and 39·5° C.), although higher temperatures even to 104° F. (40° C.) and more are not infrequent. In the beginning of every severe attack the fever rises abruptly. After a duration of several days, say four to seven, it may fall again in a manner approaching a crisis. More frequently, especially when there exists diffuse, catarrhal trouble in the lungs, the fever ends by lysis. With comparative frequency there are found to be noticeable deviations in the temperature curve; thus, for example, the high fever of the onset sinks on the second or third day, to be followed by an almost afebrile period of one or two days, whereupon a marked rise of temperature ensues. With this change in the temperature there are usually also corresponding variations in other symptoms.

We see, then, that the duration of simple, uncomplicated influenza is in the mild cases about three or four days, in the severer cases about seven to ten days. To be sure, we should also consider in this connection that convalescence is often surprisingly slow, so that the after-pains (as it were) of the disease are felt for weeks. These consist, for instance, in a certain degree of debility, and in painfulness of the muscles. Sometimes also there are complete relapses, so that directly or a short time after the disease has ended the symptoms begin anew. The special form of the disease may change in this case, so that, for example, the relapse of an influenza with predominant constitutional symptoms assumes the pronounced catarrhal form. Again, during the same epidemic it is not very rare for a patient to suffer from two attacks of influenza separated by a considerable interval of time.

Complications and Sequelæ.—While all the symptoms of influenza which we have thus far described are the direct effects of the original pathogenic cause, the majority of the frequent complications are undoubtedly dependent upon the ingress of secondary infection. The system when attacked by influenza is greatly exposed to these secondary influences, and almost all the dangerous and tedious cases of influenza become such only because of a mixed infection of this sort. This is particularly true of the lungs, in which secondary disease occurs most frequently—sometimes even in the first days of illness, but also in other cases later. The conditions here are similar to those seen in measles and whooping-cough. The simple, mild catarrh belongs to the original disease; the severe pulmonary affections are, however, invariably secondary complications occasioned by new pathogenic influences. These influences are not always the same. According to the investigations of Ribbert, Finkler, and others it is chiefly the pneumonia diplococcus and the streptococcus which are the true excitants of the secondary pneumonia seen in influenza. These cases of pneumonia are either extensive catarrhal pneumonia especially affecting the lower lobes, or more rarely croupous pneumonia with its characteristic sputum. The whole picture is then overshadowed by the pulmonary affection. The patient is oppressed for breath, has a severe cough, looks pale and cyanotic, and suffers from high fever. These symptoms persist for two or three weeks, and then gradually abate. It is in this way that influenza becomes dangerous for elderly and feeble or sickly persons. With noticeable frequency, pleurisy with effusion is conjoined with the influenza pneumonia. The exudation is generally serous, but exceptionally it is purulent.

Complications in other organs are less frequent. We should mention chiefly purulent otitis media and keratitis and other severe diseases of the eye. We have several times observed cases of acute nephritis, but this has always pursued a mild course. Among cutaneous eruptions herpes labialis is a frequent phenomenon in all forms of influenza, even the milder. Other exanthems, such as urticaria and roseola, are much less frequent. Many of the complications named may continue even after the fever and all other symptoms have ceased, so that they must be regarded as sequelæ. This is particularly true of the diseases of the ear and eye and of persistent bronchitis, but only rarely of a pneumonia assuming the chronic form. An important and for the patient a troublesome and painful sequel is furunculosis, especially if the separate boils are located in the axilla or near the anus. Very often neuralgic pains in the distribution of the trigeminus or in the course of the sciatic or other nerves will persist for a considerable time after the influenza has ceased. But these pains may sometimes be located in the muscles; thus, for example, the frequent, persistent, and troublesome pain in the eyes.

Diagnosis.—The diagnosis of influenza is in general not difficult if one has to deal with a well-marked case. The characteristic initial symptoms of fever, headache, and pain in the loins are to be considered first of all. Their onset is much more rapid than, for example, in typhoid fever. Later on the pain in the various muscles as well as the catarrhal symptoms are the most characteristic phenomena. The diagnosis is very uncertain in many mild rudimentary cases. Often it is merely the fact of the prevailing epidemic that justifies one in calling these influenza. Nevertheless, one should be very careful not to make a diagnosis too quickly, and it is indubitable that during the time of an epidemic many cases are wrongly called influenza.

Prognosis.—For an individual who is healthy and vigorous, influenza is not a dangerous disease, even in its severer forms; for elderly persons or invalids it may, however, be a serious affection. Patients with heart disease or pulmonary disease, or those suffering from chronic nervous troubles, sometimes succumb to it; so that the general mortality at the time of a great epidemic of influenza is always considerably increased. The above-enumerated pulmonary complications are by far the most dangerous; less often is a fatal termination caused by general or cardiac weakness.

Treatment.—No specific remedy for the disease is known. Many physicians maintain that the exhibition of calomel at the beginning of the attack decidedly shortens its course, but confirmatory experience is wanting. In general we must, therefore, pursue a purely symptomatic method of treatment. For the initiatory fever, the headache, and the pain in the loins, antipyrine is sometimes a good remedy, and the same may be said also of quinine, phenacetine, and antifebrin. These drugs are also prescribed for the persistent pains in the muscles which come on later. Soothing liniments and ointments may be employed, with friction, for the same purpose. The treatment of the pulmonary complications is according to the established methods. Morphine is mainly used for the troublesome cough. Apomorphine, senega, and other expectorants may be employed, and, if indicated, external remedies such as an ice-bag or dry cupping. If the patient becomes very feeble, stimulants such as champagne and strophanthus are demanded.

CHAPTER XII.

DYSENTERY.

Ætiology.—By "dysentery" is meant a disease of the colon, which appears sporadically, but more often in epidemics; it is excited by infection with an organized pathogenic poison, about which we have as yet no further knowledge; and the infection is probably at first a local one. The true home of dysentery is in warmer and tropical countries, where the disease is much more violent and wide-spread than here. For example, the mortality among the soldiers of the Anglo-Indian army due to dysentery is said to be thirty per cent. of the entire number of deaths. In our climate most of the epidemics occur at the end of summer and in autumn. Endemic influences are certainly important. The condition of the soil in some places is evidently very favorable for the development and dissemination of dysenteric germs, and that of other places is equally unfavorable. There can be no other explanation of the immunity of some localities contrasting with the great prevalence of the disease in others. How infection occurs we do not yet know. Dysentery does not seem to be directly contagious; but that it can be spread through the medium of the fæcal dejections of the sick—e. g., from privies, chamber-vessels, and bed-linen—is very probable. Many cases were formerly referred to catching cold or to some error in diet; but we must, of course, regard these merely as predisposing influences.

The objective **pathological lesion** of the colon, in all severe cases, consists in a pronounced croupous-diphtheritic inflammation. The remarks as to the general pathology of such inflammations made in the preceding chapter are equally applicable to the analogous dysenteric inflammation. In this case, too, there is first a destruction of the epithelium and then the formation of a fibrinous exudation occupying its place, and penetrating down into the tissue of the mucous membrane itself. At the same time there is an intense purulent infiltration of the mucous and submucous tissue, accompanied by extensive ecchymoses. In the most virulent cases the macroscopic appearances are marked thickening of the whole wall of the intestine, congestion of the serous layer, and the conversion of the inner surface into a mottled, dark-red, irregularly roughened area of ulceration. The disease may be confined to the rectum and the sigmoid flexure, but in severer cases it involves the entire colon as far as the ileo-cæcal valve, or even extends to the lower portion of the ileum. Besides this severe form of diphtheritic or even gangrenous dysentery, there is a milder variety, termed catarrhal dysentery. In this the mucous membrane is found in a state of intense purulent inflammation, with ecchymoses. Even here little masses of croupous exudation, which can be torn off, have replaced the epithelium; but they never form continuous layers of great extent. There is no sharp boundary-line between the two forms, the milder catarrhal-croupous and the severer diphtheritic dysentery. Numerous transitional and combined varieties exist.

We must remark, in conclusion, that precisely the same anatomical changes as are presented in true dysentery may result from other causes. Important among these is persistent fæcal impaction in the rectum, which, by a purely mechanical effect upon the epithelium, may excite a diphtheritic inflammation in the mucous membrane. And any severe constitutional disease whatsoever, such as typhoid fever, measles, small-pox, septicæmia, or phthisis, may be attended by a so-called "secondary dysentery." This is most frequent in hospitals. Whether it has the same ætiology as genuine dysentery is uncertain.

Clinical History.—Throughout the entire illness the most prominent symptoms are intestinal. There may be first of all some slight irregularity of the bowels for a few days, and then appears a moderate diarrhœa. The stools are at first feculent, although thin, and number two to six daily. After a few days the discharges increase in frequency, and become extremely characteristic.

The stools are very frequent, occurring ten to twenty, and even sixty or more, times, in twenty-four hours. In severe cases there may be a distressing and almost constant desire to evacuate the bowels. After every operation, and to some extent during it, there is tenesmus attended by intense burning pain in the anus. The stools soon lose their usual feculent character in great part if not entirely. They become scanty, so that not more than about half an ounce is evacuated each time. For the most part they usually consist of a sero-mucous fluid, in which are suspended numerous shreds and particles of varying size. These are blood-stained bits of mucus, little coagula of blood, and necrosed pieces of mucous membrane. One or another of these constituent parts may predominate, so that there may be slimy, purulent, or bloody stools, or all sorts of combinations of these varieties. We often find, besides, a few small masses of fæces, usually covered with mucus. We sometimes see numerous clumps of mucus, resembling sago or frog's spawn; they are probably mucous casts of the follicles. Under the microscope the greater part of the dysenteric discharge is seen to consist of pus-corpuscles and blood. There are also cylinder epithelium and an enormous amount of detritus, and the bacteria of putrefaction. A purely dysenteric stool has no bad odor, except that in the worst cases of gangrenous dysentery the discharges become blackish and extremely offensive.

The rectal tenesmus may be accompanied by a cramp-like pain during micturition. There are often violent attacks of colic. The abdomen is usually rather tense, and tender on pressure along the line of the colon, but without tympanites. The anus may be red, inflamed, and excoriated. Gastric symptoms are on the whole infrequent, if we except the complete anorexia which exists in all severe cases. Sometimes there is repeated vomiting. Occasionally hiccoughs prove distressing. The tongue usually has a dry, greasy coating.

The symptoms just depicted last about a week or ten days. If the case is of much intensity, the general condition is also greatly affected. The patient seems much collapsed, and is very languid and feeble, with a small and rapid pulse. The skin becomes cool and rough, the voice weak and hoarse. There is pain in the muscles. The patient wastes away. The temperature has little that is characteristic or typical. In many cases there is no fever at all, and the temperature may even be subnormal. In most cases, however, there is an irregular fever, seldom exceeding 104° (40° C.), and having remissions.

In the worst cases the general weakness may increase more and more, and death occur; but with us a favorable termination is much more frequent. The distress gradually diminishes, the stools assume more and more of a feculent character, the patient becomes stronger, and after one and a half to three weeks convalescence is established. It may be a long while, however, before a patient completely recovers from a severe attack. A third possibility is the transition of the acute into a chronic dysentery. In this the symptoms of a chronic colitis, usually attended with cachexia, may persist for months and years.

Mild, rudimentary forms of dysentery also occur, presenting less violent intestinal symptoms, and recovering at the end of a few days. In these cases, too, great sensitiveness of the intestine to disturbing influences frequently persists for quite a long time after the illness. There may be exacerbations of the disease, and relapses.

Complications of dysentery, localized in other organs, are rare, at least in epidemics here. Abscess of the liver is mentioned oftenest by physicians in warm climates, and probably is best explained as the result of metastasis by way of the portal system. Articular disturbances also occur, and inflammation in the serous membranes. A few cases of peritonitis due to perforation have been observed, and a combination of dysentery and a "general scorbutic diathesis" has been described.

The **diagnosis** is rarely very difficult. It is based exclusively upon the intestinal symptoms and the character of the stools. It is only the cases of secondary dysentery which occur in the course of other severe diseases that can easily escape observation.

The **prognosis** is mainly influenced by the character of the epidemic, which, as we have said, is in our climate usually benign. There may be danger, particularly to elderly people, from bodily weakness and collapse.

Treatment.—Prophylaxis demands that the isolation of the patient and the disinfection of the stools be as complete as possible. The healthy must be very careful during an epidemic not to catch cold, and to avoid errors in diet, for experience shows that an opposite course predisposes to the disease.

The patient must be kept warm, and must not leave his bed, even if the attack be mild. The diet must be rigorous. If the strength is fair, thin porridge, milk, and broths suffice for some days. To a feebler person we should give somewhat stronger nourishment from the start, e. g., eggs, peptonized meat, and wine. Most patients bear liquids that are lukewarm better than those which are cold.

As to drugs, the habit of almost all experienced physicians is to give at first a mild laxative. Although opium does not usually control the diarrhœa and tenesmus at all, it is the rule for decided improvement to follow the exhibition of the laxative. During the first days, or, if need be, later, we give two to four tablespoonfuls of castor-oil daily. If this medicine is very disagreeable to the patient, we can replace it by a strong infusion of rhubarb (10–100). In southern countries large doses of calomel (gr. x to xv, grm. 0·5–1) are customary, and are highly praised by the physicians there. Further on in the disease we may content ourselves with giving mistura amygdalæ; or we may administer bismuth in the following mixture: Bismuthi subnit., parts 5; mucilaginis acaciæ, syrupi simpl., āā 15; aquæ destil., 120—to be shaken before taking. But if the disease should get worse again, we should always try a laxative.

Emetics at the beginning of the disease are often employed in the tropics, but seldom with us. Ipecacuanha (*radix antidysenterica*), given in large doses of fifteen to thirty grains (1–2 grm.), is even regarded by many as a specific. Numerous attempts have been made at local treatment by enemata. Yet no brilliant results can be claimed for any of these methods or medicines. A decidedly palliative effect can be obtained from the injection of thin starch to which twenty or thirty drops of laudanum have been added. Suppositories of cocoa butter containing extract of opium often mitigate the tenesmus. Other injections are recommended, each to measure ℥ ij to iijss. (grm. 60–100), and to contain either argenti nitrat., gr. j to vj (grm. 0·05–0·30), or plumbi acetat., gr. ij to viij (grm. 0·1–0·5), or potassii chlorat., gr. xv to xx (grm. 1–1·5). Many other solutions are used. The success of this treatment is, however, dubious. In all cases the margins of the anus must be protected from inflammation by frequently washing and anointing the skin.

The treatment of weakness and collapse is by the usual stimulants—wine, ether, camphor, and the like. In chronic dysentery the main point is to persevere in a strict control of the diet. We may exhibit astringents, such as tannin and columbo. Subnitrate of bismuth is also given, and nitrate of silver and acetate of lead. And in these chronic cases a long-continued and thorough use of rectal irrigation with fluids containing some mild astringent or disinfectant may have a good effect.

[Sporadic dysentery is a self-limited disease, and, as has been shown by Flint, runs its course within ten days without medication. Treatment, however, adds to the comfort of the patient and shortens the course. It is not customary with us to use daily laxatives. If there is any doubt as to whether the intestines have been emptied, a saline should be given, the action of which should be followed by opium in sufficient doses to allay pain and tenesmus. Subsequent action of the bowels is best obtained by simple large enemata. In weak persons castor-oil is to be preferred to salines.

In epidemic dysentery active treatment is much more important. Laxatives are contra-indicated by sero-sanguinolent dejections or by asthenia, but enemata can be freely used. Stimulation is often required; nutrition must be carefully looked after, such articles being chosen as are digested and absorbed by the upper portions of the intestinal tract, leaving as little residue as possible to pass on to the inflamed colon. Opium is often demanded and tolerated in large doses, and astringents, such as the acetate of lead, gallic acid, and the pernitrate of iron, are of service.

In acute dysentery the patient should be instructed not to yield to the desire to go to stool if he can help it, and tenesmus can often be much diminished by simple irrigation of the lower bowel with water, which may be warm or cold, whichever the patient finds more agreeable.

Chronic dysentery is one of the most difficult maladies with which we have to deal. In its treatment a sea voyage, or removal for at least some months to a climate other than that in which the disease originated, is of far more value than drugs.

AMŒBIC DYSENTERY.

The amœba coli, first found by Lösch in the stools of a dysenteric patient, has received very careful study at the Johns Hopkins Hospital, and forms the subject of a most exhaustive and valuable monograph by Councilman and Lafleur.

The living organism is readily seen, and recognized especially by its active amœboid movements on the warm stage of the microscope. If the fæces contain small gelatinous masses, these will be found to provide the most fruitful field for search. The numbers of the organisms vary widely in different cases, and even in the same case, from day to day. They are said to be present in this form of dysentery alone, and have been found in secondary abscesses of the liver and the lung, alike after death and during life. They should be sought for in all obstinate cases with dysenteric symptoms.

The prognosis is uncertain, and the cases are apt to drag along with exacerbations and remissions of the symptoms. But recovery does take place. There is notable danger of the formation of secondary abscesses, in which event recovery is hardly to be hoped for. The only treatment which seems to have been of much service—beyond a general hygienic and supportive regimen—consists in the use of copious injections into the intestinal canal of solutions of quinine, in the strength of one, to one thousand or even five thousand. Lösch found that contact with a solution of the latter strength for one minute suffices to kill the amœbæ.]

CHAPTER XIII.

CHOLERA.

(Asiatic Cholera.)

Historical Remarks.—The home of genuine Asiatic cholera is India. The first epidemic with which we are accurately acquainted, and which was very widespread, occurred in 1817. The disease was probably endemic there at an earlier period. In the next few years the cholera spread in all directions, and reached Astrakhan by way of Persia. Between 1830 and 1832 the disease made its first great epidemic progress over Europe. Invading all European Russia, it reached Germany in 1831, and France and England in 1832. Then came many smaller epidemics up to 1838, when there was a complete cessation till 1846, in which year the disease, again starting from Asia, overspread Europe. There have since then been epidemics in many places, but we can not here enter into the particulars of them. The last time that cholera occurred to any extent in Germany was in 1866, during the German-Austrian war. No one has forgotten the somewhat violent epidemic which prevailed a few years ago (1883 and 1884) in France and Italy.

Ætiology.—Some time ago it had become evident that the real cause of cholera consists in the infection of the system by a specific micro-organism. Koch was, however, the first to succeed in the search for the poisonous agent. He was in charge of the scientific expedition sent out by the German Government in 1883 to Egypt and India for the purpose of investigating the disease. Koch found in the intestines of all the victims of cholera whose bodies he examined a certain kind of micro-organism which he named the comma bacillus. It is shorter than the bacillus of tuberculosis, but somewhat thicker, and it is usually bent in the shape of a comma, or even like a semicircle (see Fig. 9). In the culture-preparations, the special peculiarities of which we can not give in detail, the comma bacilli grow into long spiral threads, resembling the spirilli of recurrent fever. Examined in a liquid, the individual bacilli are seen to make vigorous movements. They flourish best at a temperature between 86° and 104° (30° and 40° C.). Below 61° (16° C.) they cease to grow, but they are not killed even by a greater degree of cold. The free access of oxygen is absolutely indispensable to their growth. They multiply very rapidly in liquids—e. g., broth or milk—and they may, under favorable circumstances, retain their vitality for many weeks, while they can be readily destroyed by desiccation. In this again they resemble the genuine spirilli, which can maintain their existence only in fluids. An endogenous formation of spores, as we shall see below to be the case with the anthrax bacilli, does not take place, but Hueppe has established by actual observation that frequently two small spherical bodies appear on some indeterminate spot of the above-mentioned spiral threads, by which the continuity of the thread is interrupted. This process goes on until finally the thread is replaced by a considerable number of very minute round cells, which are apparently united to one another by a kind of jelly (formation of zoöglœa). It is certain that the round cells do not subdivide. The way in which they are formed corresponds with quite similar processes in other spirochætæ. They are called "arthrospores," and perhaps represent a permanent form of the comma bacilli; for they resist desiccation and other injurious influences much better than do the comma bacilli themselves. By a process of growth the spherical bodies may, under favorable circumstances, give rise in their turn to new comma bacilli.

These discoveries of Koch have since been confirmed by all competent investigators, while the various alleged refutations of Koch's results have proved erroneous. It has been shown that in every case of genuine Asiatic cholera the comma bacilli are present in the intestine, and that they are never found under any other circumstances. Even the last postulate which was needed to show their pathogenic significance has been fulfilled. Rietsch and Nicati, followed by Koch himself, have succeeded in producing cholera in a guinea-pig by introducing into its duodenum pure comma bacilli.

Investigation as to the origin of cholera must, therefore, now meet this culminating question: Under what circumstances and through what channel do the comma bacilli penetrate into the human system, and in what manner do they there excite the characteristic processes of the disease? There can be no doubt that among us Europeans, and probably everywhere except in India, the cholera is invariably imported. It is equally certain that the dejections of cholera patients, which are rich in comma bacilli, are the chief if not the only agent by which the disease is spread. The bacilli which escape into the outer world with the stools find abundant means to prolong their existence. They continue their growth upon moistened bed-clothes, or in water which contains a sufficient amount of organic substances, or in food, such as fruit or milk, or in moist earth; and the ways by which they can in turn enter the system of a healthy human being are infinite in number. It is easy to understand why certain persons—e. g., laundresses and nurses—are more liable to infection than others; and it is equally intelligible that the spread of the disease should often bear a relation to certain outward circumstances. The fact has long been a familar one, that the cholera almost always progresses along the world's most frequented highways, and that it never travels faster than the means of human intercommunication render possible. This is important, because it shows plainly that the germs of the disease are not disseminated by currents of air. It is easy to understand that the distribution of the disease should sometimes correspond with that of water destined for personal use. Apparently in every case the disease-producing poison enters the intestinal canal, for the comma bacilli are found exclusively in the intestine, never in the other internal viscera; and this is true not only in the early but also in the later stages of the disease. We must therefore suppose that the bacilli are swallowed, and, if not destroyed by the gastric juice, develop their pathogenic functions in the intestine. In apparent agreement with this is the frequently observed fact that gastric catarrh, however acquired, existing at the time of an epidemic, always predisposes to the disease.

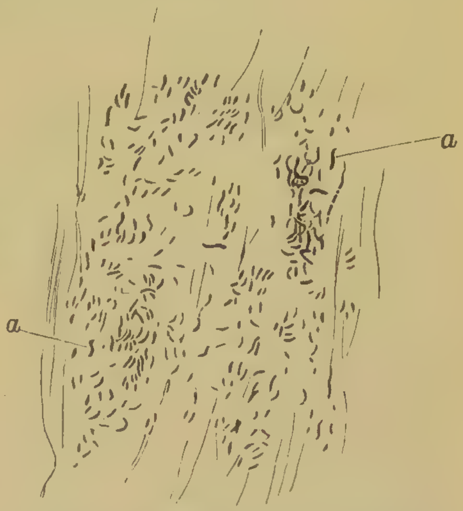

FIG. 9.—(From KOCH.) Comma bacilli from a cholera dejection which had lain for two days on a wet cloth. The S-shaped bacilli are at *a*. 600 diameters.

The views thus far expressed are opposed by Pettenkofer. He ascribes to the condition of the soil, varying with time and place, the chief *rôle* in the spread of cholera. He doubts whether the poison as contained in the stools is as yet efficient. It must, he thinks, undergo further development in an appropriate soil before it can acquire fresh pathogenic potency. His main argument is drawn from the fact that certain places, particularly those on rocky soil, enjoy immunity; the rarity of attacks upon shipboard is analogous. He points out that in cholera as well as in typhoid (*q. v.*) there is an evident harmony between the frequency of the disease and the varying stand of the water which underlies the surface of the ground. Further investigations, which will now for the first time have a firm foundation, afforded by the discovery of the comma bacillus, must decide how much influence the condition of the soil does exert upon the dissemination of the pathogenic poison. As it is, we feel certain that to give exclusive prominence to the condition of the soil, and to deny the possibility of infection in any other way, is to put a violent interpretation upon the observed facts; while the correctness of the above-stated views of Koch is becoming more and more probable. With regard to the constant presence of other micro-organisms than the comma bacilli in the intestine and the internal viscera of the victims of cholera, as claimed by Emmerich and others, we must regard them as associated appearances which have nothing to do with the true choleraic process.

Most cholera epidemics happen in the months of summer. Liability to the disease is very wide-spread, although some remarkable exceptions are seen. Sex is unimportant. Age has more influence. The disease occurs in sucklings, but, as a rule, is more rare among children than among adults. Elderly people are very apt to take the disease, while of typhoid fever the opposite is true. Most authors lay great stress upon predisposing causes. Among these, taking cold is not so important as are errors in diet and mild attacks of gastro-intestinal catarrh, which are shown by numerous observations to predispose strongly to the disease (*vide supra*). The stage of incubation seldom lasts over one to three days.

Clinical History.—As is the case in most acute infectious diseases, the intensity of the illness varies between the extremes of mildness and severity, so that usually a correct interpretation of the mildest cases is rendered possible only by the fact that an epidemic exists. These insignificant cases are called simple choleraic diarrhœa. The symptoms are those of a violent acute intestinal catarrh; the dejections are watery, rather large, painless, and number about three to eight in twenty-four hours. There is considerable malaise, complete anorexia, and thirst, and there may already be indications of severer choleraic symptoms—vomiting, slight pains in the calves of the legs, and diminished secretion of urine. Many cases recover after a few days or a week, but in others the first mild diarrhœa is succeeded, at the end of about one to three days, or rarely later still, by a severe attack of cholera. In such cases we speak of a "premonitory diarrhœa of cholera."

The mild form is succeeded in a gradual transition by the cases designated as "cholerine." Cholerine exhibits the symptoms of a violent, rather sudden cholera morbus. It often begins at night. To the diarrhœa, which now and then displays even at this time the characteristics of pronounced cholera, vomiting is soon added. The accompanying constitutional symptoms are rather severe. There is great languor and depression. The voice grows weak, the extremities are cool, the pulse is small and accelerated, painful cramps occur in the calves of the legs, the urine grows scanty and perhaps albuminous. The whole attack lasts about a week or two, till recovery is complete. The course of the disease is not infrequently varied by repeated improvements and relapses.

From these cases of medium severity there is again a continuous line of transition to the pronounced severe form of cholera proper. Statistics as to the frequency of the separate forms can not be given, since many of the milder cases escape observation.

The true attack of cholera may begin suddenly with the severest symptoms. As a rule, however, it is preceded, as already stated, by a first stage of brief premonitory diarrhœa. This, after one to three days, is replaced with equal suddenness by the severe symptoms of the second or "algid stage," or "cholera asphyxia." Its first symptoms are the abrupt appearance of great bodily weakness, chilliness, and vertigo. The characteristic gastro-intestinal symptoms soon declare themselves.

The diarrhœa grows very violent. At short intervals there are copious painless dejections, which at first retain somewhat of a feculent character, but very soon present a characteristic resemblance to "rice-water" or "whey." A single stool will measure a little less than half a pint (grm. 200). The stools have no color and almost no odor. They are watery, and usually deposit a finely granular, grayish-white sediment upon standing. Their reaction is neutral or alkaline. Only one or two per cent. is solid matter, with a little albumen and a relatively large amount of sodic chloride. In many severe cases the dejections contain more or less blood. The microscope reveals epithelium, triple phosphate, and numerous micro-organisms. Of these last a part are the comma bacilli, and a part are bacteria of putrefaction, etc. If the comma bacilli be demonstrated, of course the diagnosis is absolute. To accomplish this we take a coagulum of mucus from the stool, and, spreading it upon a cover-glass in as thin a layer as possible, carefully warm the glass by passing it repeatedly through a flame, in order to dry the mucus and fix it in its place. The preparation is then stained with an aqueous solution of methyl blue. If the bacilli be very abundant, the microscopic examination suffices for their demonstration, although complete certainty about their identity depends upon their behavior in pure culture-preparations. These must therefore be instituted in all doubtful cases; but it would lead us too far if we entered upon the particulars relating to such cultures.

These excessive evacuations are but very rarely absent or nearly absent. They are more apt to fail if death occurs at the end of a few hours—*cholera sicca*.

[In cholera sicca the intestines after death contain the characteristic rice-water material which, perhaps owing to paralysis of the muscular coat, was not expelled during life.]

The appearance of the diarrhœa is soon followed by frequent though rarely distressing vomiting. The vomitus consists in part of ingested liquids and in part of an actual transudation through the mucous membrane of the stomach and intestine. Hiccoughs may accompany and follow the emesis.

In addition to these prominent digestive symptoms of vomiting and profuse diarrhœa there are complete anorexia and excessive thirst. The tongue has a thick, dry coat. The abdomen is usually flat and soft, or it may be concave and hard. Sometimes we may feel fluctuation in the intestines, due to their being filled with fluid. There is not much real abdominal pain; what there is, is described as a "feeling of heat and pressure" around the umbilicus.

At the same time very severe symptoms develop in other organs. The circulatory system is chiefly affected.

The action of the heart may be stimulated at the beginning of the attack. The patient complains of palpitation and great precordial anxiety. After a brief time, however, cardiac weakness appears, and continually increases. The action of the heart becomes very weak, and the heart-sounds feebler and feebler. The pulse at the wrist grows very small, and is usually somewhat accelerated. In a severe case the pulse vanishes completely after a few hours.

This collapse of circulation makes itself quickly evident in the appearance of the patient. The face and extremities grow cool, and then ice-cold; the complexion becomes partly livid and partly a bluish gray; the lips are almost black. The surface temperature may fall below 95° (35° C.), while in the rectum febrile temperatures may often be observed, reaching 102° (39° C.) and higher. The eye and cheek grow very hollow, the skin becomes wrinkled, and loses all its elasticity. The voice grows hoarse and feeble (voice of cholera). Respiration is laborious and superficial. The mind may remain unclouded to the end, but usually there is great apathy, and all acuteness of perception is impaired. But few patients are restless and excited. Reflex action is much impaired.

One characteristic symptom is the cramps in the muscles. These are usually very painful, and consist in tonic contractions of the muscles, particularly those of the calf of the leg, but also those of the toes, thighs, arms, and hands. The cramps occur spontaneously or upon the least provocation, last a few minutes, and recur at short intervals. The precise reason of their occurrence is not yet known. It may be the effect of poison (*vide infra*). They can be observed in other severe acute diseases, although most marked in cholera. They sometimes occur in cholera morbus.

In a well-developed attack of cholera there is almost invariably oliguria or anuria. The urine, if any be secreted, is concentrated, with abundant sediment, and very often contains albumen. In many cases not one drop of urine reaches the bladder for days, and this condition persists till death or recovery.

The symptoms thus far depicted, if taken as a whole, represent the algid stage, which seldom lasts more than one or two days. In many cases death occurs during this period. It is ushered in by the tokens of extreme general prostration, and may take place after a few hours, or more frequently in the second half of the first day. But in other cases the "stage of reaction" succeeds. This may be a true compensatory period, leading directly to convalescence. The evacuations become less frequent and more feculent, and the vomiting ceases. The pulse becomes stronger, the cyanosis and coolness of the extremities diminish, and an abundant perspiration is not infrequent. After a few days urine is again excreted, which is almost invariably quite albuminous, and usually contains casts and red blood-globules. If convalescence be uninterrupted, however, the urine very soon becomes perfectly normal, and after a week or two the patient is to be regarded as completely recovered.

Departures from this favorable course of the stage of reaction are frequent. Recovery may be interrupted by repeated relapses into the previous condition, and sometimes with a fatal result. Or, instead of convalescence, there is developed a severe third stage, usually with fever. This stage ordinarily bears the generic name of cholera typhoid, although it is subject to manifold variations in its clinical symptoms as well as its exciting causes.

Cholera typhoid may present an actually typhoidal general condition with severe fever. There is a considerable elevation of temperature, headache, and dullness. The pulse is full and rapid, the face flushed. The skin, particularly that of the extremities, sometimes presents the so-called choleraic eruption, in the form of an erythema, roseola, urticaria, or the like. This variety of cholera typhoid ends after a few days in recovery, or else passes into one of the following conditions.

A second form of cholera typhoid is distinguished by the development of the most diverse local inflammations. Thus, there may be a severe dysenteric or diphtheritic inflammation of the small and large intestine, attended by offensive purulent and bloody stools. Pneumonia is also possible, as well as purulent bronchitis, diphtheritic inflammation of the larynx, pharynx, bladder, and female genitals, parotitis, and sometimes erysipelas and pyæmia. And when we consider that, besides all these conditions, the usual intestinal symptoms, or those of choleraic nephritis, may exist also, it is evident how varied the clinical picture may be. The development of these local affections frequently lays the foundation for numerous sequelæ.

Choleraic nephritis gives rise to the third or uræmic variety of cholera typhoid. The secretion of urine is almost suspended. The little that is still passed contains numerous casts, albumen, and frequently renal epithelium and white and red blood-globules. Somewhere toward the end of the first week, or possibly earlier, there are grave nervous symptoms, to be regarded as uræmic: first there is headache and vomiting, then sopor and coma, or delirium and convulsions. Most of these cases are fatal.

Pathology.—We are now acquainted with the manifold symptoms and varieties of the disease. If we seek for the pathological changes which control the process, and endeavor to find some correspondence between them and the symptoms, we shall be disappointed. At least, in its early stages, cholera is merely a severe local disease of the intestine. We find the serous layer of the coils of the small intestine rose-red from congestion. The mucous membrane is in a state of catarrhal inflammation: it is swollen, reddened, and at first covered with a layer of tough, transparent mucus; but very soon an abundant transudation flows into the canal, so that the intestinal coils are filled with a large amount of clear fluid, looking like "rice-water" or "gruel," and so devoid of bile as to indicate the suspension of its secretion. The signs of inflammation of the mucous membrane now grow more pronounced. The solitary follicles and Peyer's patches become swollen, with edges of a vivid red, and frequently there are many small ecchymoses in the mucous membrane. The extensive desquamation of the epithelial lining of the intestine has also been regarded as important, because it was regarded as in part the cause of the copious transudation. Still it may be questioned whether the desquamation is not, at least to some extent, a post-mortem change. In yet later stages of the disease the intestinal trouble very frequently assumes a croupous-diphtheritic character. The surface is necrosed and ulcerated in many places, and the contents of the intestine are no longer colorless, but sanious and bloody, with a foul odor.

Otherwise most of the post-mortem lesions correspond to what was obvious at the bedside. The muscles exhibit an early and persistent rigor mortis, and frequently contract in such a way as to throw the corpse into some unusual posture. All the internal organs are remarkably dry, pale, and anæmic. The left ventricle is contracted. The blood lies mostly in the large veins, the right side of the heart, and the cerebral sinuses. It is thickened, is but little clotted, and is said to resemble the juice of bilberries or huckleberries. The spleen is not enlarged—an exception to the rule in infectious diseases. The kidneys present marked passive congestion, most pronounced in the cortex. The microscope reveals a greater or less degree of parenchymatous nephritis, with great destruction of the epithelium. If death takes place at a rather advanced stage of the disease, the tissues have lost their characteristic dryness, and the most diverse local lesions, including nephritis, may be found to have occasioned death.

If we search for the connection between the pathological changes just described and the cause of the disease, or again between these lesions and the clinical symptoms, the first point to guide us is that the comma bacilli are found only in the lumen of the intestine, and never in the blood or in other parts of the body. The intestinal symptoms are satisfactorily explained by this abnormal state of the intestine, but for all the other grave symptoms we have to seek some special cause. The desiccation which the body undergoes as a result of the excessive liquid dejections can not fail to affect the tissues, but can not fully explain the symptoms, for at least the circulatory disturbances and the cardiac failure may develop before large evacuations have occurred. It has also been settled beyond question by means of the newer investigations that precisely the worst symptoms of cholera, namely, the muscular cramps, the subnormal temperature, and the changes in the blood, are occasioned by the chemical results of tissue metamorphosis in the comma bacilli, that is, by the so-called toxines. Many of these have been already isolated chemically by Brieger. In this connection the fact discovered by Hueppe is of the greatest interest, that the lack of oxygen which the bacilli in the intestinal canal must experience favors the abundant production of toxines.

As to the complications which occur in the later stages of the disease and which are embraced under the generic name of cholera typhoid, we regard them as mainly secondary. The choleraic process itself does not cause them, but is merely the occasion for their appearance. The examination of the intestine in such cases shows that numerous other varieties of bacteria follow closely upon the comma bacillus, gaining entrance to the system by treading in its footsteps.

The **diagnosis** of a pronounced case of cholera has no difficulties at the time of an epidemic. We must always be somewhat cautious about sporadic cases, for violent intestinal disturbance, simulating perfectly the milder forms of cholera, may be excited by other causes. In this connection we should mention the *cholera morbus* common among us; and poisoning, particularly acute arsenical poisoning, may give rise to symptoms wonderfully like cholera. But now that Koch's discovery has been made, the diagnosis of all such doubtful cases becomes perfectly certain if we can demonstrate the presence of comma bacilli in the stools (*vide supra*). We have no doubt that this demonstration will also lead us to a decisive conclusion as to the ætiological importance of mild choleraic attacks.

The **prognosis** should always be guarded at the beginning, even if the symptoms be mild, for, as already mentioned, a simple diarrhœa may prove to be "premonitory" of a severe attack of cholera. During the real attack the prognosis grows graver in proportion as the case presents the characteristics of asphyxia and cyanosis. The mortality in many epidemics is frightful. All the inhabitants of a house or street may in a brief period be swept away. Minute statistics are difficult to give. If we count the typical cases alone, the mortality is not infrequently fifty to seventy per cent. In about two thirds of the fatal cases death occurs during the first days of the stage of asphyxia, and in about one third during the second period, known as "cholera typhoid." The influence of the diet and the hygienic surroundings of the patient before his illness is important. A greater proportion of children and old people perish than of the middle-aged.

Treatment.—The measures to be taken to prevent the spread of the disease, when it has once started in a place, we can not here discuss. We can merely say that the greatest care should be taken to have the ingesta pure, particularly the milk and water, that the further extension of the disease may be hindered simply by isolating the localities attacked as completely as possible, or at least regulating intercourse with them very strictly. We must try to prevent the communication of the disease by isolating individuals attacked, and by disinfecting the dejections with five-per-cent. carbolic solution, and likewise disinfecting everything that may have been contaminated by the excreta, such as linen and bed-clothes, for which dry heat is the agent. We must content ourselves with a brief mention of these facts. It is very important to disinfect the stools as promptly as possible, since Wood has discovered the remarkable fact that the cholera bacilli in the intestine, because of the lack of oxygen there, become much more sensitive to external influences although they produce more toxine, as stated above. Individual prophylaxis is of the greatest importance. It has been proved again and again that a mild intestinal catarrh will predispose to cholera, and will aggravate the attack if cholera does occur; so that the slightest gastric or intestinal disturbance at the time of an epidemic of cholera demands the greatest attention both as to diet and medicine. We may well quote from the last proclamation of the Prussian Department of Public Improvement (Cultusministerium), that, "by exercising and promoting cleanliness and moderation, each person will not only best protect himself, but also most efficiently support the efforts of the authorities in behalf of the common weal."

[The vital importance of the serious treatment of a beginning diarrhœa during a cholera epidemic can not be too strongly insisted on. Rest, simple diet, and a little medication will, in the vast majority of instances, entirely prevent serious consequences. The apparently trifling character of the symptoms is apt to lead people into a false security. Those who can leave an infected district should do so without delay.

With reference to the prevention of an epidemic, a pure water supply and strict cleanliness in its broad sense possess far more virtue than cordons of troops or measures of quarantine. It is more practicable to destroy the soil than to keep out the seed in these days of constant and rapid international communication. The systematic disinfection of all cholera discharges or articles soiled by them should be a matter of course.]

The drug chiefly used at the beginning of cholera is opium, which forms the chief constituent of the various "cholera drops." The best form is the tincture, in doses of ten to twenty drops, or gr. ½ to j (0·03–0·05 grm.) of powdered opium, repeated every two or three hours. A more complicated formula is: Tr. opii, 1 grm.; vin. ipecac., 3; tr. valerian. æth. [P. G.: valer., part 1; sps. ætheris, 5], 10; ol. menth. pip., gtt. v. M. S.: Twenty to thirty drops. Or we may give tinctura opii benzoica [an elixir of which two hundred parts contain one part of opium, four of benzoic acid, and two parts each of camphor and oil of anise].

The opium treatment approved itself in the last epidemic, although a few physicians regard it as irrational, and prefer to give at the beginning of the disease one or two good-sized doses of calomel (gr. v to viij, grm. 0·3 to 0·5). Cantani and other Italian physicians praise highly enemata of a solution of tannin (warm water, previously boiled, two quarts (2,000 grammes); tannin, gr. xlv–xc (3·0–6·0); acaciæ, ℥ ij (50·0); tinct. opii, gtt. 30–50) or some disinfectant. Hueppe advises large doses of salol internally.

When the attack is fully developed we usually continue the use of opium. The patient is wrapped up in warm blankets and subjected to friction; or warm oil may be rubbed into the skin. Hot tea may be given, or strong coffee, or broth, or mulled wine. Hot baths, to which mustard may be added, have proved beneficial in repeated instances. Vomiting is to be controlled by morphine or ice. The painful cramps in the calf of the leg require subcutaneous injections of morphine. The feebler the action of the heart becomes, the more energetic must be the stimulants employed. We may give champagne, or inject camphor or ether. The attempt has been made again and again to make good the loss of fluid by injecting a solution of common salt beneath the skin or into the veins. Samuel recommends for this purpose a solution containing six parts of sodic chloride and one part of sodic carbonate in one thousand parts of water, at a temperature of about 100° (38° C.). Finally, injections have been made into the peritoneal cavity of a 0·5 per cent. solution of sodic chloride, or in other cases of a 0·3 per cent. solution of sodic carbonate, and, it is claimed, with benefit.

Great caution must be exercised about the diet, not merely during the attack itself, but for a considerable time afterward. At first we can allow only thin porridge, milk broths, and possibly a soda biscuit. It is advisable to administer dilute hydrochloric acid with the food.

The treatment of cholera typhoid varies greatly, of course, according to the kind of attack. The separate affections should receive their customary treatment.

[In the first stage, absolute rest, opium, and lumps of ice by the mouth *ad libitum* are the chief measures on which reliance is to be placed. It should be remembered that the entire function of the intestinal tract is reversed; thus, instead of an absorbing, it has become an excreting surface.

In the stage of collapse the nervous system is more or less paralyzed, the blood is damaged by the loss of its watery constituents, and the circulation of that fluid is greatly impeded. The subcutaneous or gastric absorption of drugs is consequently delayed or suspended. The utility of any active internal treatment during this stage is very questionable. Certainly narcotism by opium is highly undesirable. Mild external stimulation and the tentative administration of ice and small quantities of champagne or food are, at all events, not likely to do harm. Nature sometimes reasserts herself when the conditions are seemingly desperate, and the third stage, or that of reaction, comes on. In this stage, careful nursing and a sensible symptomatic but in no way meddlesome treatment are most likely to be followed by good results.]

CHAPTER XIV.

MALARIAL DISEASES.

(*Intermittent Fever. Fever and Ague. Swamp Fever.*)

Ætiology and Pathological Anatomy.—Malarial poisoning is the best example of a purely "miasmatic" affection. The poison which produces the disease is without doubt localized in certain places, in which every human being is liable to become its victim. But if an infected person comes to a place free from malaria and not naturally favorable to its development, there is no danger that he will cause the disease in others. The disease is never caught through contact with the patient. It is not at all contagious; the malarial poison, after it has once penetrated into the body, has practically no opportunity to escape again in an efficient form from the diseased system into the outer world. But the blood of a patient injected into a healthy person may transfer the disease (Gerhardt and others).

If we except the polar zones, there are few regions where malaria is not endemic in certain parts, at least from time to time, if not constantly. There is, however, great variation in the virulence as well as in the number of cases. While the common forms of intermittent fever are very frequent in Germany, in numerous places, yet the grave forms of the disease are very rare. Other lands are notorious for the severe malarial diseases, e. g., Hungary, the lands lying on the lower Danube, the Roman Campagna, the Pontine marshes, Sicily, and numerous districts in other parts of the world, chiefly tropical. Numerous observations have only served to confirm the statement that the soil is the true home and cradle of the malarial poison, and that the virus, escaping thence into the lower strata of the atmosphere, may be taken into the system, probably during inspiration. Permanent dampness of the soil is essential to the development of the malarial poison. This explains why marshy districts are so often malarial. The ground must not be covered by a great amount of water, but must during the dry season lie exposed to the atmospheric air. The access of air to the moist soil seems to be a second essential condition for the development of the malarial germs. A third influential factor is the temperature of the air, as proven by the great prevalence of the disease in southern countries and in the summer season.

With regard to the nature of the malarial poison, the work of late, and in especial of Italian, investigators (Marchiafava and Celli, Golgi, and others), has led to interesting conclusions. According to these authorities, the generators of malarial disease, the malarial "plasmodia," belong to the lowest class of animal life, the protozoa, and to that subdivision known as sporozoa. The plasmodium enters the blood and penetrates the red blood-corpuscles, where it has a lively amœboid motion. It evidently lives upon the tissue of the red corpuscles. The hæmoglobin of the latter is thus changed into melanin, a substance which apparently contains no iron, and the black granules of which are usually seen in the body of the plasmodium. After the red globule occupied by the plasmodium is completely destroyed, the micro-organism becomes free in the blood-current. Particles of pigment collect in its center, while on its periphery new generations are formed by splitting and subdivision. These new bodies appear to be mainly present in the spleen, liver, and marrow during the afebrile interval; while at the time of the paroxysms of fever they enter the blood once more and penetrate into the red globules. It is a very interesting fact that the different forms of intermittent fever, and in especial the tertian and quartan varieties, probably owe their development to different kinds of plasmodia, in which the developmental phases occupy different lengths of time. We can therefore by means of inoculation transfer from one human being to another only that variety of intermittent which the first patient exhibits, whether tertian or quartan. The quotidian type is probably due to the development of two cases of tertain fever in the same individual, side by side; whereby each separate tertian owes its development to a special generation of the plasmodium. It is Golgi's view that the severe tertian forms of malaria are caused by a special variety of plasmodium, which is distinguished by the appearance of crescentic forms of development.

Although no one has yet been successful in establishing this view of the plasmodia by means of pure cultures and inoculation of the same, yet the relation of the plasmodium to malaria seems evident from the fact that in every patient with malaria the plasmodia can be easily demonstrated in the blood (even without any staining), while these same forms are never detected in the blood except in malaria. The way in which the plasmodia enter the human body is not yet known. It may be by inhalation or by the stings of insects.

The investigations with regard to the plasmodium of malaria have also explained the long-recognized fact that in chronic malaria large amounts of pigment collect in the internal viscera. These deposits are most abundant in the spleen, which in the chronic varieties of the disease develops into a firm, hard tumor; but they are also found in the bone-marrow, liver, brain, and kidneys, leading finally in the liver and the kidneys, in frequent instances, to processes of chronic degeneration and inflammation. It is especially noteworthy that in those patients who present the most marked cerebral disturbances (pernicious comatose fever, *vide infra*), the cerebral capillaries are found to be completely occluded with pigmented plasmodia.

[Periodical fever is very widely distributed in the United States, and in the southern portions occurs in severe though not in the severest forms. Some regions which were formerly free from it are no longer so, and, *vice versa*, some regions which were greatly subject to it are now exempt; these changes are closely connected with the clearing and upturning of virgin soil largely impregnated with decaying vegetable matter, and with the subsequent cultivation of the same for considerable periods of time. The poison does not extend far above the surface of the ground, as is shown by the relative safety of sleeping in the upper as compared with the lower story of a house; during the night the poison seems to exist in greater intensity than during the day. Attacks are more liable to occur during the spring and autumn than at other seasons.

The hopes which have been entertained in some quarters that malarial regions might be rendered healthy by large plantations of the Australian eucalyptus globulus, a rapidly growing tree which absorbs immense quantities of water, do not seem likely to be realized in the light of French experience in Africa, and in that of the Trappist monks in Italy.]

Klebs and Tommasi-Crudeli have made extensive investigations as to the nature of the malarial poison. We must regard it as organic. The authorities just named state that the true cause of malaria is a specific variety of bacillus. They found peculiar bacilli and their spores both in the earth of malarial regions and in the adjacent strata of the atmosphere; and, by infecting rabbits with these, they were able to induce attacks of fever, swelling of the spleen, and the characteristic formation of pigment matter (*vide infra*). Before this, bacilli and spores had been found in the blood and spleen of patients suffering from malaria. It can not yet be said just how much significance these discoveries have.

[To Laveran really belongs the credit for the discovery and accurate description of the plasmodium. His work in Algiers has been substantiated by observers in widely separated malarial regions, and, at the same time, no very important additions have been made to it.

It is difficult to overrate the diagnostic and therapeutic importance of this discovery. The ordinary intermittents are usually easy of diagnosis, but the remittent forms often simulate typhoid fever, while the pernicious form may resemble uræmia or intracranial disease. Especially in pernicious cases life may be saved by prompt and appropriate treatment, which must be based, of course, on accurate diagnosis. The following details may be of use to those who are not familiar with the method of blood examination. The finger tips should first be scrubbed with a nail-brush, and soap and water, and then washed in alcohol or ether, to obviate the danger of mistaking minute particles of dirt for the pigment-granules derived from the destruction of red blood-corpuscles. A portion of a drop of blood is then to be received on a scrupulously clean object- or cover-glass, and squeezed out between the two, so that a thin layer of blood with separation of the individual corpuscles is secured. The amœboid forms of the plasmodium retain their movements for a number of minutes in a warm room, much longer, of course, with a warm stage. The presence of pigment granules in the red corpuscles may be the readiest indication of the presence of the organism. The crescentic forms are found more especially in chronic cases which have been under treatment by quinine, as is stated by Osler. Many of the other forms can be found in the blood only during a paroxysm. The editor has seen them in abundance in a cachectic malarial patient from Panama, and failed to find them again after a single large dose of quinine, which terminated the chills.

In dried and stained specimens it is easier to detect the organisms, but amœboid movement is, of course, lost.]

Liability to the disease is very wide-spread. No race, no age, no sex, enjoys immunity. It is a noticeable fact that those who have had the disease once are all the more apt to have it again. Former patients, although they feel perfectly well in a non-malarious region, are very liable to fresh attacks, or at least much discomfort, as soon as they re-enter an infected district. The time of incubation does not seem to be constant. It is put at from six to twenty days, but may be shorter. We shall consider below chiefly the common forms of intermittent, such as appear among us in Germany, contenting ourselves with a very brief description of the severer forms.

VARIETIES OF MALARIAL DISEASE.

1. **Intermittent Fever.**—This is the simplest form, and has for its especial characteristic the relative brevity of the febrile attacks, which almost always exhibit a remarkably uniform type. A febrile attack of this kind is frequently the very first symptom of the disease. In other cases the paroxysm of fever is preceded by a prodromal stage lasting several days, during which the patient feels languid, has no real appetite, complains of headache and pain in the back of the neck and in the limbs, and often even thus early presents a slightly yellowish complexion and an enlarged spleen.

In the typical attack of intermittent fever there are three stages. The attack begins with a chill. There is pronounced malaise, attended by intense chilliness and more or less shivering. The skin is cool and pale, the face may be somewhat livid. The temperature of the interior of the body is elevated, and rapidly rises higher. In by far the greater number of cases the attack occurs in the morning, or at least before noon, and but seldom later in the day. This cold stage varies greatly in length, usually lasting an hour or two.

The chilliness is followed by the hot stage. The skin grows burning hot, the face flushes, the pulse, which was before small, becomes full, and the action of the heart is excited. At first the temperature continues to increase, and reaches in this stage its maximum for the attack. It is exceptional for it to remain under 104° (40° C.), and by no means rare for it to touch 106°, or even 107° (41°–41·5° C.). This stage almost always lasts longer than the preceding, generally about three to five hours. The temperature may begin to fall as early as the latter part of the hot stage, but may persist till the beginning of the third stage.

In this, sweating, stage the skin grows moist, and there is soon a profuse general perspiration. The patient begins to feel much better. In a few hours the temperature usually becomes normal, and, after lasting in all about eight to twelve hours, the attack is over. It may be shorter or rarely longer. Usually, however, the temperature keeps on sinking slowly, so as to be still subnormal even on the next morning, perhaps not above 97° (36° C.).

There are certain peculiarities in the temperature-curve which we have ourselves observed. The elevation of temperature is almost invariably more rapid than its decline. The rise is most rapid during the first hours of the cold stage, and slower during the first portion of the hot stage. The ascent is but very seldom interrupted. During the hot stage, when the fever is highest, in the neighborhood of 106° F. (41° C.), there are not infrequently two little summits to the fever-curve, if the temperature be taken at short intervals. But the temperature may for hours remain the same. The temperature generally begins to fall some little time before the perspiration is evident. The decline is slow. It may be perfectly continuous, or it may be interrupted by fresh elevations, which are sometimes slight and sometimes considerable. In many cases the descent is by steps, the temperature remaining the same for half an hour or an hour, and then abruptly falling a couple of degrees and remaining for a time at this new level.

The chief characteristic is not, however, the nature of the single attacks, but the peculiar manner of their repetition. If the case is not under treatment, the single attacks keep recurring for a time, either daily, as in the quotidian variety, or every second day. This latter type of tertian intermittent fever (cf. Figs. 10 and 11) is probably the most frequent. There may exceptionally be still longer afebrile intervals. Thus we have *quartans*, *quintans*, etc. If there are two attacks in one day, a rare event among us, we have a double quotidian. If there is a violent attack every second day, and on the intervening days there are milder

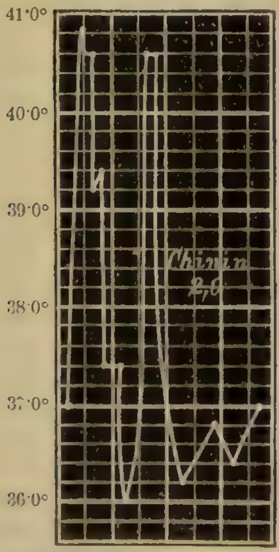

FIG. 10.—Quotidian intermittent fever.

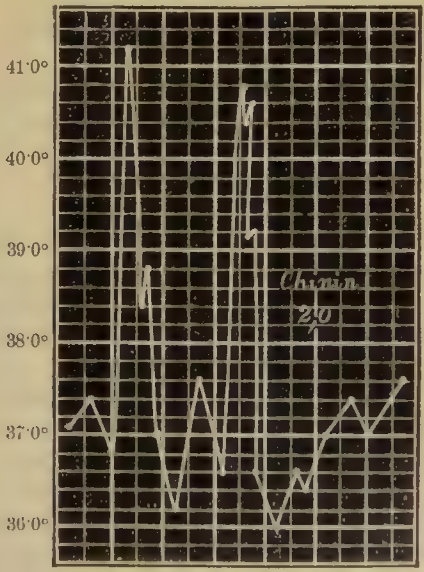

FIG. 11.—Tertian intermittent fever.

[Chinin 2·0 = Quinine, 30 grains.]

attacks, it is a case of double tertian. Very often the attacks do not recur at just the same time of day, but a few hours earlier each time. Less frequently they are later. This peculiarity is expressed by the term "anticipating" or "retarding," as the case may be—e. g., a retarding tertian ague. In cases of long standing, the paroxysms may finally lose all regularity, so that the fever is described as "erratic."

Next to the febrile attacks, the swelling of the spleen is the most constant and important symptom. It is usually considerable and capable of demonstration by percussion and palpation. At first the tumor increases with every fresh attack, and diminishes but little during the intervals. After the patient is freed from his attacks of fever the spleen may continue enlarged for some time. It is tender on pressure. The liver may likewise be swollen, but this is less constant and also less important.

Certain changes in the skin are very characteristic, chief among which is a peculiar yellowish-brown discoloration. This is due to an abnormal deposition of pigment in the skin. Herpes on the lips or nose is seen very frequently during the attacks. We have seen one case of herpes on the cornea. Mention has also been made of urticaria, purpura, and other eruptions.

Other internal organs than those already spoken of are rarely much disturbed. One symptom should be mentioned, which we have ourselves seen several times, viz., a quite marked acute dilatation of the heart during the attack. There were no bad results, and the normal condition was soon re-established. We may hear during the attack functional cardiac murmurs of a blowing character. Thoracic examination, particularly if made during the attack, may afford the signs of a dry bronchitis. Sometimes there is considerable diarrhœa, or other evidence of intestinal derangement. Catarrhal jaundice is confined to the severer cases. Sometimes the urine has a moderate amount of albumen. Genuine nephritis is met with only in the graver varieties of the disease. The increased excretion of urea on the days of the fever results, as in any fever, from the increased destruction of albumen. Severe pain in the cervical and upper dorsal vertebræ is regarded as characteristic of intermittent.

Besides the typical attacks, rudimentary and modified ones are not rare, in which the separate stages are ill defined, or in part wanting. We are most apt to see this in cases which have been already treated with quinine. Children do not have a true rigor. They merely become pale or livid. They may present marked nervous symptoms.

2. **Pernicious Intermittent Fever.**—This dangerous form occurs only in the true malarial districts, and is often preceded by a few attacks of a milder character. Then appear, in addition to the more or less perfectly marked stages of the febrile attack, other graver symptoms which not infrequently end in death. Severe nervous symptoms are most frequent. There may be unconsciousness, coma, delirium, or epileptiform or tetanic convulsions. None of these symptoms persist longer, as a rule, than does the common sort of an attack, and in a favorable case vanish completely when the sweating, which is usually profuse, begins. The great danger comes from the recurrence of the attacks. A second form of pernicious intermittent fever causes violent gastro-intestinal symptoms, which may almost exactly imitate the algid stage of cholera, with vomiting, diarrhœa, and collapse; or there may be severe cardialgia, dysentery, and the like. In the so-called pernicious intermittent with jaundice, intense jaundice appears during the attack, with vomiting and diarrhœa, and sometimes the gravest nervous symptoms. There are certain very peculiar forms, in which local diseases, such as pleurisy or pneumonia, can be demonstrated during every attack, but vanish wholly or in part when the temperature declines, only to appear again during the next attack.

[The pernicious form occurs in isolated cases wherever the ordinary variety of the disease prevails, but is much more common in the Southern and Western States, and there varies in frequency in different years. Periodicity in the attack is not always observed. The pernicious character is not always manifested in the first attack, one or more mild paroxysms being often precedent. In this country the algid form of pernicious periodic fever is often called "congestive chills," and this form is more common than the comatose or another form not mentioned by the author—the hæmorrhagic. In the latter the blood escapes from the kidneys, and less constantly from the mucous membrane. During the late civil war the mortality in the white soldiers of the United States army from pernicious malarial fever was 23·91 per cent.]

3. **Remittent and Continuous Forms of Malarial Fever.**—These are generally severe, and are seen, like the preceding, only in the worst haunts of malaria. The proof that they have the same ætiology as intermittent fever lies in the fact that they are sometimes developed out of the milder forms; but it is to be noticed that many types of disease which physicians in the tropics describe as malarial affections have not yet been proved to our satisfaction to have an actual identity of origin with the common intermittent fever. The symptoms of this variety are likewise those of a severe constitutional infection. Gastro-intestinal symptoms may predominate; or there may be such grave nervous symptoms as coma, delirium, and convulsions; or there may be jaundice, hæmaturia, and even a general hæmorrhagic diathesis; or various local disorders may exist, such as pneumonia, nephritis, and hepatic and splenic abscesses. The fever is high, but without any sort of regular intermissions, maintaining, for one or two weeks a remittent or a tolerably continuous type. Milder forms may end in recovery after eight to fourteen days, but often death ensues at this time, or even earlier.

[The remittent form apparently shows a greater intensity of the poison or a greater susceptibility of the individual. In the United States army, from 1861 to 1866, its mortality was twelve times as great as that from the intermittent form.]

4. **Chronic Malarial Cachexia.**—This occurs in the true malarial regions, and affects not only people who have had frequent attacks of pronounced intermittent or remittent fever, but also those who have never had acute attacks. The condition is chronic. It may exhibit a genuine intermittent character. The patient usually has a decidedly yellowish, malarial complexion, and almost always the spleen is evidently enlarged. There are no regular febrile attacks, but merely symptoms of general debility, anorexia, tendency to diarrhœa, or, more rarely, constipation, vertigo, wakefulness, frequent perspiration, pains in the muscles and joints, dyspnœa, and palpitation. There may be such nervous symptoms as trembling, paralysis, and mental disturbance; or we may see intestinal symptoms and jaundice. Dropsy occurs; also epistaxis, cutaneous ecchymoses, and other signs of a scorbutic condition. The spleen and liver gradually become greatly hypertrophied and melanotic. At the same time there may be an irregular fever, approaching either an intermittent or a remittent in type. Finally, secondary diseases are possible—e. g., tuberculosis, amyloid, or dysentery—and these may prove the immediate cause of death. The milder forms may be cured, but seldom unless the patient abandons for ever the malarial district.

5. **Masked Intermittent.**—This is the designation of cases where, although there is no fever, certain other disturbances arise in regular intermittent attacks. Chief among these is neuralgia. Its favorite seat is the supra-orbital branch of the trigeminus. It may occur in the other branches of the same nerve, or in the sciatic, the anterior crural, the nerves of the brachial plexus, and elsewhere. Cardialgia and enteralgia may occur in the same way. These attacks last from thirty minutes to several hours, and are frequently associated with all sorts of constitu-

tional symptoms, but, as we have said, are afebrile. There may be a splenic tumor, which aids diagnosis; but this sign may be wanting.

Numerous other intermittent disorders besides neuralgia have been described as masked intermittent. The list includes anæsthesia, convulsions, and paralysis; also intermittent hæmorrhage, œdema, cutaneous affections, and intestinal symptoms. We must add, however, that those who have described diseases of this sort, some of which seem strange enough, have not always been as critical as they ought, and have omitted to prove satisfactorily that such cases should be referred to malarial poisoning.

Diagnosis.—It is often very difficult to diagnosticate a case of intermittent fever at the first visit, particularly in a place where malarial poisoning is infrequent. The history of the case is by no means always enough to put one on the right track; and a single examination of the patient may prove equally negative in its practical results, whether it is made during the febrile stage or in the interval. Continued observation, however, will almost always disclose the regularity of the febrile attacks, the splenic tumor, the characteristic complexion, and the herpes; and our diagnosis becomes evident. Still it is not very exceptional for an intermitting fever to be taken at first for an intermittent malarial one, while eventually some quite different disease is found to produce the symptom. Pyæmia may give rise to mistakes of this kind; also purulent phlebitis, acute ulcerative endocarditis, and even tuberculosis. We should be very cautious in making a hasty diagnosis of "irregular intermittent fever." Our own experience has taught us that almost invariably the case turns out to be something else. Where there is doubt we may, in addition to a careful consideration of all the symptoms and a thorough physical examination, be aided by the therapeutic action of quinine (*vide infra*). If a high fever of intermitting type is affected by large doses of quinine but temporarily if at all, then a diagnosis of malarial intermittent fever is rendered doubtful.

Treatment.—Malarial poisoning is one of the few diseases upon which we can make a direct attack with assured success. In quinine we possess a remedy which probably acts upon the very cause of the disease, and the therapeutic efficiency of which is undisputed. Quinine is therefore the sovereign remedy in all forms of malarial poisoning, and is often the only drug employed. In the mild cases, which are the only kind that occur among us, we do not usually give the remedy upon the instant that the patient comes under treatment. It is best to wait for one or two attacks, partly to make sure of our diagnosis, and partly to learn what the type of the fever is, whether quotidion, tertian, anticipating, or recurring at the same hour. And in most cases this delay works no harm to the patient. During the attack itself there is seldom any use in special treatment. Of course, the patient must stay in bed and be kept warm during the cold stage, and have lighter coverings during the hot stage. During the afebrile interval the patient may be up if he feels strong enough and is careful. Quinine is given about five or six hours before the next attack is due. It is best to administer one large dose of twenty to thirty grains (1·5–2 grm.), either in solution or in capsules of seven grains (0·50 grm.) each. If the quinine be given in powder or capsules, it is a good way to follow it with a few drops of muriatic acid, to promote its solution in the stomach. Often one large dose prevents the next attack. In other cases it does occur, but with less subjective disturbance, no chill, and more moderate fever. We must then give another large dose before the second attack is expected. If the attack does not take place, then we may give for several days quinine to the amount of eight grains (0·5 grm.) *per diem*. After all, relapses are possible, even at the end of several weeks; but they yield readily to quinine.

Judging from our own experience, conchinine is the only one of the other preparations of cinchona which is as efficient as quinine. It costs only half as much, and is prescribed in just the same way. A disadvantage is that it is more apt than quinine to excite vomiting. All the other preparations of Peruvian bark, such as chinoidine and cinchonine, are much more uncertain in their action.

In the treatment of pernicious intermittent fever, of the masked forms, of the remittent and continued fevers, and of malarial cachexia, the chief remedy is likewise quinine, given in sufficient doses. Baccelli has shown that sometimes in pernicious fever the direct injection of quinine into a vein may save life. In all cases of considerable duration it is also of the greatest importance to remove the patient from the malarial region, if it can possibly be done. This often proves to be the only way to avoid relapses and attain a perfect cure.

In cases of longer standing, quinine sometimes loses its power. Then we resort to arsenic. It is frequently employed in malarial cachexia and in intermittent neuralgia, either alone or combined with iron. We give gtt. v to viij of Fowler's solution two or three times a day in water. It is still better to give pills of arsenious acid containing gr. $\frac{1}{30}$–$\frac{1}{20}$ (0·002–0·003) and gradually increasing to a daily dose of gr. $\frac{1}{6}$–$\frac{1}{5}$ (0·010–0·012). It may be added that arsenic is also said to have a prophylactic virtue: a long-continued use of it is stated to render a person proof against malarial poisoning. It may also be mentioned that a favorite remedy for chronic malarial cachexia among the Italian peasants is a decoction of lemons. It is even highly praised by local physicians. A finely sliced lemon is put with three glassfuls of water and boiled down to one glassful. We will not speak of the numerous other remedies recommended, such as eucalyptus, piperine, pilocarpine, berberine, and many others, for they can be entirely dispensed with.

The management of the severe varieties of malarial disease involves symptomatic treatment as well as the administration of quinine. We can not enter into the particulars. In combating the grave, nervous, intestinal, pulmonary, and renal symptoms, the dropsy and the anæmia, the physician must conform to the general rules of treatment.

[There is nothing to be gained by allowing a patient to have an unnecessary chill. If there is a reasonable probability that his paroxysms are due to malaria, a prompt effort should be made to cut them short. Four hours is the shortest time that it is safe to allow for quinine by the stomach with probability that the expected chill will be prevented. The drug acts much more promptly when given hypodermically. The hydrobromate is preferred by some to the sulphate for subcutaneous use on account of the necessity of using acid to dissolve the latter, and the consequent risk of abscess. Such a risk should have no weight if the physician has any suspicion that he has to deal with the pernicious form of the disease. If the stomach is irritable, the remedy can be given by enema. Quinine can also be given by suppository, though it may thus produce some irritability of the rectum. But the impossibility of disguising the bitter taste of the remedy or of making children swallow capsules renders this a valuable means of treatment sometimes in infants and young children.

Warburg's tincture is an antiperiodic which does good service in cases which do not yield to the ordinary methods of treatment.

The hypodermic injection of pilocarpine is reported to abort an impending chill.

Some prefer divided and smaller to the single and large dose of quinine or one of its substitutes, a difference of view which is of minor importance.

In the remittent forms boldness in the use of quinine is required. Cinchonism should be induced as promptly as may be, and maintained to a mild degree for several days; the quantity of the drug can then be gradually diminished.

The treatment of the pernicious forms of periodic fever presents itself under three main heads:

1. The prevention of pernicious paroxysms.
2. The treatment of the paroxysm when present.
3. The prevention of a recurrence.

1. We have seen that very frequently the pernicious character is manifested after the occurrence of one or more mild attacks; consequently, in localities and seasons marked by the occurrence of grave cases it is an imperative duty to treat every mild case promptly and energetically, a course which unquestionably saves many a life.

2. The management of the paroxysm differs according to the form which it assumes; in other words, is largely symptomatic. Bemiss (Pepper's "System of Medicine") says: "The cure of a congestive chill is one of the most difficult problems the physician can possibly encounter." Heat externally, opium and chloroform by the mouth, and morphine and atropine subcutaneously, nutrition by the stomach or rectum, according to circumstances, and alcoholics if the action of the heart is feeble, are the measures of widest application.

Whatever the type of the attack, a weak heart calls for alcoholic stimulation. Cinchonism is always to be induced as rapidly as possible.

In the comatose form it is to be remembered that the cerebral and other nervous symptoms are not due to congestion, but probably to a combination of the malarial and secondary blood-poisons. To quote Bemiss again: "Efforts to nourish the patient must never be relaxed. One must see many of these cases before he can realize how often they recover from conditions apparently hopeless when promptly treated and properly nourished."

The hæmorrhagic form, like the others, demands cinchonism and careful nutrition, but also hæmostatics. Purgative doses of calomel are indicated in some cases of each form, but should not be given in a routine manner.

3. Prompt cinchonism is the chief means of attaining the third aim of treatment. Removal to a healthy locality should be secured if possible, and the general condition of the patient requires careful attention.

It remains to add that those going to a malarial region can often avoid contracting the disease by taking advice of a local physician as to hygienic precautions, and by moderate divided doses of quinine.]

CHAPTER XV.

TYPHO-MALARIAL FEVER.

[THIS is not a distinct disease, but expresses a combination in the same individual at the same time of the effects of the special poison causative of each affection. Typhoid being a continued fever, its complication with malaria results in a pyrexia of a remittent type. Typho-malarial fever occurs in malarial regions, especially in the Southern States, and may be seen in non-malarial regions in the persons of those in whom malaria, contracted elsewhere, is in a more or less active state.

The characteristic symptoms of the two diseases are intermingled, those of typhoid, the graver disease, usually predominating. The history of the case and careful observation of the symptoms will generally clear up any doubts felt as to the diagnosis in the early stages. It would naturally be supposed that the combined affections must produce an illness more severe in character and more unfavorable as regards prognosis than belongs to simple typhoid. Such does not, however, seem to be the case. Woodward's statistics show that the mortality of uncomplicated typhoid was far greater among the white and colored troops alike during our late civil war than was the mortality of typho-malarial fever.

The treatment as regards the typhoid fever differs in no way from that suitable for cases of the ordinary affection; the periodic element demands the management appropriate to simple intermittent or remittent fever.]

CHAPTER XVI.

DENGUE.

[THIS affection has never appeared in Germany, and hence, doubtless, was omitted by the author. The name "dengue" is supposed to be a Spanish corruption of dandy, the term dandy fever having been applied to the disease by the West India negroes on account of the stiff carriage of those affected by it. Another name is "break-bone fever."

The disease generally appears in epidemics, and is almost exclusively confined to tropical and semi-tropical countries. In 1780 an epidemic supposed to be dengue prevailed in Philadelphia, and outbreaks have occured repeatedly in the Southern States during this century. In 1880 Charleston, Savannah, New Orleans, and other Southern cities were visited by it. There are those who maintain that dengue and epidemic influenza are identical, a view which the facts do not seem to the editor to bear out.

Ætiology.—As to the causation but little is known. Those who have had opportunities of studying the disease consider it both contagious and infectious, and the inference that it depends on a specific germ is readily suggested. It seems to prefer low lands along the sea-shore, and to be influenced by meteorological conditions in that it generally prevails during the summer and disappears as cold weather comes on. Neither age, sex, nor condition affords any protection from the disease; it was thought by Dickson that one attack generally confers immunity for life.

Pathology.—The disease is so rarely fatal that few opportunities have been afforded for its post-mortem study. So far as is known, it has no peculiar lesions. The prominence and the character of the muscle and joint pains have led some observers to think the affection related in some way to rheumatism.

Symptoms and Course.—The onset is usually sudden, but a pronounced chill is said never to occur. Prodromata similar to those of other infectious diseases are sometimes observed, but the first symptom is very often pain and stiffness in the muscles and joints, with frequent swelling of the latter. The large and small joints are equally affected, and the pain is increased by motion. With the pain there is a rise in temperature and in the frequency of the pulse. The pain is apt to increase during the first two or three days and disappear on the fifth, but irregular remissions are liable to occur. As the thermometer falls the pain and other symptoms diminish, but reappear in full force with any subsequent rise. During the later days of the disease a skin eruption appears on the upper part of the body, and in severe cases becomes general in about two days. This eruption is very variable in character; it may appear simply as an erythema, or simulate the eruptions of scarlet fever, rubeola, lichen, or urticaria; it is apt to be associated with well-marked heat and itching of the skin. In mild cases the eruption is evanescent or absent. Swelling of the lymphatic glands is not uncommon; in severe cases the mucous membrane of the mouth, throat, and nose is reddened, and hæmorrhage from the outlets of the body has been observed. Pregnant woman are apt to miscarry. Delirium is rare in adults, but common in children; the face is generally flushed, and the eyes are injected; the tongue becomes increasingly coated as the disease progresses, the appetite is lost, nausea is not uncommon, vomiting is rare. The bowels and the kidneys present no constant or distinctive symptoms.

In mild cases recovery is sometimes rapid; sometimes, and especially after severe cases, convalescence is very tedious, the muscular and articular pain and stiffness passing off gradually, and the glandular swelling lasting for weeks. Copious skin eruptions are followed by desquamation.

Diagnosis and Prognosis.—As to diagnosis there can seldom be any difficulty during the prevalence of an epidemic. The first cases are the only ones which are liable to be mistaken, and even their nature can not remain long in doubt. The prognosis is uniformly good.

Treatment.—We are acquainted with no agent capable of aborting or cutting short the disease; nor is there any known measure of prophylaxis except for an individual to keep away from those places in which the affection is known to prevail.

The treatment of the attack is simply symptomatic; notable pain calls for opium in some form. Quinine has not seemed to be of service. Debility following the attack demands suitable alimentation and tonics.]

CHAPTER XVII.

YELLOW FEVER.

[THIS disease is not a visitant of Germany, but its consideration can not be omitted from a text-book on the practice of medicine for use in America. In the following description the aim will be to bring out the more important features of the disease, while for fuller details the reader is referred to larger works and monographs.

Ætiology.—Yellow fever is an acute infectious disease, confined within certain geographical limits, and occurring chiefly in epidemics of greater or less extent. In certain localities, notably Havana and New Orleans, a season rarely passes without some sporadic cases. The influence of temperature is well established; the disease prevails, namely, during the summer or the warm season, and is abruptly arrested by one or two decided frosts; dampness is favorable to it. That it depends ultimately on a special cause and does not originate *de novo* are undisputed facts; but we still remain in ignorance as to the precise nature of this special cause. The poison appears to be more active at night than during the day, prefers low-lying districts, and in them hugs the ground to a certain extent. Bad sanitary conditions are most important accessory causes of the disease, furnishing the soil for the multiplication of the poison. There can be little doubt that, under the observance of strict personal and public cleanliness, yellow fever visitations might be made simply a matter of history. The transmission of the poison probably takes place solely through the atmospheric air, thus finding its way to the blood through the lungs; conclusive evidence is lacking that it gains entrance to the system through the alimentary canal. While the air is the medium of transmission, the distance to which the poison can be carried by the air alone is very short;

it can, however, be transported to an indefinite distance by fomites, especially if inclosed in trunks, packing-cases, letters, and the like. Its vitality may thus be maintained for very long periods. It is a remarkable fact that in large cities the infection may be of great virulence, but confined to a limited district or districts, by carefully shunning which unprotected persons are comparatively safe. An infected area is apt to extend itself, but the progress is slow, and is interrupted by streams of water, high walls, or simply streets.

That the disease is not, strictly speaking, contagious is the nearly unanimous opinion of those competent to form one. In other words, the poison is not thrown off in a matured state from the body of an individual suffering from the disease; but, after being so thrown off, remains in the atmosphere or lodges on solid bodies, and then undergoes changes which render it active for evil. One attack of the disease renders the system of that person insusceptible forever after; the natives of a yellow-fever district are far less liable to contract the disease than are those who move into the district from elsewhere; until these have passed through an attack they can not consider themselves as acclimated. The negro race is susceptible to the disease, though in a less degree than the whites, and in the colored the affection is far less fatal. Neither age nor sex has any special bearing on susceptibility. That fear, anxiety, worry, or anything which tends to depress the nervous system increases the individual liability is highly probable. The stage of incubation is very variable, ranging from one day to three weeks or even more.

Pathological Anatomy.—The disease involves no constant and peculiar lesions. In rapidly fatal cases, congestion and often hæmorrhage are found, especially in the nervous system, liver, kidneys, and digestive tract. In fatal cases of longer duration more or less parenchymatous degeneration is found. A fatty degeneration of the liver is quite common, and imparts a yellow coloration to the organ, giving rise to the terms *café au lait*, or box-wood liver. Jaundice of the skin and tissues generally is also observable after death, and depends upon causes in no way connected with mechanical obstruction to the flow of bile into the intestine during life. Splenic enlargement is conspicuous by its absence.

Course and Symptoms.—The onset of the disease is generally sudden, but may be preceded for a few days by malaise and other signs of general constitutional disturbance; the initial chill is seldom severe, reaction following soon and the thermometer rising to 102°-105°; hyperpyrexia is rare. The pulse-rate does not increase proportionately with the fever. The face becomes flushed and the eyes injected and watery; headache and pain in the back are early and usually very prominent symptoms. The bowels are confined; the tongue is apt to be clean if it was so before the disease came on; the stomach is usually somewhat irritable, and there may be vomiting; moderate epigastric tenderness is common; the mind remains clear, as a rule, but delirium is not very uncommon, and in children a convulsion may usher in the attack as in other acute infectious diseases; the condition of the urine is at first not remarkable, but albumen may soon appear. This hot or febrile stage may last from twelve hours to several days. The pulse genally declines in frequency before the disease has reached its maximum. As the fever disappears the other symptoms vanish also, and the second, or "stage of calm," begins. From this point recovery may be rapid and uninterrupted, the whole disease consisting of but a single febrile paroxysm of greater or less intensity and of short duration.

In grave cases, and gravity is often foreshadowed in the first stage by marked capillary congestion of the surface of the body irrespective of the intensity of the other symptoms, and after a stage of calm lasting for some hours, but rarely exceeding twenty-four, more distinctive symptoms appear. The pulse is very compressible, the surface of the body is cool, vomiting occurs or becomes more prominent, and hæmorrhage is now apt to take place. The escape of blood into the stomach, its retention and the changes which it there undergoes, and its subsequent expulsion, explain the dreaded and characteristic symptom known as "black vomit." Tarry stools sometimes are observed. Hæmorrhage elsewhere is also common, occurring from the gums, the nose, eyes, uterus, kidneys, into the skin, etc. Albuminuria with casts is very common. Jaundice, sometimes of a lemon-yellow hue, comes on, and is rarely lacking in severe cases. From this symptom the name of the disease is derived.

In cases marked by more or less complete suppression of urine, coma and convulsions, probably largely uræmic, come on. Some severe cases are of the "walking" type, the patients going about while the malady is far advanced, or even up to the time of death. As a rule, however, muscular prostration is marked.

If the disease does not prove fatal in this third stage, convalescence comes on more or less gradually, and is followed by complete recovery; relapses, however, occasionally occur.

The duration of the affection is variable but, on the whole, short, usually being less than a week.

Diagnosis.—In mild cases the symptoms are not distinctive, and the diagnosis at the commencement of an epidemic is not likely to be reached except by an experienced observer, and even by him more or less conjecturally. During an epidemic the severe lumbar pain, the headache, the suffusion of the eyes, and the moderate gastric irritability, are all-sufficient for diagnostic purposes. Severer cases are also marked by capillary congestion of the surface of the body, and the third stage with the black vomit, hæmorrhage, jaundice, slow pulse, scanty urine, and prostration is characteristic. Of course, all the above symptoms are not present in every case. The only disease which can well give rise to confusion is remittent fever with jaundice. This affection has a different temperature curve, is not confined to the yellow-fever zone, is controlled by quinine, and is not accompanied by black vomit.

Prognosis.—This varies in any given locality with the character of the prevalent epidemic. The death-rate is sometimes very high, sometimes moderate; it is greater in hospital than in private practice.

In the first stage of the disease the chief element in the formation of the prognosis seems to be the presence of marked and general capillary congestion of the skin, a symptom which foretells a severe attack. The absence of this symptom is rather less important than its presence. Cases may turn out to be severe in which it is lacking. The frequent deceptiveness of the stage of calm is to be remembered.

Yellowness, black vomit, and suppression of urine are symptoms denoting the greatest gravity, but do not justify the complete abandonment of hope.

Treatment.—There are no means in our power of aborting the disease. Prevention is to be attained by cleanliness in its large sense, and by careful quarantine against things rather than persons. Individuals from infected localities may safely be admitted into non-infected localities, provided that they and their clothing and effects are thoroughly disinfected. Merchandise, the mails, and the like, must be excluded or disinfected. So also vessels and all other means of communication.

The earlier proper treatment can be instituted, the better. Absolute rest and good ventilation are the first requisites. Emetics and cathartics are not indicated by the disease itself; the condition of the stomach and bowels should be inquired into, and indigestible food or an accumulation of fæces should be removed if present. A hot mustard foot-bath early in the attack is useful. For the lumbar pains, opium or morphine are indicated. Sinapisms, or other similar external counter-irritants, with ice internally, and hydrocyanic acid or chloroform, are serviceable against gastric irritability. High fever is to be combated by cold spongings, the wet pack, and the cold bath. For hæmorrhage, styptic remedies may be used, though it is doubtful whether, when given internally, they are really of much benefit. Of course, no medication is to be resorted to which is likely to heighten a tendency to emesis.

Suppression of urine is to be met by dry cups to the loins, diuretic remedies internally if the condition of the stomach allows, or the hot-air bath. The results of pilocarpine are disappointing according to Bemiss, who states that he has seen good effects from large enemata of water, preferably cold, if there be notable fever in these cases. Prostration is an indication for the use of alcoholic stimulants, among which iced dry champagne ranks high. It will be seen that the treatment is entirely symptomatic. The disease is self-limited, has a short course, and the patient will recover if he can be kept alive until the poison is exhausted. During the attack and until convalescence is thoroughly established the management of the diet is all-important. Small quantities of the most easily assimilable food may be given at short intervals if they are tolerated by the stomach; if not, alimentation must be by the rectum, and the lower bowel in this disease is generally in a fair condition for this method of nutrition.

Courage and hopefulness on the part of the physician may do much good, and the services of a skillful and experienced nurse are of the utmost importance. I am told that in New Orleans, and perhaps elsewhere, it is customary, for those who are not protected and can afford this course, to secure in advance a nurse as soon as an epidemic breaks out. They then take to their beds at the first sign of illness.]

CHAPTER XVIII.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

(*Spotted Fever. Cerebro-spinal Fever.*)

Ætiology.—The epidemic form of cerebro-spinal meningitis has been known only since the beginning of this century. The first epidemics were observed in southern France and in Geneva. Smaller ones occurred in Germany in 1822 and 1853; but it was not till 1863 that the disease became at all frequent among us. Since that date there have been more or less extensive epidemics almost every year. The southern and central portions of Germany are particularly liable to them. Sporadic cases may occur at any time.

Most of the epidemics appear in the winter and spring. We do not know any particular factors which promote the disease. It often seems to be decidedly endemic. Barracks, work-houses, and the like have been marked by tolerably extensive epidemics. Whether the disease can be carried by patients to places previously free from it is still uncertain. It is not directly contagious. Children and young adults are the most frequent victims; but now and then elderly persons are attacked. Sex can not be shown to have much influence.

That the disease is infectious is clearly shown not only by its epidemic and endemic character, but by its whole course. The peculiar pathogenic organisms and the manner of infection have not yet, however, been ascertained. It is an interesting fact that Fränkel's pneumonia bacilli have been recently several times detected in the purulent exudation upon the meninges, so that some investigators have suggested that croupous pneumonia and epidemic meningitis may be closely related from an ætiological standpoint, even if they are not actually two forms of a single disease. The author can not as yet wholly accept this view. It is indeed certain that the pneumonia bacilli may sometimes secondarily reach the meninges and here excite a purulent suppuration ("pneumatic meningitis," that is, metastatic meningitis complicating pneumonia); nor is it impossible that the pneumonia bacilli sometimes, and particularly at the time of an epidemic of pneumonia, do enter directly into the meninges and occasion a meningitis; but for the genuine epidemic meningitis we must seek some special pathogenic cause. Otherwise it would be impossible to explain why the same germs should sometimes find their way exclusively into the lungs and at other times and in other localities invariably into the cerebral meninges.

With regard to the gate of entrance of infection in primary epidemic meningitis, perhaps the specific poison may enter through the nostrils and the cribriform plate to the otherwise so well protected envelopes of the central nervous system.

[Sanitary conditions seem to play a less important *rôle* in this than in many other infectious diseases. During the epidemic which visited New England in 1873 the writer was interne at the Massachusetts General Hospital, and there saw a number of cases. The disease was also prevalent among horses at the same time, and it is curious to note that a like association of the affection in men and animals was observed in Vermont in 1811, and in New York city in 1871. During the year 1873, 216 deaths were returned as due to this malady in the city of Boston.]

Pathological Anatomy.—The autopsy discloses an acute purulent cerebro-spinal leptomeningitis. It is only in rapidly fatal cases that slight and incipient lesions have been met with. As a rule, the extent and intensity of the objective lesions correspond to the severity of the symptoms. In the brain the purulent inflammation attacks the convexity as well as the base. It is usually most marked along the larger blood-vessels and in the fissures of the cortex. Of the spinal cord the posterior surface suffers most. Frequently the lumbar portion is more affected than the parts above. It is, however, exceptional for the disease to be limited to the meninges ; it is prone to extend into the underlying parenchyma. The microscope reveals clumps of pus-corpuscles about the blood-vessels, where they penetrate into the tissues, and not infrequently there are numerous centers of genuine encephalitis. These latter may be visible to the naked eye. Exceptionally there may even be cerebral abscesses of considerable size. The vessels are distended with blood, clear into the central ganglia, and ecchymoses are frequent. The cerebral ventricles are usually enlarged, and filled with a cloudy serum, or even with pus. It is plain that these lesions of the cerebro-spinal parenchyma greatly modify the clinical picture, and that they must frequently have more to do with the severity of the symptoms than has the leptomeningitis itself.

Clinical History.—Prodromata are relatively rare, and if present they are not severe, being confined to general malaise, with slight headache, and pain in the limbs. Usually the disease begins rather suddenly; there is intense headache, often mainly felt in the back of the head, pain and stiffness in the back of the neck, and great general discomfort. It is not rare for vomiting to occur at first. Very often there are among the early symptoms such important mental disturbances as stupor or delirium. There is usually fever from the first. An initial rigor may occur, but it is not the rule.

The intensity of these first symptoms is uncertain. Subsequently to them the course of the disease may vary greatly. First there are very acute, violent forms, termed "explosive" (*meningitis cerebro-spinalis siderans*, *méningite foudroyante*), where the cerebral symptoms are very severe, and the patient survives only a few days or even hours. Again, there are abortive cases. These begin with equally threatening symptoms, but after a few days completely recover with remarkable rapidity. The majority of cases last about two to four weeks. In severe cases death may come as early as the first week. The disease is often protracted to six or eight weeks' duration, or even longer, and may end in death after all. Cases that last a good while sometimes exhibit a remarkably intermittent character. Finally, the number of mild cases is not small in which none of the symptoms are very pronounced, and recovery is relatively early.

The symptoms of the disease may be divided into (1) the severe general symptoms, referable to the brain and spinal cord; (2) the more localized, nervous symptoms; and (3) the results of the constitutional infection, including fever and diseases localized in other parts of the body.

1. Among the less definite cerebral symptoms headache is important. It is usually terribly severe. It is chiefly occipital, but sometimes is frontal or temporal. Like most of the symptoms of meningitis, the headache undergoes very frequent changes in intensity during the course of the disease. For a time it may remit, only to recur with fresh severity. Marked vertigo and a sense of fullness in the head may accompany it.

The pain in the head is re-enforced by intense pain in the nape of the neck and back, due to the spinal meningitis. There is almost always considerable tenderness along the whole spinal column. The erector spinæ is contracted, making the back straight and rigid, or even producing opisthotonos; and the head is bent backward by the reflex contraction of the muscles in the back of the neck.

In most of the severe cases intelligence is blunted; we find all degrees of disturbance, from slight drowsiness to delirium on the one hand, or deep coma on the other. These symptoms likewise may undergo frequent variation in their intensity. General convulsions occur in very severe cases alone, and are of evil omen.

The vomiting is also to be regarded as of cerebral origin. It frequently is an early symptom, but may be deferred.

2. Symptoms referable to the individual cerebral nerves are manifold and variable. The most frequent disturbances are in the nerves that supply the motores oculi. They include strabismus; nystagmus, or slow movements independent of volition; unilateral or bilateral ptosis; slow reaction of the pupils, or inequality of them, or myosis or mydriasis. In the area of distribution of the facial there is often a noticeable contraction of the muscles, giving the face a peculiar, painfully distorted look. Trismus, or tetanus of the masseters, is rare, and usually a bad sign.

Disturbance of the nerves of special sense is frequent. Deafness may be due to the stupor, but is often the result of an extension of the inflammation to the acoustic nerve. The purulent inflammation may be propagated as far as the labyrinth, or even into the middle ear. Tinnitus aurium is also frequent. Disturbances of vision are far less frequently observed, but optic neuritis has been repeatedly found by the ophthalmoscope. Severe purulent irido-choroiditis has been also observed. It is probably due to extension of the inflammation along the sheath of the optic nerve. Conjunctivitis and keratitis sometimes occur; but they are probably caused by external injuries rendered possible by the imperfect closure of the lids, or the diminished sensitiveness of the parts. We have several times found the sense of smell diminished.

Disturbances in the area of distribution of the spinal nerves are, on the whole, less frequent. The only one of value in diagnosis is the cutaneous hyperæsthesia. It is apt to be particularly severe in the extremities, and it may be so extreme that the light touch of a finger or a needle causes great pain. Sometimes there is a slight twitching in the muscles of the extremities. This has, however, no special significance. There is often rigidity and stiffness of the muscles. Kernig has called attention to the frequent appearance of flexure contractions in the legs, and sometimes also in the arms, if the patients are caused to sit up or if the thigh is passively bent upon the trunk. As might be expected, there is no invariable rule about the reflexes. The cutaneous reflexes are usually well marked, and the tendon reflexes may be; but in some cases we have found the tendon reflexes markedly diminished or even abolished. Such a condition is probably due to some lesion of the fibers of the posterior nerve-roots.

All of the nervous symptoms above enumerated result from one of two causes—either the roots of the nerves are affected by the purulent exudation, or the inflammation extends inward to the central organs themselves. This extension is the explanation also of other symptoms sometimes observed—viz., hemiplegia, paraplegia, partial convulsions, and aphasia.

3. In addition to all these nervous disturbances, we see also symptoms referable to other parts of the body. Of this class there is one cutaneous affection which is a very valuable aid to diagnosis. Herpes labialis or herpes facialis is apt to appear soon after the beginning of the meningitis. It is seen in more than half the cases, and as frequently in severe as in mild attacks. Other eruptions occur now and then—e. g., roseola, urticaria, or petechiæ. Sometimes they are so symmetrically distributed upon the two halves of the body as to suggest the idea of a nervous origin.

The digestive system seldom displays severe symptoms beyond the vomiting already mentioned. Anorexia and constipation are, indeed, usually present, as in many grave diseases. We have seen mild dysentery a few times. Now and then a slight jaundice has been noticed. The spleen is often somewhat swollen, but very rarely attains great size.

Swelling of the joints has been observed quite often; it is much more frequent in some epidemics than in others. The enlargement may be an early or a later symptom. It does not usually prove to be of grave omen.

The urinary apparatus is seldom affected. The urine may contain some albumen and a few casts. Polyuria is an interesting symptom, probably of nervous origin. It is more apt to occur in the latter part of the disease. In a number of cases sugar has been found in the urine. Cystitis is a secondary disorder which is not very rare, particularly in severe cases where the catheter has been used.

Pulmonary and bronchial symptoms are likewise secondary. They occur very often in bad cases. It is evident how easily the stupor of the patient may lead to the inhalation of solid matter, with consequent bronchitis and lobular pneumonia.

Lesions of the circulatory system are rare. Acute endocarditis has been observed only a few times. The pulse is usually somewhat accelerated, seldom rendered slower. Very frequently the pulse-rate is remarkably variable, undoubtedly because of variation in the supply of nervous force. Slight irregularities in the pulse are also common.

4. The fever in epidemic meningitis conforms to no single type. It does not correspond at all to the severity of the other symptoms; the worst cases may run their course with little or no fever. In most cases the fever has irregular remissions. It seldom exceeds 104° (40° C.). Sometimes the fever exhibits a decidedly intermittent character. It is in these cases particularly that we find the variation in the intensity of all the symptoms of which mention has been made repeatedly. The variations in the temperature do not, however, always run parallel with the changes in the other symptoms. In mild cases the fever is usually moderate and brief. The abortive attacks may present high temperatures at first, but these quickly abate. In case of a fatal issue there is sometimes hyperpyrexia before

death, reaching 108° to 109° (42°–43° C.). In the severer but not fatal cases the fever declines slowly but irregularly. The fever may be over, long before the other symptoms disappear.

It is impossible to portray all the forms, symptoms, and courses the disease may have. The chief forms have been already mentioned; but in reality these are only types which run into one another without sharply defined border-lines. It is in itself a characteristic of epidemic meningitis that most of the more tedious cases have a variable, uncertain course. We may even meet with a complete intermission of all the symptoms, lasting for quite a while, so that the return of the trouble may fairly be called a relapse.

Sequelæ are not rare after severe cases. Persistent deafness is the most frequent. It results from the complications, already mentioned, which affect the labyrinth and the middle ear. Little children may become deaf and dumb. Again, vision may be deranged, because of retinitis, atrophy of the optic nerve, or corneal opacities, etc. It is not very rare for meningitis to leave grave nervous disorders behind it. These are frequently the symptoms of a chronic hydrocephalus. We may observe headache, sudden unconsciousness, or even convulsions, mental impairment, and weakness of the extremities. Or there may be localized disturbances due to permanent injury of limited portions of the brain or spinal cord, such as hemiplegia, paraplegia, and aphasia. From many of these conditions there may be a gradual recovery, but others prove incurable.

The **diagnosis** of cerebro-spinal meningitis is not difficult in a well-developed case, particularly if the prevalence of an epidemic puts us in mind of the disease. Diagnosis is more difficult in sporadic cases, and most of all when the patient does not come under observation till he is very ill and when we can not obtain the previous history. Important factors are the abrupt onset, the speedy appearance of grave cerebral symptoms, the characteristic headache and pain in the back, the stiffness of the neck, and the herpes labialis.

If we find evident symptoms of meningitis, we have still to decide whether the case is one of primary epidemic disease, or secondary, perhaps due to extension from some other part. Bearing this last possibility in mind, we should examine the ears carefully; for, as is well known, chronic otitis media may set up a purulent meningitis. Again, it may be very difficult to exclude a tubercular meningitis. Here we should consider other conditions that might render tuberculosis probable, such as the general condition of the patient, heredity, previous pleurisy, the results of thoracic examination, or scrofulous disease of the bones or joints. The existence of herpes points toward epidemic meningitis, for it is exceptional in the other forms of the disease. It is sometimes difficult to distinguish between meningitis and severe cases of other acute infectious diseases—e. g., typhoid fever and septic diseases. Here we must weigh all the circumstances carefully.

This is a good opportunity to mention the secondary meningitis which is said to occur with relative frequency just at the time of an epidemic. The combination of croupous pneumonia (*q. v.*) with purulent meningitis has been repeatedly observed. Still, it is not easy to determine whether the cause of this secondary meningitis is actually identical with that of the epidemic form (*vide supra*). In other acute diseases, like typhoid and articular rheumatism, when they occur at the time of an epidemic, the "tendency to meningitis" is potent enough to make meningeal symptoms more frequent than usual. It has not been clearly demonstrated, however, that this fact is actually due to the epidemic meningitis.

The **prognosis** depends chiefly upon the severity of the cerebral symptoms. Yet we should be guarded in our utterances, even when the case seems mild, or has apparently made the first steps toward convalescence. The disease sometimes changes for the worse at a late period. In general the mortality is about thirty to forty per cent. Probably this estimate does not take into account many very mild cases.

Treatment is purely symptomatic. There is no specific for meningitis. A valuable remedy is cold applications. Ice-bags are placed upon the head, and, if possible, along the spine. There are long and narrow rubber ones for the latter purpose. These applications are borne well by most patients and afford decided relief. The local abstraction of blood has also an undeniably beneficial influence, however difficult this may be to explain. Leeches are put behind the ears, and cupping-glasses on the back of the neck and along the spine. Mercurial ointment is often rubbed in, not only locally but also in the same way as in treating syphilis. Its use is doubtful. The narcotics are of great value. The best is morphine given subcutaneously. It lessens the pain, and often affords the uneasy and delirious patient rest and sleep. Chloral and bromide of potash may also be employed. Iodide of potash is often given internally, to the amount of twenty to thirty grains (grm. 1·5–2) in a day. It is said to act as an "absorbent," particularly in tedious cases.

The fever hardly ever requires special treatment. If the fever intermits, still quinine exerts no permanent influence. Antipyrine is better borne, and it also sometimes relieves the nervous symptoms. Bathing involves manipulations which most patients find unpleasant and painful, so that baths can seldom be employed, at least in the more acute stages of the disease. Later, warm baths are often beneficial. Local complications—e. g., affecting the eye or the ear—require special treatment. The swelling of the joints which sometimes occurs we have thought to be somewhat relieved by salicylic acid.

CHAPTER XIX.

SEPTIC AND PYÆMIC DISEASES.

(*Spontaneous Septicopyæmia.*)

THE septic and pyæmic processes which follow serious injuries or operations belong to surgery; but analogous diseases occur in persons who are apparently in perfect condition. They take the form of an extremely severe acute infectious disease, usually fatal. There is often the greatest difficulty in diagnosticating these cases during life. Probably the most intelligible way in which to present these interesting and clinically important diseases will be to start with their pathology, and subsequently to speak of their ætiology and clinical history.

Pathological Anatomy and Ætiology.—The most striking features at the autopsy of such cases is that there is never found a lesion of one organ exclusively. Several, or it may be almost all of the organs, exhibit numerous limited foci of disease. The lesions sometimes consist for the most part of multiple abscesses, sometimes of numerous ecchymoses, and sometimes of combinations of the two. The abscesses are found chiefly in the lungs, kidneys, liver, spleen, muscles, heart, brain, and thyroid gland. Quite extensive purulent inflammation is also found. This attacks the joints by preference, but also the pleura and meninges and the eye, where it causes purulent choroiditis, panophthalmitis, and purulent degeneration of the vitreous. The ecchymoses are most frequent upon the surface of the body, the serous membranes (e. g., the pericardium and the pleura), the retina, and conjunctiva; and also in the brain and the pelvis of the kidney. Besides these multiple abscesses and ecchymoses, there is frequently another disorder, which seems to be the very focus of the disease, viz., acute ulcerative endocarditis (cf. the appropriate chapter). This usually attacks the mitral valve, more rarely the valves of the aorta, and quite exceptionally the valves of the right side of the heart. Finally come a number of changes common to all severe constitutional infectious diseases—acute splenic tumor, "cloudy swelling" of the liver and kidneys, a dryness and dark-red color of the muscles, etc.

A glance over this pathological picture makes us feel certain that some pernicious agency pervades the whole system. And this factor we can, in all cases, demonstrate beyond a doubt to be bacteria. In this way is established the etiological unity of many forms of disease which were once regarded as quite distinct because of their different localizations, for instance, endocarditis, osteomyelitis, and phlegmon. These micro-organisms are found not only in the exudations due to the endocarditis, but also in the midst of numerous small foci of inflammation situated in the internal organs, where they usually completely fill some little blood-vessel with what is called an embolus of micrococci. The large foci of inflammation visible to the naked eye are mostly purulent—i. e., are abscesses. Most of the internal viscera also contain very minute foci, devoid of nuclei, and in a state of "coagulation-necrosis." These are visible through the microscope alone. They may be combined with ecchymoses, and usually they are when seen already surrounded by a zone of secondary inflammation. This necrosis of tissue seems to be the first thing which the bacteria accomplish. The cutaneous, retinal, and other ecchymoses are frequently attended by the presence of bacteria; but this relation is not always observed. The relations here are perplexed, because we have to consider not merely the especial peculiarities of the different forms of bacteria (*vide infra*), but also the direct effect of the micro-organisms themselves in distinction from the chemical poisons ("toxines") generated by the same. The formation of pus itself seems to be invariably dependent upon the presence of bacteria, while on the other hand the hæmorrhages may apparently follow purely chemical intoxication. The prevailing custom is to term the cases of multiple abscess pyæmic, and those where there are merely ecchymoses and foci of inflammation, without actual suppuration, septic in the narrower sense of the term. But as the two forms are often combined, we also speak of a "septicopyæmia." From a strictly ætiological standpoint it is not probable that all the septicopyæmic diseases are to be regarded as identical. It is certain that in most cases of pyæmia the *Streptococcus pyogenes* may be regarded as the special cause of the disease. In less frequent instances it is the *Staphylococcus pyogenes aureus* which enters into the general circulation and produces pyæmic conditions, and it is probable that still other varieties of bacteria are sometimes concerned. Whether in human beings specific septicæmic bacteria are ever present is still unsettled. The general opinion is, as already mentioned, that septicæmia in the narrower acceptation of the word corresponds more to an intoxication of the body, whether due to the toxines of putrefaction or to those generated by the pyogenic bacteria. Brieger's admirable investigations have shown that the *Streptococcus pyogenes* produces large amounts of trimethylamin and the staphylococcus large amounts of ammonia.

Of course the bacteria, which are the real cause of the disease, must have penetrated into the body from the external world. In fact, careful search will reveal, in the great majority of cases, the place of infection. It follows that the idea of an actual "spontaneous" pyæmia, arising within the system, must be entirely abandoned.

The factors which most frequently excite septic or pyæmic infection are as follow: 1. The condition subsequent to labor or abortion, particularly the latter. The raw surface of the uterus furnishes ingress to the septic poison. Nor is it by any means the invariable rule that the uterus and its appendages should exhibit any considerable pathological change as a result of this absorption. We do find, often enough, diphtheritic and gangrenous inflammation at the place where the placenta was inserted, or purulent thrombi in the veins of the uterus and of the pelvis; but in other cases the uterus is merely a gate of entrance for the poison, remaining itself unharmed. 2. The septic poison may also be absorbed through slight abrasions of the skin, etc.; and these may be almost completely healed by the time the severe symptoms of disease are developed. Bed-sores belong in this category. 3. Ulcers of the mucous membranes may give rise to infection. This is the explanation of those cases of sepsis which complicate typhoid fever, dysentery, or diphtheria. 4. Lastly, we sometimes find no other source for the pyæmia than a suppurating disease of the bones, joints, or other parts, previously existing. The above enumeration by no means exhausts all the possibilities. Still, it will be found to explain most cases. The more minutely we search for a possible place of entrance for the septic virus, the less often we fail to find one. [The editor has seen two clear cases, one confirmed by autopsy, of general septic infection and malignant endocarditis as a sequel of gonorrhœa.]

When the poison has once made its way into the system, it can be disseminated through various channels. It may be carried by the lymphatics into the general circulation. A purulent phlebitis may be excited at the point of infection; and this in turn may excite, chiefly through embolism, secondary abscesses. These abscesses occur first in the lungs and then in other organs. It seems to be possible for a purulent phlebitis to arise in a vein remote from the place of infection. The valves of the heart often greatly promote the dissemination of the septic matter. The virus is prone to fasten upon them, probably purely from mechanical causes. This results in acute endocarditis. In such a case we must regard the endocarditis merely as one of the symptoms of the universal septic infection. But the valves are a fertile soil for the propagation of the poison, and emboli carry away from them a great deal of infectious matter to the various organs; and so the acute endocarditis becomes in many instances the central factor in the whole process. Yet in other cases there is little or no endocarditis.

Clinical History.—It is our intention to discuss below those cases chiefly which are of interest to the physician rather than the surgeon—i. e., where the septicopyæmia is an apparently primary, acute, and grave disease. Many of the essential traits of this type of disease are identical with those of the pyæmia which complicates the effects of serious wounds or the inflammation subsequent to childbirth; but it is precisely because no cause at all presents itself that many cases of the disease seem so obscure, and are so often wrongly diagnosticated. Besides, the patient may be very ill indeed before the physician sees him; and this adds greatly to the difficulties of a correct diagnosis.

The beginning of the disease is usually rather abrupt. An apparently healthy person is attacked with febrile symptoms, headache, and "rheumatic" pains in the muscles, joints, and loins. There may also be gastro-intestinal symptoms of considerable severity, including vomiting and diarrhœa. Usually the patient feels ill enough to take speedily to his bed. The symptoms now increase rapidly, and develop into a severe illness which may resemble either a bad case of typhoid fever or miliary tuberculosis. Or the cerebral symptoms, such as headache, stupor, and delirium, may become so prominent that the attack seems like meningitis. If the trouble in the joints (*vide infra*) predominates and there are signs of endocarditis, the disease may at first be taken for a violent attack of acute articular rheumatism.

Taking up the separate symptoms, we shall first name those which belong to every severe acute infectious disease and have nothing characteristic about them. In this list belong the general prostration, the anorexia, the mental disturbance, the stupor and delirium, the headache, the subjective symptoms of fever, the dryness of the tongue, and finally the acute splenic tumor which can often be made out. There are, however, other and more characteristic symptoms; and it is chiefly upon these that the diagnosis rests, provided we can make one at all. These are:

1. *The Course of the Fever.*—In many cases it must be confessed that this is not at all characteristic. It may even be so like that of typhoid fever as to lead to a wrong diagnosis. But in other cases the temperature-curve does aid us greatly, viz., when it represents an intermitting fever with marked elevations, reaching 106° (41° C.) and higher, and often accompanied by a chill, and with subsequent deep depressions. The curve may thus come to resemble closely that of a quotidian or even tertian intermittent fever. Sometimes, again, the course of the fever is made up of similar paroxysmal elevations, separated by periods of ordinary remitting fever.

2. *Cutaneous Symptoms.*—These are very frequent, and a great aid to diagnosis. The hæmorrhages into the skin are of chief importance. They may be either punctiform petechiæ or more extensive ecchymoses. If petechiæ, it may be very hard to distinguish between sepsis and the purpura of small-pox (*q. v.*). Of other cutaneous appearances, the first in relative frequency is an erythema resembling scarlatina. It is not improbable, as we have already said, that many cases which have been described as severe scarlet fever occurring during childbed were in reality septic disease. Roseola, wheals, pustulous eruptions, herpes, and phlegmonous inflammations have also been observed.

3. *Ocular Disturbances.*—The purulent inflammations of the eye, which are probably of embolic origin and which may develop into diffuse septic panophthalmitis, have been known for some time. Lately, Litten and others have called attention to more minute changes in the fundus of the eye. These are revealed through the ophthalmoscope, and have great diagnostic value. Chief among them is retinal hæmorrhage, which is sometimes accompanied by a white spot in the center, corresponding to a necrosis of the retina in that place; but there may be similar white spots without hæmorrhage.

4. *Circulatory Disturbances.*—An ability to recognize the cardiac lesions would be very desirable; but often this is impossible before death. The pulse is indeed frequently much accelerated and irregular; but such signs alone lead to no definite conclusion. Endocardial murmurs are often wanting, even in cases where the autopsy discloses abundant exudation and ulcers upon the valves. Still, in some cases of this sort we have found the heart-sounds noticeably deficient in clearness. Sometimes we hear blowing sounds, which might, however, quite naturally be regarded as functional. There are no noticeable changes in the blood. Bacteria have not yet been demonstrated in the blood of the patient during life. Sometimes a distinct, though moderate, increase in the number of white blood-corpuscles is observed.

5. The grave cerebral symptoms are for the most part quite analogous to those in other severe acute infectious diseases. They may be present, and yet no marked objective lesions may be found after death. In other cases they have an anatomical basis—in purulent meningitis, hæmorrhagic pachymeningitis, cerebral hæmorrhage, or abscess. These conditions, just enumerated, may excite localized cerebral symptoms, e. g., hemiplegia.

6. Affections of the joints are comparatively frequent, and of great value in diagnosis. We may find purulent inflammation, or even periarticular abscesses. If they appear early in the attack, they may, as we have said, lead to an erroneous diagnosis of acute articular rheumatism. Suppurative processes affecting the periosteum and the marrow of the bones not infrequently accompany the joint

affections. If there is decided suppuration in the bones we speak of an acute osteomyelitis, especially in the lower extremities. This condition is almost always occasioned by the staphylococcus aureus. In earlier times such cases were termed bone-typhoid. Finally, abscesses in the muscles are not uncommon.

7. Renal changes are frequent, but seldom produce striking clinical symptoms, or prove of value in diagnosis. The urine often contains a moderate amount of blood and albumen; but yet it may not be essentially altered in cases where the autopsy discloses extensive renal abscesses or ecchymoses, or hæmorrhages into the mucous membrane of the pelvis. In other cases, however, an acute septic nephritis is conjoined with the infarctions and abscesses, and then the urine exhibits all the characteristics of acute Bright's disease, having a large amount of albumen, red and white blood-corpuscles, epithelium, and casts.

8. The pulmonary symptoms are in part secondary. Bronchitis and lobular pneumonia develop as in all other severe constitutional diseases. The pulmonary abscesses of themselves give rise usually to no objective symptoms—or, at most, to a marked dyspnœa, out of all proportion to the scanty physical signs. Empyema is a not infrequent result of infection of the pleura, due to the foci of disease which are situated upon the outer surface of the lungs. If the aspirating-needle shows the actual existence of empyema, this fact may make the diagnosis of the constitutional disease much easier.

9. Of the abdominal symptoms we have already mentioned the acute splenic tumor. It is almost impossible to diagnosticate infarctions and abscesses in the spleen. If the spleen is enlarged and noticeably painful, we may suspect their existence. There are sometimes quite severe intestinal symptoms, such as a profuse "septic diarrhœa," in cases where the autopsy does not show any particularly grave lesions. Still intestinal ecchymoses and intestinal diphtheria have sometimes been observed. We should mention that often the skin has a faint jaundiced hue. This is sometimes the result of duodenal catarrh, but perhaps it is at other times hæmatogenous.

Course of the Disease and Prognosis.—The entire course of a septic case may be comprised within a few days, for a severe attack is always thus quickly terminated by death. Protracted cases are also seen, where the sufferings last one to two weeks, or even longer; but in these, again, the end is almost invariably unfavorable. It is not impossible that there are milder and curable forms. Our acquaintance with these last is, however, so slight that we can not state any particulars about them.

Diagnosis.—It is self-evident that a disease which combines symptoms so manifold and so ambiguous must be very difficult to recognize. We will recapitulate the chief diseases to be excluded. A case may greatly resemble typhoid fever when there is persistent prostration, diarrhœa, an eruption like roseola, and an enlarged spleen. In discriminating, we should consider with great care the possible ætiology—e. g., external injuries; and we should look for septic retinitis, swelling of the joints, cutaneous ecchymoses, and an intermitting form of fever. It is all the more possible for the disease to resemble meningitis, because, as we have said, meningeal disturbance may be one of the symptoms of the sepsis and color the whole picture. Here the symptoms of septic poisoning already mentioned would be of some value in diagnosis, and the physical signs of endocarditis or of a greatly enlarged spleen would be worth still more. There may be equal difficulty in the differential diagnosis between acute sepsis and acute miliary tuberculosis. Here we should consider carefully each separate symptom, and, above all, the ætiology, searching for something that would explain the occurrence of sepsis on the one hand, or of acute miliary tuberculosis (*q. v.*) on the other. If we found miliary tubercles in the choroid by means of the ophthalmoscope, or tubercle bacilli in the blood, all doubt would vanish. At the beginning of a septic attack the rigors may arouse suspicions of intermittent fever. Usually the early appearance of other symptoms corrects this idea; but, if not, the powerlessness of quinine will. If a severe acute nephritis has developed itself in a septic case, all the symptoms may be erroneously referred to uræmia. But persistent observation will usually lead us to the right conclusion. As to the conditions of great prostration resembling acute sepsis, which occur in acute (primary) ulcerative endocarditis and in severe articular rheumatism, see the appropriate chapters.

The **treatment** can be merely symptomatic. Of course we try again and again to cut short the attacks of fever by large doses of quinine or antipyrine, but never with any but temporary success. Baths, stimulants, and, if necessary, narcotics are the other chief remedies employed.

CHAPTER XX.

HYDROPHOBIA.

(*Rabies canina.*)

Ætiology. Rabies in Dogs.—A peculiar infectious disease sometimes occurs in dogs, and more rarely in some other animals—viz., the wolf, fox, cat, cow, and horse. Men who are bitten by the animal may catch the disease, and thus become the victims of terrible symptoms originating in the central nervous system.

Two forms of madness are distinguished in dogs—the raving madness and the quiet madness. Bollinger describes the raving form as beginning with prodromata, the melancholy stage, lasting one to three days. The animal is low-spirited, timorous, and without appetite. Then comes the stage of irritation or of mania, in which the animal is attacked with an impulse to bite. It seems determined to run away and rove about, and it utters a peculiar howl. The dog will not touch his ordinary food, but often swallows straw, hair, earth, bits of wood, etc. In the third or paralytic stage paralysis appears. The dog looks lean and wretched, and always dies on the tenth day at the latest. In what is called the quiet madness there is no maniacal stage. The symptoms of paralysis, affecting chiefly the hind limbs and the lower jaw, occur earlier and are sooner fatal. Marked pathological changes are not found. There are pulmonary and intestinal catarrh and passive congestion of the viscera, and the stomach often contains foreign bodies in place of the usual partially digested food.

[On the Western plains hydrophobia is said not infrequently to follow skunk bites. The bite is inflicted during sleep on persons passing the night in the open air or in tents to which the animal can gain access.]

Rabies is transferred to the human being almost invariably by the bite of some raving animal, and this animal is almost always a dog. The poison, which is not yet known in its pure form, is evidently contained in the saliva or slaver and in the blood of mad animals, and can, by means of these substances, be successfully inoculated upon other animals. Pasteur has discovered another way to produce the disease experimentally. He takes minute portions of the brain, medulla oblongata, or some other internal viscus of a mad dog, and either injects them into the veins of a healthy animal, or trephines, and then inserts them beneath the meninges. The virulence of the rabic poison when thus manipulated undergoes, under special conditions, very peculiar alterations, which will be detailed at the close of this chapter.

The liability to rabies does not seem to be universal among human beings. About one half of those who are bitten by mad animals exhibit no subsequent symptoms. Still this can be only in part due to inherent immunity from the disease, and must in part result from imperfect infection. The duration of incubation till rabies finally breaks out seems to vary greatly. As a rule it is about three to six months, but observers have reported instances both of shorter and of much longer duration.

Clinical History.—The disease begins with a general feeling of indisposition, anorexia, headache, and uneasiness. This last is partially explained, to be sure, by a dread of what is impending. If the bite was in the face, frequent convulsive sneezing may occur. Even now, in this prodromal stage, a marked aversion to liquids is a usual and early symptom. The attempt to swallow excites slight convulsive disturbances. Painful sensations may arise once more in the bitten place, although this has usually been cicatrized long before, and the neighboring lymph-glands are often found to be swollen.

Only a day or two later the second, hydrophobic stage begins. The especial characteristic of this consists in the peculiar attacks of tonic convulsions. The pharynx suffers most, but convulsions also seize the muscles of respiration and those of the trunk and extremities. A terrible feeling of anxiety and oppression accompanies these attacks, so that one who has once witnessed the sight can never forget it. The convulsions always seem to be reflex, and are produced by the slightest causes, particularly by any attempt to swallow, or sometimes by the very sight of water. They recur at gradually diminishing intervals, and last from a few minutes to half an hour. The excitement of the patient may reach the pitch of delirium or mania. The pulse is full and rapid. The temperature is usually only slightly elevated, but it may be high. There is great thirst, accompanied by burning pain in the throat. Often there is marked salivation.

This condition lasts one to three days. Then death occurs, ushered in by violent convulsions. Or death may be preceded by a brief third stage of paralysis, during which there are no convulsive attacks. Cases of recovery in man, if they ever happen, are extremely rare.

The result of the autopsy is practically negative. The disease is an infectious one, and therefore we should hardly think it *a priori* certain that such objective cerebral lesions would be found as might of themselves account for the grave clinical symptoms. The microscope has repeatedly detected very minute hæmorrhages, clusters of lymph-cells around the blood-vessels, etc. The throat may present the signs of catarrh. The lungs are congested, and often œdematous. The blood is dark, with few clots. The heart, liver, and spleen are apparently normal.

The **diagnosis** is usually easy, particularly if we know of the possibility of infection. We are guided by the convulsions following attempts to swallow, as well as by the whole group of symptoms. Hydrophobia is distinguished from traumatic tetanus by the absence of trismus and of the characteristic tension of the muscles of the back and abdomen, by the convulsions coming in separate attacks, and by the usually greater length of incubation. There is only one form of tetanus which bears very great resemblance to rabies, viz., the so-called hydrophobic tetanus (*vide infra*). It should be mentioned that the mere dread of hydrophobia may cause an easily excited person to have the nervous symptoms of the disease, but of course without disastrous results. Hysteria, also, may give rise to convulsions on swallowing somewhat resembling those of hydrophobia.

However hopeless **treatment** seems, we must at least try to mitigate the patient's suffering. Narcotics accomplish this best—e. g., opium or chloral, or, most useful of all, the inhalation of chloroform. Curare has been administered repeatedly, and does seem to lessen the violence of the attacks.

Prophylaxis is extremely important. We can not consider in detail the regulations (muzzling) which the government should make in order to prevent the spread of the disease. As to individual prophylaxis, every suspicious bite should be very thoroughly disinfected, and then cauterized either with nitrate of silver, caustic potash, or the red-hot iron. It has also been recommended that the entire wound or scar should be excised, along with any swollen lymphatic glands which may be found in the neighborhood. Internal remedies to prevent the outbreak of the disease are probably quite useless. Cantharides, belladonna, calomel, and arsenic have been given for this purpose, but without success. On the other hand, Pasteur has recently made a series of extremely remarkable observations which have led to a special method of prophylactic inoculation against rabies in human beings. If a bit of the spinal marrow taken from a mad dog is introduced beneath the dura mater of a rabbit by means of trephining, the animal exhibits the symptoms of rabies after fourteen days' incubation. If in the same way a second rabbit is inoculated from the first, and so on, the virulence of the inoculated material increases gradually with every inoculation, while the period of incubation grows shorter and shorter, till it lasts but seven days. Beyond this point the period of incubation does not seem to diminish. If, on the other hand, the same series of inoculations are made on apes, the virulence of the poisonous matter does not increase, but diminishes. And if dogs are inoculated with material artificially attenuated in this manner, the animals remain in good health, and furthermore attain an immunity against more virulent inoculations, so that they may be bitten by mad dogs without becoming infected.

Pasteur has also announced a still more simple and valuable method of artificial attenuation of the virus. He removes small portions of the spinal marrow of rabbits which are suffering from rabies in its most violent form produced by the above detailed method, and these bits of marrow he exposes to air which has been wholly deprived of moisture. In this way the poison contained in the spinal marrow gradually and progressively loses its virulence, until it finally becomes inert. A portion of spinal marrow which by long drying has completely lost its original virulence is then rubbed up in sterilized bouillon and injected into some animal—for instance, a dog—and then in regular succession pieces of marrow which have been dried for shorter and shorter periods and so contain more and more of the poison, until finally the point is reached when it is possible to use for the injection pieces which are perfectly fresh and extremely poisonous, without affecting the animal's health. That is, the animal has attained immunity from the disease. This second method has now been employed by Pasteur on several thousands of human beings who were said to have been bitten by mad dogs; and he claims that only a comparatively small number of the individuals inoculated by him have eventually suffered from rabies.

It is true that many doubts are still expressed with regard to the practical value of Pasteur's investigations. The above detailed experimental observations must, however, have some foundation in fact, although they should be tested carefully and repeatedly. If we compare Pasteur's statements with the facts known about prophylactic inoculation with anthrax (see the appropriate chapter) and about vaccination, we certainly can not restrain the thought that we are here upon the threshold of discoveries the future significance of which is immeasurable.

CHAPTER XXI.

GLANDERS.

(*Farcy.*)

Ætiology.—Glanders is a disease of the horse and some animals allied to it—viz., the ass and mule. It can, however, be transferred to man. It is characterized by peculiar new growths, either like nodes ("farcy-buds"), or more rarely diffuse. These are very prone to suppurate and break down. Such nodes, and the ulcers which they leave behind them, occur most frequently in the mucous membrane of the nose. In horses the purulent nasal discharge is one of the earliest and most important symptoms of the disease. Similar nodes are found in the larynx, lungs, liver, spleen, and kidneys, and often also in the skin. The cutaneous swellings and deep, crater-like ulcers belong to that form of the disease which is called "farcy." The corresponding lymphatic vessels and glands are usually much swollen. The animal has fever, grows weaker and weaker, and almost invariably dies at the end of one to three weeks.

Glanders in man is always referable to infection from a diseased animal, although in certain instances it is impossible to demonstrate the source. The disease is therefore commonest among persons who have much to do with horses—e. g., hostlers, coachmen, farmers, and cavalrymen. The virus is usually conveyed by the pus and nasal secretions of the diseased animals. A little of this falls upon some excoriation on the hand or some crack in the skin, and is absorbed. Man does not seem very liable to the disease; it is of rare occurrence.

Löffler and Schutz have discovered the specific disease-producing agent. These investigators were able to demonstrate in all the products of glanders delicate bacilli about the size of the bacilli of tuberculosis. These bacilli could be reared artificially, and, if inoculated upon horses and other animals, gave rise to a typical attack of glanders in every instance. The bacilli of glanders can scarcely ever be detected in the blood. It is also very interesting that they rapidly lose their virulence in purified cultures outside of the living body. This is one more proof of the fact, which is lately coming more and more into prominence, that the external influences surrounding the life of bacteria modify greatly their biological peculiarities.

Clinical History.—The period of incubation lasts about three to five days, and sometimes longer. The first symptoms are local, if the infection has resulted from a visible injury. There is considerable swelling and pain in this spot, and usually considerable lymphangitis in its neighborhood. In other cases, however, the disease begins with indefinite constitutional symptoms, such as fever, headache, and pain in the limbs, so that there may be some resemblance to a beginning typhoid fever. The local and general disturbances increase, and the disease soon attacks other parts of the body. There are usually pustules, or larger abscesses in the skin. These burst and discharge offensive pus, leaving behind them irregular, deep ulcers. Not infrequently the joints are swollen. The mucous membranes are also attacked; chief among these troubles are ulcers in the nose. The nose swells as if with erysipelas, and there is a purulent, foul-smelling discharge. The nose rarely escapes. The conjunctivæ, throat, mucous membrane of the mouth, and the larynx also undergo inflammation and ulceration. A violent, diffuse bronchitis develops. Sometimes there is considerable disturbance of the stomach and intestine, giving rise to vomiting and diarrhœa. At the same time the constitutional symptoms become more and more severe. The patient grows stupid or delirious. In some few cases the severe cerebral symptoms are due to a purulent meningitis, perhaps through extension of the inflammation by contiguity from the nose. The fever is high. Sometimes it is quite continuous. More rarely there are chills and great elevations, as in the fever of pyæmia. The pulse is rapid and small. The spleen is seldom much enlarged. The urine may contain a trace of albumen.

In these severe acute cases the termination is almost always fatal. Death occurs at the end of about two to four weeks. There are cases with a more chronic course, with tedious persistence of the troubles in the skin and mucous membranes, and milder febrile and constitutional symptoms. Such attacks appear at first tolerably favorable, but may later assume the acute form, or they may run on for months, and at last end in complete recovery.

The **autopsy** reveals a condition greatly resembling that in pyæmia. We find abscesses in many parts, particularly the muscles and the lungs, and, next in frequency to them, the spleen, brain, and other viscera. In the mucous membrane of the nasal cavities, the pharynx, and the larynx, are found nodes and ulcers, such as occur in the horse. As in septicæmia, there are often numerous hæmorrhages into the serous and mucous membranes. It has already been mentioned that the specific bacilli of glanders are present in the abnormal secretions.

Diagnosis.—Without the aid of ætiological factors the diagnosis of glanders is often very difficult. Indeed, until recently there have been instances where even the autopsy did not suffice to exclude pyæmia. But now that the specific bacilli have been discovered we can clear up all doubts. We can not, however, enter into a particular description of the distinguishing characteristics of these bacilli. Their demonstration requires pure cultures. At the bedside also ætiology is all-important in diagnosis—e. g., exposure to infection, or occupation. Experience with a limited number of cases renders it probable that in the future we shall be able to demonstrate the bacilli, during the life of the patient, in the nasal secretions or the contents of the abscesses. The most characteristic symptoms are the nasal and cutaneous. In a case that takes a chronic course there is a possibility of mistaking the cutaneous ulcers for syphilitic sores.

We have already implied that the **treatment** of acute cases is almost hopeless. We must do all we can in the way of cleanliness and disinfection to improve the local condition of the skin, the nose, and the throat. Appropriate agents are carbolic and salicylic acids and chlorine-water. Further treatment, by means of baths, quinine, and stimulants, should be in accordance with the general rules for the care of severe acute infectious diseases. Potassic iodide has been recommended as an internal remedy.

CHAPTER XXII.

MALIGNANT PUSTULE.

(*Anthrax. Charbon. Splenic Fever. Mycosis intestinalis. Carbunculus contagiosus.*)

Ætiology.—The cause of malignant pustule is the infection of the body with a specific kind of bacilli, the *bacillus anthracis*. This organism was discovered by Pollender in 1849, and a few years later, independently, by Brauell.

These bacilli are very minute cylinders, about seven to twelve micromillimetres in diameter. They are found in enormous numbers in the blood and organs of animals which die of anthrax. Aniline-staining makes them more easily visible. By means of blood containing the bacilli, Davaine (1863) and others have inoculated many animals with the disease, including mice, rats, guinea-pigs, cows, sheep, goats, and birds. The bacilli can also be isolated and cultivated, and then produce infection. This is proof positive that they are the actual carriers of contagion. The rapid increase of the anthrax bacilli in the blood goes on by subdivision. In the artificial cultivations, however, the bacilli grow, as Koch has shown, into quite long threads, in which shortly appear minute, brilliant egg-shaped bodies (cf. Figs. 12 A and 12 B). The threads become disintegrated, setting free the little shining ovoids, the spores of anthrax, to grow into bacilli. The bacilli can live only a relatively brief time; but the spores have unusual tenacity of existence. They may remain dried up for years, and then be brought to further development if placed in favorable conditions of heat and moisture. If the spores are transferred to animals, they develop into bacilli, and there is scarcely room to doubt that men and animals are quite as often infected by spores as by full-grown bacilli. There are facts which render it not improbable that the anthrax bacilli exist in other places than the bodies of men or animals, and may there complete their circle of development. Such places are marshes, the banks of streams, and the like. If it is possible for them to be carried by high water to the pasture lands, we have an explanation of those sudden endemic appearances of anthrax which sometimes occur in places previously free from the disease.

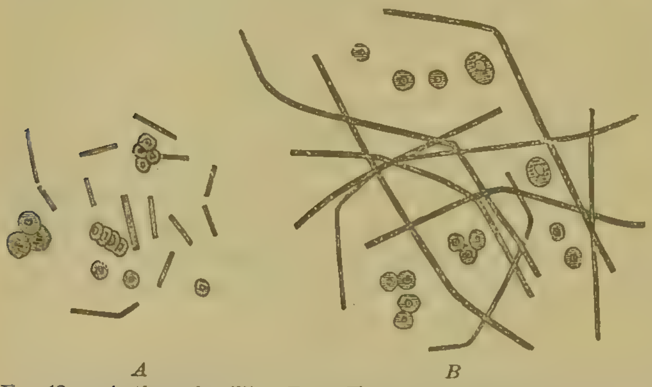

FIG. 12 A.—Anthrax bacilli. (From KOCH.) 650 diameters. *A*, from the blood of a guinea-pig. *B*, from the spleen of a mouse after three hours' culture in the aqueous humor.

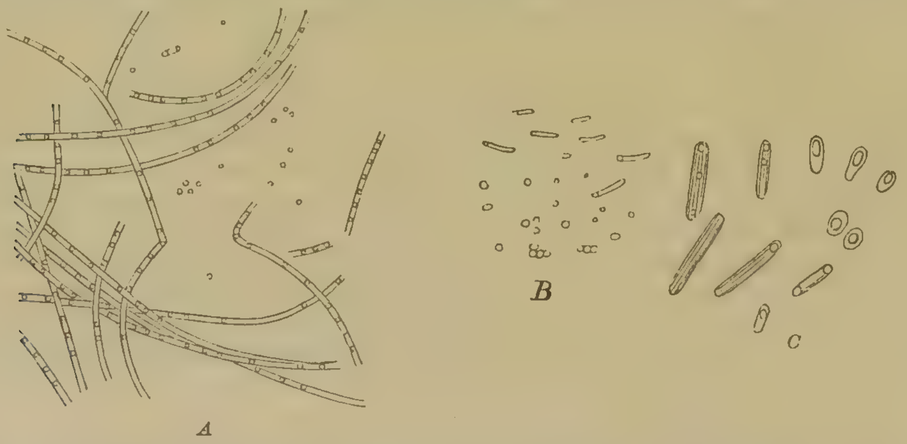

FIG. 12 B.—Anthrax bacilli; spore formation and spore germination. (From KOCH.) *A*, from the spleen of a mouse after twenty-four hours' culture in the aqueous humor, spores arranged like strings of beads in the filaments. 650 diameters. *B*, germination of the spores. 650 diameters. *C*, the same, with a higher power. 1650 diameters.

Anthrax in animals is of great practical importance, because its favorite victims are the herbivorous domestic animals—viz., the cow, sheep, and horse. Among these it is terribly destructive. It is remarkable that the carnivora enjoy almost complete immunity. The disease usually runs a very acute course in animals. Indeed, it often seems like apoplexy; the apparently healthy animal suddenly falls, suffers for a few minutes from convulsions and dyspnœa, and dies. Other cases have a somewhat longer and more intermittent course, but in these also recovery is very rare.

Probably human beings are infected in most cases by direct inoculation. Shepherds, farmers, butchers, and others who come in contact with animals suffering from anthrax, are liable to infection through any little wound or scratch upon the hands. Very often the disease is caught from hides, hair, or other parts of dead animals. In workshops and factories where wool and hides have been used which came from diseased animals, anthrax has repeatedly occurred. Curriers, rope-makers, paper-makers, and those who handle horse-hair and wool, are all exposed. Anthrax has also earned the name of "rag-pickers' disease." Another way of infection, supposed to happen among animals as well as men, is through the sting of insects—e. g., flies—bringing the poison from diseased animals. It is not likely that the virus can be absorbed through the unbroken skin, or by the lungs. It is certain, however, that the intestine may afford ingress to the infectious matter. Koch has proved this by putting spores in the food of sheep. Intestinal mycosis in man (*vide infra*) may very possibly be due to a similar mode of infection. Many cases of poisoning from eating meat have been referred to the ingestion of the flesh of animals who died from anthrax.

Clinical History.—Anthrax in man has two distinct forms. These may appear in combination. The first begins with a local disorder of the skin at the point of infection—viz., the malignant pustule, or anthrax carbuncle. The second and rarer form presents the symptoms of a severe acute constitutional infection. An accompanying cutaneous disorder is sometimes observed.

1. The **malignant pustule** usually comes on the hand, the arm, or the throat, and appears from three to seven days after infection. A vesicle forms at the infected spot, grows rapidly, becomes excoriated, and usually takes on a characteristic appearance, being of a dark-bluish or black color. The surrounding parts become diffusely swollen and red. Secondary vesicles may surround the original one. The swelling becomes more and more extensive. Inflamed lymph-vessels or veins radiate in red lines from the pustule, and the neighboring glands are also affected. These appearances are accompanied by fever, and more or less prostration. In a favorable case the swelling subsides, the scab falls off, and there is at last complete recovery. But in other cases the constitutional infection becomes more and more prominent, and eclipses the local disorder. The fever and prostration increase. Severe intestinal symptoms appear, or else stupor, delirium, and other nervous disturbances; and death may ensue after a few days' illness.

2. **Intestinal Mycosis.**—A better name would be intestinal anthrax. A quite different picture is presented by this second form, which gets its name from the marked intestinal lesions. In this the cutaneous disorder, if it exists at all, is insignificant, compared with the severe constitutional disturbance. It is only within a few years that the labors of Buhl, Waldeyer, E. Wagner, Leube, and others have shown that attacks of this kind have any connection with anthrax. So few cases have thus far been observed that it is impossible at present to give a definite and complete description of the disease.

In cases of this sort the attack is usually rather sudden, beginning with chilliness, vomiting, headache, and languor. The diagnosis is usually very obscure at first, unless the calling of the patient suggests the possibility of anthrax. On careful examination, we may find some places where the skin is broken, or possibly a small characteristic pustule. In a case which came under our own observation a pustule had existed on the back of the right hand for some weeks before severe symptoms appeared, but had not attracted the attention of the patient at all. In this case, therefore, the constitutional infection seems to have come from the local disease. But in other cases cutaneous troubles, in the form of small carbuncles may occur secondarily in the course of the disease. Hæmorrhages into the skin and mucous membranes also occur.

Of other symptoms, the gastro-intestinal deserve to be mentioned first. Vomiting occurs frequently, and also a moderate, painless, and sometimes bloody diarrhœa. There is usually severe dyspnœa, and a marked sense of oppression in the thorax, but without objective pulmonary signs. Very soon there is collapse; the nose and extremities grow cool; the pulse is rapid, but small; and there is lividity. In a few instances tetanic or epileptiform convulsions have been observed. The temperature is seldom much elevated. It may be subnormal. In a few days the prostration becomes complete, and death ensues.

Milder forms apparently occur, but here the diagnosis may not be absolutely certain. We have seen a few such cases originating in a rope-walk where Russian hair was used. The constitutional symptoms were only moderately severe, the fever was mild, and recovery occurred after about two or three weeks.

Pathology.—In the fatal cases of anthrax the intestinal lesions are the most characteristic. Besides the signs of catarrhal inflammation, we find peculiar lesions in the mucous membrane of the small intestine, and sometimes in the upper portion of the colon. These consist of dark, infiltrated spots, with hæmorrhages, the spots being somewhat larger than a silver dime. The microscope reveals numerous collections of anthrax bacilli, situated chiefly in the lumen of the blood-vessels. The spleen is usually only moderately enlarged, but dark and congested. There may be ecchymoses in the kidneys, the brain, and the serous membranes. Often there is swelling of the lymph-glands. In one case which we saw, with slight intestinal lesions, the mesenteric glands were considerably enlarged, and the bronchial lymph-glands were perfectly enormous. The bacilli are found in all the organs mentioned.

The **diagnosis** of a malignant pustule is seldom difficult, particularly if attention be directed to the ætiology. All doubt is over if we find the bacilli. The cases of intestinal mycosis may be more obscure. Here, too, the demonstration of bacilli in the blood is, of course, of the greatest importance, but reports have thus far been scanty of endeavors to find them during life in man.

Treatment.—1. Prophylactic inoculation. Toussaint and Pasteur were the first to show that the virulence of anthrax bacteria can be artificially diminished by certain external influences. If the bacilli are kept under cultivation for several weeks at an unchanging temperature of 106° to 107·5° (42°–43° C.), they preserve their external appearance completely, as well as their ability to grow, but gradually lose their power of infection. Inoculations made with this "vaccine virus" produce at most an insignificant disturbance. But what is especially remarkable is that the animals thus vaccinated are said to enjoy immunity thereafter from infection with actual anthrax. Pasteur was the first to make this assertion; and he proposed that the prophylactic inoculation of sheep and other animals liable to anthrax should be undertaken on a large scale, promising the farmers that very great benefit would result. This promise has not yet been completely fulfilled, although there can be no doubt that in general Pasteur was correct. Experiments instituted by Koch and others have shown that, although Pasteur's vaccination protects against the artificial inoculation of anthrax, it does not, at least as now performed, afford immunity from the natural anthrax, which usually results from infection within the intestine. French investigators have lately made known new methods of producing an artificial diminution of the growth and virulence of anthrax bacilli, and in part also of other varieties of bacteria. Chauveau has found that cultures of anthrax bacilli exposed for several days to an atmospheric pressure of three to twelve atmospheres, or to compressed oxygen, lose a portion of their virulence; and that animals inoculated with bacilli thus attenuated gain an immunity to inoculations with the original anthrax poison. The statements of Arloing are very remarkable. He says that the direct play of sunlight, or even of a concentrated artificial light, upon the cultures exercises a restraining influence upon the growth and poisonous properties of the bacilli, and that inoculation material weakened in this way may be employed for the protection of animals.

2. The treatment of malignant pustule is surgical. Cauterization is often tried with such materials as caustic potash, nitric acid, or carbolic acid; but it should always be borne in mind that such manipulations may easily contribute to a local extension of the anthrax poison. For the same reason one should hesitate to incise or to excise the pustules. We are therefore usually obliged to confine ourselves to the prescription of a suitable position for the affected member, and the application of an ice-bag over the diseased spot. The treatment of intestinal mycosis must be purely symptomatic. We may try the effect of calomel, salicylic acid, stimulants, and baths.

CHAPTER XXIII.

TRICHINOSIS.

(*Trichinatous disease.*)

The Natural History of Trichinæ.—The *trichina spiralis*, one of the class of round worms or nematoda, has long been known to occur occasionally in the muscles of men and certain animals; but it was not until 1860 that Zenker showed that trichinæ are capable of exciting in man a dangerous and sometimes fatal disease. Since then numerous individual cases and quite extensive epidemics have been reported; and the labors of Virchow, Leuckart, and others have taught us the anatomy and mode of development of this peculiar parasite.

The trichina appears in two shapes—as intestinal trichina and as muscular trichina. The intestinal form is a small white worm, visible to the naked eye. The female is 3–4 mm. long, the male only 1–1·5 mm. They have well-developed digestive and sexual organs. The male is distinguished by two little processes at the tail. The muscular trichina (*vide* Fig. 13) is a small worm 0·7–1 mm. long. It is found coiled up among the muscular fibers, inside a connective-tissue capsule, which is often calcified.

The events in the life of the trichina are remarkable. If living muscular trichinæ reach the human stomach, viz., through the eating of trichinatous pork, the capsules are dissolved, and the trichinæ, thus set free, grow in two or three days into sexually perfect intestinal trichinæ. In the uterus of the impregnated female the eggs develop into embryos, which are born already hatched. The birth of embryos begins seven days after the ingestion of the muscular trichinæ, and seems to continue for some time. A single female is said to produce more than one thousand embryos. These latter begin their travels soon after birth, and reach the voluntary muscles. As to the routes they choose we are still somewhat in doubt. Some authorities state that the trichinæ penetrate through the walls of the intestine and the abdominal cavity into the connective tissue. Others affirm that they enter the lymphatic vessels, or exceptionally the blood-vessels. They penetrate into the primitive fibers of the muscles, and cause them to disintegrate. Finally, they coil themselves up, attain the size of muscular trichinæ in about fourteen days, and become encapsulated. Each capsule usually contains but one, although it may inclose as many as four. The capsule is formed partly by an excretion from the trichina, and partly from the reflex hyperplasia of the surrounding connective tissue. The process of development is now complete. The muscular trichinæ seem, unlike the intestinal form, to have a very long lease of life, and usually endure till the death of their host. They are often found accidentally at autopsies. They are most abundant in the diaphragm, the intercostal muscles, the muscles of the larynx and throat, and in the biceps.

Ætiology of Trichinosis.—The only cause yet known for trichinatous disease in man is the ingestion of trichinatous raw or underdone pork—e. g., smoked ham. Swine are pre-eminently subject to trichinæ. They probably become infected in various ways, e. g., from the fæces of human beings and swine suffering from trichinosis, or through the ingestion of the trichinatous flesh of other swine. The waste of slaughter-houses is often fed out to swine, and the disease thus disseminated. Many affirm that swine are also infected by eating rats infested with trichinæ.

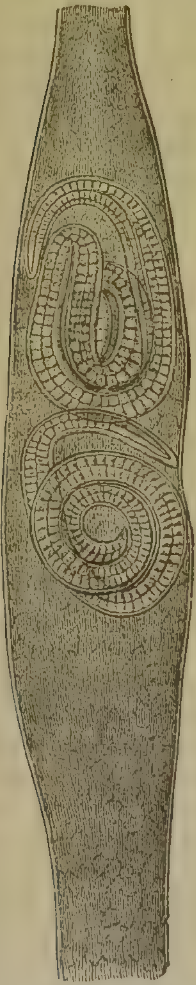

FIG. 13.—(From HELLER.) An isolated primitive bundle with two free trichinæ in the sheath of the sarcolemma. Much enlarged.

Clinical History.—The symptoms in man correspond in general to the developmental and vital processes of the trichinæ, as above depicted. In individual cases, however, the separate stages are quite often obscured, probably because all the parasites do not develop simultaneously, or because there are relapses. The first symptoms are gastro-intestinal. At the commencement there is a feeling of pressure in the epigastrium, with nausea and vomiting. Later, diarrhœa is prominent, becoming in some cases so violent as to remind one of cholera. It is not impossible, although rare, to find intestinal trichinæ in the stools. Sometimes there is constipation instead of diarrhœa. In some cases the initial gastro-intestinal symptoms are but slight. Frequently, even in the beginning of the disease, there is complaint of pain and stiffness in the muscles, too early for it to be due to the migrations of the trichinæ.

The genuine severe muscular symptoms, due to the myositis produced by the trichinæ in the muscles, do not begin till the second week, or even later. In many cases, where the invading parasites seem to be relatively few in number, the muscular symptoms are slight, or wholly absent. In the more severe cases, however, they may be extremely violent and distressing. The muscles become swollen, firm and hard, very tender on pressure, and very painful. The patient avoids all movements and contraction of the muscles as much as possible, lying, with flexed arms and with legs either extended or likewise flexed, motionless in bed. The patellar reflex almost always disappears, and on testing the electrical reactions there is found a considerable diminution of muscular excitability to both the galvanic and faradic currents, sometimes associated with delayed contractions, and abnormally long duration of the same after the stimulus ceases (Eisenlohr). The masseters and the pharyngeal and laryngeal muscles are attacked, so that there is difficulty in mastication and deglutition, and hoarseness. The participation of the motores oculi causes pain in the eyes. The condition of the diaphragm, intercostals, and abdominal muscles causes serious difficulty in respiration. There is distressing dyspnœa, and expectoration is so hampered that secretions accumulate in the air-passages. Some of the fatal cases of trichinosis are principally due to this impairment of respiration. The condition may be aggravated by diffuse bronchitis or lobular pneumonia.

Third in the list of important symptoms comes œdema. It appears, toward the end of the first week, in the eyelids. Somewhat later it involves the upper and lower extremities. What produces it is not quite clear. It has been regarded as in part inflammatory and in part the result of occlusion and thrombosis of the smaller lymphatics. Cutaneous eruptions also develop—e. g., vesicles, wheals, petechiæ, and pustules. Frequently there is profuse perspiration, consequent upon which abundant crops of miliaria or sudamina may appear.

In well-marked cases there may be quite high fever and other severe constitutional symptoms in addition to the local disturbances already discussed. The temperature may for a time reach 104° to 106° (40°–41° C.); but the fever is seldom continuous for any length of time, being usually interrupted by frequent and considerable intermissions. There are also a rapid pulse, headache, stupor, and other symptoms suggesting typhus or typhoid fever. In fact, the first case in which trichinosis was recognized at the autopsy (by Zenker of Dresden) had been regarded before death as typhoid. The urine may be albuminous; and, in rare instances, nephritis is seen.

The duration of the disease varies widely. There are mild cases often unrecognized, which get well after slight symptoms have lasted two or three weeks. More pronounced cases occupy six to eight weeks, or even a much longer time. Of the more severe cases about one third prove fatal, usually from the fourth to the sixth week. Sometimes death is caused by the severity of the constitutional disturbance, but usually from disabled respiration. Even if the case ends favorably, recovery is often very tedious.

Pathology.—The autopsy reveals little that is characteristic excepting the changes in the muscles. There are sometimes the signs of hæmorrhagic catarrhal inflammation of the small intestine. The spleen is not enlarged. Very often the liver is decidedly fatty. What should cause this in trichinosis has not yet been determined. The lungs often present islets of lobular pneumonia, or even sometimes of gangrene. The trichinæ are found in the muscles, beginning with the fifth week. They can be recognized by the naked eye as little whitish lines. We have already named the muscles chiefly infested. Under the microscope we see the fibers in which the trichinæ lie transformed into a fine granular mass. The nuclei of the muscular fibrillæ are greatly increased in number in the neighborhood of the coiled-up parasite. Finally, the sarcolemma collapses, and becomes greatly thickened upon its external surface by a hyperplasia of connective tissue. The muscles also present many other degenerative changes, such as a flaky disintegration, waxy degeneration, and the formation of vacuoles. There is furthermore a marked increase of nuclei in the interstitial tissue of the muscles. Within the intestines are sometimes to be found, even after several weeks' illness, numerous living intestinal trichinæ—a fact of importance from a therapeutic point of view.

Treatment.—The trichinæ may still be alive in pork that has been smoked or salted or half-cooked—e. g., many sausages are unsafe. The only possible prophylaxis, as far as the individual is concerned, is therefore to avoid all such food. A real protection for the public against the disease is also afforded by governmental microscopic inspection of meat, as already established in many places.

When an individual has become infected with trichinæ, if it is possible that intestinal trichinæ still are present, the treatment must always begin with the exhibition of purgatives, such as compound infusion of senna, calomel, or castor-oil. Of the remedies which are calculated to destroy the intestinal trichinæ, glycerine, which was first recommended by Fiedler, seems to be the most efficient. It must be given in rather large doses, say a tablespoonful every hour. Other drugs are much less reliable, but we will name among them benzine in the dose of one to two drachms (grm. 4 to 8) in capsules, and picric acid in pills—the daily dose being 5 to 8 grains (grm. 0·3–0·5).

Later on, when the invasion of the muscles has already begun, we are unfortunately almost without resource. The muscular pains can be alleviated by narcotics, particularly morphine subcutaneously, poultices, and chloroform-oil as an embrocation.* Protracted warm baths are excellent. Antipyrine and salicylic acid are also said to do good in many cases.

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* Generally one part of chloroform to ten of olive-oil. It is not officinal in Germany, but is weaker than the linimentum chloroformi (U. S. P.).—TRANS.

DISEASES OF THE RESPIRATORY ORGANS.

SECTION I.

*Diseases of the Nose.**

CHAPTER I.

CORYZA.

(*Snuffles. Rhinitis.*)

Ætiology.—The well-known symptoms of coryza depend upon a catarrhal inflammation of the nasal mucous membrane. Although this catarrh may often be due to infectious influences, still we can not deny that it is one of those diseases which may be caused by taking cold. Daily experience teaches us how often coryza follows an evident exposure to cold, like wetting the feet. We may mention its contagiousness as an argument in favor of its infectious character, and this may be illustrated by the fact that it may be conveyed by handkerchiefs, kissing, etc., but an experimental transmission of common coryza has not yet been successful.

Coryza may also arise from the action of chemical irritants or mechanical irritants, like dust, on the nasal mucous membrane. The iodine coryza, which occurs from the internal use of iodine, is especially noteworthy. In this form iodine can easily be detected in the nasal secretion. The idiosyncrasy of many people to ipecacuanha is also well known, the very smell of it setting up a coryza. A severe coryza is the chief symptom, too, in hay fever,† which is probably due to the action of the pollen of certain grasses on the respiratory mucous membrane.

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* Special treatises on the pathology and therapeutics of nasal diseases are to be found in the following works: Michel, "Krankheiten der Nasenhöhle." Fraenkel, "Diseases of the Nose," in "Ziemssen's Cyclopedia." Störk, "Klinik der Krankheiten des Kehlkopfes, der Nase, und des Rachens." Schech, "Krankheiten der Mundhöhle, des Rachens, und der Nase." Moldenhauer, "Krankheiten der Nasenhöhlen," etc.

† The disease called hay fever (*catarrhus æstivus*) is of frequent occurrence in England and North America, although rare with us in Germany. It usually affects men in middle life, less often women. Some individuals are peculiarly liable to the disease. For them an attack may be produced merely by walking across a meadow or near a grain-field at that season when the grasses are in bloom, i.e., about May to July. As already intimated, it is supposed that the grains of pollen excite the disease, being diffused in the air and thus drawn into the nostrils. At any rate, they have repeatedly been found in the nasal secretion and also in the tears of affected persons. The *symptoms* consist in a very severe coryza, with burning of the nose and violent sneezing. The erectile tissue of the nose is probably acutely swollen. Usually these symptoms are accompanied by a well-marked conjunctivitis with œdema of the eyelids. In severer cases there is furthermore a catarrh of the larynx and bronchi. There is frequently a tendency to violent attacks of asthma ("hay-asthma"), especially at night (see the chapter on bronchial asthma). The *treatment* consists first in avoiding the cause by change of residence, as by going to the sea-shore. For the nasal catarrh douches are chiefly recommended, such as a solution of 1 part of quinine to 500–1,000 parts of water, or a solution of carbolic acid. The administration of iodide of potassium might be tried, particularly where there is asthma.

Finally, we must bear in mind that coryza may often be only a symptom of another disease like measles, syphilis, or glanders, and that severe purulent inflammation of the nasal mucous membrane may be excited by the presence of the secretion from a gonorrhœal or blennorrhœal conjunctivitis.

The **symptoms** of coryza are in most of the milder cases of a local nature only. The secretion is troublesome; at first it is scanty and mucous, but later it becomes more abundant and watery; and sometimes it is purulent. The nasal passages are not infrequently closed from the swelling of the mucous membrane. The patient necessarily has to breathe through the mouth, which explains the well-known nasal speech. This closure of the nares may give rise to dangerous attacks of dyspnœa in children, especially in infants, who have to breathe through the nose when sucking at the breast. The sense of smell is always diminished. The local sensations of pain and burning are due chiefly to a mild inflammation of the skin of the nostrils and upper lip set up by the irritation of the secretion. The irritated condition of the inflamed mucous membrane occasions a feeling of tickling and itching in the nose, and frequently by a reflex action violent sneezing. The symptoms are more severe if the cavities adjacent to the nose are attacked by catarrh, and if in them accumulations of secretion occur. Marked pain in the forehead occurs in catarrh of the frontal sinuses. The sinuses of the ethmoid and sphenoid bones, and the antrum of Highmore, may also be implicated. Much more frequently a severe coryza sets up an inflammation in adjacent mucous membranes. Thus we find following a coryza a conjunctivitis, an affection of the ear, a sore throat, or a laryngitis. In persistent coryza an eczema is not infrequently excited on the skin of the upper lip, and mention has already been made of the fact that coryza may sometimes act as the exciting cause of an erysipelas.

In severe coryza we may sometimes have quite a marked general disturbance, and often slight elevations of temperature. The "coryza fever" in children, for instance, is well known.

Treatment.—Special treatment is usually unnecessary, for most cases recover of themselves in a few days. With abundant secretion, especially in fresh cases, Hager's "coryza remedy" [as an inhalation] is worthy of trial; this consists of ten parts each of alcohol and carbolic acid, and five parts of ammonia-water. A snuff of calomel is also greatly praised. When the secretion forms abundant dry scabs, an attempt should be made to wash them out by injections of warm fluids, like warm milk. The upper lip and the nostrils should be smeared with vaseline or simple ointment to protect the skin from the action of the secretion. Only in the rare cases of a severe purulent catarrh can an energetic local treatment of the nasal mucous membrane be necessary. Here we may use douches, sprays, or inhalations of astringents like tannin or alum, or we may apply caustics like nitrate of silver.

CHAPTER II.

CHRONIC RHINITIS.

(*Rhinitis chronica hypertrophica and atrophica. Ozæna.*)

1. **Chronic Rhinitis.**—It is in many cases impossible to determine the causes of hypertrophic rhinitis. Sometimes the condition seems to develop as a sequel to frequently repeated nasal catarrh, although in this case the relation is often reversed—it being the chronic rhinitis which occasions a predisposition to the frequent acute exacerbations of the catarrh. Certain diatheses (anæmia, scrofula)

appear to influence the development of the disease. This is also true of occupations which expose the individual to dust or smoke, and sometimes of malformation of the nose (for instance, deviation of the septum) and perhaps also of hereditary predisposition.

The anatomical changes consist in a slow but progressive swelling and hypertrophy of the mucous membrane. This seems spongy, and of a red or reddish-gray color. The greatest change is almost always found over the inferior turbinated bone, and next to that over the middle. In advanced cases the mucous membrane presents rough, uneven swellings, and even polypi. These changes are often visible upon inspection of the nostrils anteriorly, but they may escape discovery until a rhinoscopic examination of the posterior choanæ is made.

The disturbance occasioned by chronic hypertrophic rhinitis may be very considerable. Nasal respiration is obstructed, the voice nasal, the senses of smell and taste impaired. The nasal secretion is for the most part increased, but it may be diminished. Often there is a tendency to nose-bleed. Many patients complain also of headache.

The frequent involvement of neighboring organs is important. This applies particularly to the ear. Deafness is caused both by the obstruction of the openings of the Eustachian tubes, and not infrequently also by extension of the catarrh to the lining membrane of the tubes and the middle ear. Very frequently the disease is associated with chronic naso-pharyngitis or pharyngitis. The visible portion of the nose is not infrequently affected, as shown by redness and swelling of its tip.

A fact of especial interest is that such a diseased state of the nasal mucous membrane may give rise to reflex neuroses (Voltolini, Hack, and others). Although, in our opinion, many of the specialists on the nose go too far in this direction, there is no room for doubt that attacks of migraine, vertigo, certain varieties of headache, and, above all, many forms of bronchial asthma, may bear a close relation to diseases of the nose. We shall revert to this point later on. (See especially the chapter on bronchial asthma.)

The *treatment* of chronic hypertrophic rhinitis, in order to be successful, demands complete destruction and removal of the hypertrophic portions by means of the galvano-cautery. For particulars, we must refer to the directions of specialists. In milder cases, however, benefit may follow the insufflation of a powder composed of one part of nitrate of silver to ten or twenty of common starch, or applications of lunar caustic may be made.

2. **Chronic Atrophic or Fetid Rhinitis. Ozæna Simplex.**—This frequent and peculiar form of chronic rhinitis is the cause of that well-known condition which is characterized by a peculiar and extremely disagreeable odor from the nose, which is therefore usually termed ozæna, from the Greek word ὄζειν, to stink. The disease consists in a slow, progressive atrophy not only of the mucous membrane with its vessels and glands, but finally also of the bones, and this atrophy is not preceded by hypertrophy. Thus the nasal cavities become abnormally large. The turbinated bones grow smaller and smaller, so that finally they are represented merely by narrow ridges. Furthermore, the scanty purulent secretion has a tendency to dry up and form adherent greenish-yellow scabs and crusts, which undergo a peculiar putrefactive decomposition and give rise to the unbearable stench. It is possible, but not yet demonstrated, that a specific kind of bacterium plays a part in the process.

The ozæna generally develops in childhood. It usually begins insidiously, but in other cases apparently is a sequel of some acute disease, such as measles. Anæmia and scrofula deservedly rank as important predisposing causes.

The subjective symptoms are often not marked. This is partly explained by the fact that the patient has usually completely lost his sense of smell, but for that very reason the discomfort of his friends may be the greater. The feeling of dryness in the nose may prove annoying, and there are often complaints of headache and of pressure in the eyes. Inasmuch as the naso-pharynx and the posterior pharyngeal wall are almost always implicated in the process, the patient often suffers from hacking and a tendency to cough and vomit. Such portions of the secretion as are swallowed sometimes give rise to a considerable chronic disturbance of the stomach. Upon physical examination we are first struck by the unusual breadth of the nostrils. With the rhinoscope the extent of the atrophy is still better seen. The mucous membrane is pale or slightly red, and covered with dry scabs. Sometimes superficial ulcers are formed. Usually, as we have said, the superior portion of the pharyngeal mucous membrane shares in the disease. The posterior wall of the pharynx is seen to be atrophied, smooth as if it were varnished, and often covered with crusts. The process may involve the soft palate, and even the larynx, and not infrequently the disease is associated with inflammation of the middle ear.

It should be added that the true ozæna must not be confounded with other processes which likewise give rise to a foul smell from the nose. Tuberculous disease of the nasal mucous membrane and nasal bones is not rare, particularly in "scrofulous" children (Demme); nor should we forget the syphilitic affections of the nose, tertiary and hereditary syphilis.

Treatment.—The treatment of ozæna can be made effective only by the aid of local applications as prescribed by specialists. Even then the treatment is a prolonged one, and demands much patience on the part of both patient and physician. Besides local applications, we must also bear in mind the necessity of constitutional treatment, especially in syphilis and tuberculosis.

The object of local treatment is to remove the secretion in order to get rid of the bad odor. Nasal douches, with disinfectant solutions like permanganate of potassium (1–3,000) or carbolic or corrosive sublimate, are here most useful. The solution is carefully injected into the nose, or the fluid is allowed to run gently into one nostril from an irrigator while the patient keeps his head bent forward; it then runs through the naso-pharynx and out through the other nostril. The patient soon learns to retain the fluid in the pharynx and eject it from the mouth. All nasal douches must at first be used with care and under the eye of the physician. The fluid should be injected at the lowest pressure possible, so that none of it may enter the adjacent cavities or the Eustachian tube. Furthermore, all solutions used as a douche must be luke-warm—90° to 95° (25°–28° R.). Besides the regular use of douches, painting and the insufflation of powders, like boracic acid, aceto-tartrate of aluminum, etc., are sometimes employed. The insertion of tampons of dry absorbent cotton is to be recommended; under their use the secretion dries less easily and the odor is diminished. These tampons should be changed daily. It is advantageous to medicate the tampons with a one-per-cent. solution of creolin or with Peruvian balsam or some similar drug. Tincture of iodine is also recommended. Of late many attempts have been made to treat chronic nasal catarrh by the galvano-cautery. With regard to the details of this as well as of other methods, we must refer to special treatises on the subject.

CHAPTER III.

NOSE-BLEED.

(*Epistaxis.*)

Although in many cases nose-bleed is only a symptom of some other disease, still we are justified in a short description of it, partly because frequently repeated nose-bleeds often first call our attention to some other existing disease, and partly because the treatment is of practical importance.

Many persons are subject to habitual nose-bleed, which comes on either from slight causes, from violently blowing the nose, from physical exertion, from overheating, or even without any special cause. This habitual nose-bleed is sometimes, but by no means always, the sign of a general hæmorrhagic diathesis, which is hereditary in many families. (See the chapter on Hæmophilia.) In other cases the nose-bleed is the result of some chronic disease. It occurs especially in leukæmia, in disease of the heart, in contracted kidney, and as a symptom of the so-called hæmorrhagic diseases, like scurvy, purpura hæmorrhagica, etc. It is also not uncommon in acute febrile diseases, like typhoid and scarlet fever. Finally, diseases of the nose itself may give rise to hæmorrhage. The occurrence of nose-bleed as a form of so-called "vicarious menstruation" has often been described, but we must always be very guarded in admitting it as a fact.

In many cases nose-bleed is a very transitory symptom, wholly without danger, and in one sense it may even be advantageous. When there is headache, or a feeling of fullness in the head, there is often relief after an epistaxis. Nose-bleed is dangerous, however, when it takes place in those who are already weak and anæmic, or when it is so persistent and abundant as to cause a marked general anæmia. The latter is recognized by the pallor of the face, by the appearance of general weakness, by vertigo, tinnitus, and a weakened pulse. In such cases the physician's interference is always necessary. In every case of nose-bleed it is important to examine the posterior wall of the pharynx in order to see whether the blood is not flowing backward from the posterior nares. The hæmorrhage is often thought to stop when no more blood comes from the nostrils, and yet the blood keeps flowing posteriorly.

In every severe nose-bleed rest is the chief thing to be enjoined, and the patient must be told to avoid unnecessarily blowing, wiping, or drying the nose. By quietly and persistently closing the nostrils with a handkerchief a thrombus is often formed without any further medication, and the bleeding stops. The application of cold water (iced water), in which a little vinegar may be put, is a good thing. If the bleeding does not stop, we may next try a tampon of common absorbent cotton or styptic cotton in the nostril from which the blood comes. If this does not succeed, the posterior nares must be plugged by means of a "Bellocq's canula." In case of emergency we may use an elastic catheter, which is passed through the inferior meatus into the pharynx and out by the mouth. The tampon is fastened to the catheter and brought up into the posterior nares by drawing the catheter back through the nose. Internal remedies to check the blood are very uncertain in their action. Ergotine, in one-grain pills (grm. 0·03), every three or four hours, is the first one to employ, if we wish to try to check the bleeding by this means.

SECTION II.

DISEASES OF THE LARYNX.

CHAPTER I.

ACUTE LARYNGEAL CATARRH.

(*Acute Laryngitis.*)

Ætiology.—Taking cold plays a prominent part in the ætiology of acute laryngeal catarrh, as every one knows. Its influence can not properly be wholly denied, since the more intimate relation between taking cold and the origin of a catarrh is still unknown. The disposition to laryngitis differs very much in different people, so that some take a catarrh much more easily and more frequently than others. Besides cold, direct irritants which attack the laryngeal mucous membrane often set up a laryngitis; among these are in particular the inhalation of smoke and of injurious gases and vapors. Many laryngeal catarrhs, too, arise from excessive speaking, shouting, or singing, particularly if other injurious influences act on the larynx at the same time. Finally, laryngitis may appear as a complication or as a secondary affection, in other diseases, especially in measles, less frequently in typhoid, scarlet fever, and erysipelas. Catarrh of the larynx is very often combined with catarrh of the nose, the pharynx, and the larger bronchi.

Symptomatology.—Although the symptoms of laryngitis usually make the diagnosis easy and certain, yet an accurate understanding of the extent and intensity of the catarrh can be obtained only by a laryngoscopic examination,* which therefore should be employed in every severe case. The laryngeal mirror shows a decided reddening and swelling of the mucous membrane, varying with the intensity of the catarrh, and most marked on the true and false vocal cords and between the arytænoid cartilages. We often see small collections of mucus here and there on the membrane. In individual cases different parts of the larynx are especially affected. In intense inflammations superficial erosions are often met with, especially on the vocal cords. In other cases the mucous membrane shows a grayish-white coloring in some places, due to a thickening of the epithelium. Small hæmorrhages in the mucous membrane are also occasionally seen. Very often we see on phonation an incomplete closure of the glottis, so that a little oval space is left between the vocal cords. This slight "catarrhal paresis of the vocal cords" is probably of muscular origin, and depends chiefly upon an affection of the thyro-arytænoid muscles.

Of the other symptoms of laryngeal catarrh, hoarseness is particularly to be mentioned, for in many cases the diagnosis of laryngitis may be made from this alone. It is either due directly to the anatomical changes of the cords, or to the paresis just mentioned. The degree of hoarseness is of course very different in different cases, and varies from a simple "roughening" or "deadening" of the voice to a complete loss of voice (aphonia).

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* More extensive observations on laryngoscopy and on many details of the pathology of laryngeal diseases, which have been carefully investigated by specialists and which can not be mentioned here, are to be found in the following works: Türck, "Klinik der Krankheiten des Kehlkopfes," 1866. Semeleder, "Laryngoskopie," 1863. Tobold, "Laryngoskopie," 1874. Störk, "Klinik der Krankheiten des Kehlkopfes, der Nase, u. des Rachens," 1880. Mackenzie, "Diseases of the Throat and Nose," 1880. B. Fraenkel and v. Ziemssen, "Diseases of the Larynx," in Ziemssen's "Cyclopædia." Gottstein, "Krankheiten des Kehlkopfes," third edition, 1890. Schrötter, "Vorlesungen über die Krankheiten des Kehlkopfes."

The cough in laryngitis may be very severe, and is often recognizable by its harsh, hoarse ring as a "laryngeal cough." It is at first usually dry, and later on it is associated with a scanty muco-purulent expectoration, which is sometimes tinged with blood.

Pain in the larynx is generally only moderate. The subjective symptoms consist chiefly of a disagreeable feeling of itching, burning, and dryness in the throat. After prolonged speaking, however, the pain in the larynx may sometimes be quite severe. External pressure on the larynx is often somewhat painful. Difficulty in swallowing, when it occurs, is usually due to an accompanying pharyngitis, but it may also be dependent upon an affection of the epiglottis and the arytænoid cartilages.

The general health is affected in very different degrees. Many patients feel quite well except for the hoarseness, while others are affected with considerable debility, mild headache, and even at times slight febrile disturbances.

Dyspnœa is not present in the common laryngitis of adults, even if there is decided swelling of the false vocal cords or of the ary-epiglottic folds. There is, however, a severe form of acute laryngitis, the so-called *laryngitis hypoglottica acuta gravis* (*chorditis vocalis inferior*), affecting not only children, but adults, in which well-marked symptoms of suffocation may be present. In this form there is an acute, very well marked swelling of the mucous membrane in the inferior, "sub-chordal," laryngeal space, which leads to a stenosis.

In children, however, on account of the greater narrowness of the child's larynx, symptoms of stenosis are not rare even in the milder forms of laryngitis, and therefore they have led to the establishment of a special disease, the so-called false croup.

The **false croup** (*laryngitis stridula*) of children usually follows a slight coryza. A harsh, hollow, ringing cough comes on, almost always suddenly and usually at night, by which the child is awakened out of sleep. The paroxysms of coughing are broken by long-drawn, noisy inspirations. The child is anxious and restless, the respiration is labored, the pulse is rapid. Such attacks recur several times during the night. The next day the child is quite lively, plays about, and has at most a slight cough. The next night, rarely sooner, the same severe attacks are repeated. After that there remains, as a rule, nothing but a slight catarrh, which completely disappears in a week or two. These sudden attacks have their origin partly in a marked swelling of the mucous membrane, occurring during sleep, partly in a neglected accumulation of secretion, and probably often also in a reflex spasm of the glottis. No other anatomical cause than a simple catarrh of the larynx is apparent, and on examining the pharynx, and, if possible, the larynx also, we find no trace of that diphtheritic process which is usually present in true laryngeal croup. It is remarkable that many children, and sometimes several children of the same family, have a specially marked predisposition to false croup. The statement, therefore, that a child has had the croup several times almost always means that it has had this form of false croup just described.

Acute laryngitis lasts only a few days in mild cases, and a week or more in severe cases. With improper care and unreasonable conduct on the patient's part an acute catarrh may run into the chronic form. We hardly ever see a fatal result in adults, even in the severe form, or in the false croup of children.

The **treatment** of acute laryngitis requires that especial attention be paid to the removal of all injurious influences. In every severe laryngitis the patient should stay in his room, and children are better off in bed. The patient should talk as little as possible. In all severe cases smoking, too, is to be forbidden. It is a good plan to furnish plenty of warm drink. Hot milk, mixed with Seltzer or Ems water, is readily taken by most patients. If there is an inhaler at our disposal, we may let the patient inhale simple steam, or a one- or two-per-cent. solution of common salt Inhalations of astringents are usually unnecessary. The patient may also breathe steam without any special apparatus. When there is marked irritation from coughing we may give a little morphine. With more marked local symptoms, especially if there is much pain on swallowing from swelling of the epiglottis and the mucous membrane over the arytænoid cartilages, the patient may suck pieces of ice slowly. In severe cases of acute laryngitis, with evident symptoms of stenosis, ice must be energetically used as an internal and an external application. Sometimes, too, a few leeches applied in the region of the larynx afford distinct relief. Among external applications a mustard plaster over the front of the neck is to be recommended when there are marked local symptoms. Cold, wet compresses about the neck are also of advantage in all cases.

In the false croup of children we should use, as a rule, the same treatment as has just been described. The child should take warm drink, and a mustard paste or hot poultices should be applied to the neck. We should be rather cautious with regard to the favorite treatment with emetics, such as ipecac and sulphate of copper, although it can not be denied that they sometimes work very well.

These means are entirely sufficient for the treatment of acute laryngitis. It is only exceptionally that we find ourselves led to employ in acute laryngeal catarrh an energetic local treatment of the laryngeal mucous membrane, like painting with a 1–15 solution of nitrate of silver.

We must bear in mind that a rational hardening process is of distinct prophylactic value in persons, especially in children, with a recognized tendency to laryngitis, sore throat, etc. The best method is to bathe the neck and chest with cold water regularly morning and night.

[A mild emetic can do no possible harm in false croup, and very often cuts the attack short. The application of a sponge, moistened with water as hot as the child will bear, to the region of the larynx deserves mention.]

CHAPTER II.

CHRONIC LARYNGITIS.

(*Chronic Laryngeal Catarrh.*)

Ætiology.—Chronic laryngitis develops from an acute catarrh, or comes on gradually from the action of injurious influences on the larynx (see the preceding chapter). Chronic laryngitis, therefore, is in many cases a disease arising from the occupation, and is seen especially in singers, public speakers, criers, inn-keepers, etc. It is very frequent in drunkards, and in such cases it is almost always associated with a chronic pharyngitis. It is frequently stated that too long a uvula sets up a chronic laryngitis by constant irritation of the entrance to the larynx, and that if the uvula is amputated the disease is cured.

Symptomatology.—A laryngoscopic examination is very desirable in acute laryngeal catarrh, but it is the physician's absolute duty to make one in every chronic laryngitis, for only too frequently a persistent hoarseness is referred simply to catarrh when the laryngoscope gives quite another cause for it, such as paralysis of the vocal cords or new growths. Furthermore, we must always remember that a chronic laryngitis may be a complication of tuberculosis, syphilis, or chronic nephritis. On the other hand, those physicians who make a specialty of laryngology often neglect a careful and satisfactory examination of the rest of the body when there are laryngeal troubles.

The laryngoscopic appearance in chronic catarrh may be so like that in an acute catarrh that we can not distinguish between them without the history obtained from the patient. The redness of the mucous membrane, however, is usually less intense, and the vocal cords have more of a dirty grayish-red appearance. Quite frequently in persistent catarrhs a thickening of particular parts of the mucous membrane is developed, especially of the folds between the arytænoid cartilages. This swelling is of practical importance, because it furnishes a mechanical hindrance to the closure of the arytænoid cartilages, and in that way contributes to the development of the hoarseness. We also find limited and marked thickening of the epiglottis, the false vocal cords (especially in public speakers and preachers), and the true vocal cords. Türck has described a peculiar form of chronic laryngitis, in which rough prominences are formed in the middle of the true vocal cords, under the name of *chorditis tuberosa*. We not infrequently find in chronic catarrh superficial erosions, especially on the true vocal cords. Superficial but very painful fissures occur upon the posterior wall of the larynx between the arytænoid cartilages. We also very often see a disturbance of motion of one or both vocal cords, due sometimes to muscular paresis and sometimes to mechanical conditions.

The other symptoms of chronic laryngitis are hoarseness, cough, and abnormal sensations in the larynx. The hoarseness is of every degree, from mere roughness, frequent "cracking" of the voice, to almost complete aphonia. The cough is ringing, hoarse, deep, and rough. The expectoration is scanty, usually simply mucous, but sometimes a little bloody. The subjective sensations in the larynx are a feeling of burning and itching, and of dryness and tickling. They usually increase after any protracted use of the voice.

We must also mention as a very rare but practically important and peculiar form of chronic laryngitis the **chorditis vocalis inferior hypertrophica** (Gerhardt), or **laryngitis hypoglottica chronica hypertrophica** (Ziemssen). In this form there is a very gradual hypertrophy, and finally a contraction of the mucous and especially the submucous connective tissue in the inferior laryngeal space. More rarely the same changes are seen in the upper part of the larynx. The special symptom of the disease, besides a chronic hoarseness, is the appearance of a gradually increasing stenosis of the larynx. The respiration is always labored, the inspiration noisy and protracted. In many cases there are at times such attacks of suffocation that life can be prolonged only by tracheotomy. The diagnosis can be made only by the aid of the laryngoscope. We see beneath the glottis a little fissure surrounded by the thick and swollen mucous membrane of the laryngeal walls.

The precise ætiology of this disease is as yet unknown. It appears to have nothing to do with syphilis, contrary to the former belief.

The **treatment of chronic laryngeal catarrh** is always a tedious and laborious task, the success of which depends in great measure upon the good will and energy of the patient. In the first place, then, we should endeavor to remove as far as possible those injurious influences which have excited and kept up the catarrh. It is often easier to give good advice here than to follow it. Nevertheless, it is the task of the physician to impress upon the patient the urgent necessity of taking care of the larynx, and to forbid as far as possible all protracted speaking, singing, staying in smoke or dust, smoking, and drinking alcoholic liquors.

Local treatment takes the second place. Among the most useful means to employ are inhalations of astringent solutions, like a one-per-cent. solution of either tannin or alum. When there is great sensitiveness of the larynx, the patient may also inhale narcotics, a mixture of fifty parts of cherry-laurel water with a thousand parts of water, or a four-per-cent. solution of bromide of potassium. The inhalations should be used two or three times a day, and last about five minutes each time. Direct applications to the larynx are much more effective than inhalations, but these can be employed only by the aid of a laryngeal mirror. Of these we use, first of all, nitrate of silver, at first in a weak solution (one to thirty); later in a more concentrated form (one to ten, or even one to five). These applications are made every two or three days. Besides nitrate of silver, the larynx may also be painted with pure tincture of iodine, or with iodine and glycerine, or with concentrated solutions of alum or tannin. Where the secretion of mucus is considerable ,benefit will be obtained from inhalations of turpentine, or of *oleum pini* or *oleum pini pumilionis* (P. G.).

Water-cures are also often prescribed in chronic catarrh of the larynx. These are so far of advantage that, from the greater care which the patient takes, and from the good air, the catarrh improves. Empirically, we prescribe, especially for "full-blooded" patients, the cold sulphur springs, like Nenndorf, Eilsen, or Weilbach, or the sulphate of sodium waters, like Carlsbad and Marienbad, while we send those of delicate constitutions to Ems, Salzbrunn, Salzungen, Reichenhall, or Ischl.

The treatment of laryngitis hypertrophica, when it leads to stenosis, must be mechanical. Schrötter, in particular, has devised several methods in order to dilate the stenosis gradually by the introduction of bougies and harder dilators. The details of this treatment are to be found in the later special works referred to above.

CHAPTER III.

LARYNGEAL PERICHONDRITIS.

Ætiology and Pathological Anatomy.—The inflammation of the perichondrium of the laryngeal cartilages is in very rare cases apparently a primary disease. It is much more frequently secondary to other laryngeal affections, especially tuberculosis and syphilis of the larynx. Furthermore, it develops secondarily in severe acute diseases, most frequently in typhoid fever, more rarely in small-pox, diphtheria, etc. Superficial ulcerative processes in the mucous membrane often precede the perichondritis in these cases, and the participation of the perichondrium in the inflammation arises from their gradual deepening. Anatomically, we have to do as a rule with a purulent inflammation, which usually leads to the formation of circumscribed abscesses. Most laryngeal abscesses have their origin in the perichondrium.* The perichondrium is in part destroyed by the abscess and in part elevated from the cartilage. The cartilage then becomes necrotic, breaks in pieces, and is expelled in small particles or in masses.

Perichondritis occurs most frequently in the cricoid and arytænoid cartilages, much more rarely on the internal or external surface of the thyroid cartilage. Hence we distinguish an internal and an external perichondritis. A perichondritis of the epiglottis has also been repeatedly observed.

Symptomatology.—In the rare cases of primary perichondritis, marked laryngeal symptoms are speedily developed in a person previously healthy. These symptoms are pain and tenderness on pressure over the larynx, hoarseness, and cough; and to them are usually soon added the signs of a dangerous stenosis of the larynx. In secondary cases, which occur almost always in patients who are already seriously ill, the symptoms of stenosis are often the first to point to a severe disease of the larynx. On laryngoscopic examination, besides the general redness and swelling in particular places, we can sometimes recognize a circumscribed protrusion of the mucous membrane caused by the abscess. We often find, besides, a considerable collateral œdema of the surrounding mucous membrane, which œdema frequently has a greater share in the production of stenosis than has the primary affection itself. The dreaded œdema of the glottis (œdema of the ary-epiglottic ligament) in typhoid, tuberculosis of the larynx, etc., is usually due to perichondritis of the cricoid or arytænoid cartilages. Finally, we can see with the laryngoscope, especially in perichondritis arytænoidea, a considerable disturbance of motion of the affected arytænoid cartilage, and also of the vocal cords. In the later stages, if the abscess has been opened, or if it breaks of its own accord, and the whole cartilage or a part of it is expelled, we can make out the extent of the destruction that results more accurately by the laryngoscope.

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* Pure submucous abscesses, the so-called phlegmonous laryngitis, occur only in very rare cases.

Laryngeal perichondritis terminates fatally in a great number of cases from the development of stenosis. In other cases the most threatening symptoms may be averted for a time, but the primary disease, such as tuberculosis, finally comes to an unfavorable termination. In the rare cases in which recovery occurs after primary perichondritis or after the termination of the primary disease, such as typhoid, this recovery is often incomplete, since a chronic stenosis of the larynx remains from the ensuing cicatricial contractions.

The **diagnosis** is usually obscure during the first period of severe symptoms of stenosis, since it is difficult to make a laryngoscopic examination, and it is also not always easy to determine the condition. We are usually justified, however, in making the diagnosis, if in those diseases which we have mentioned, in which we know by experience that a perichondritis quite frequently occurs, the danger of suffocation arises in addition to the other laryngeal symptoms. It is of practical importance to recognize stenosis of the larynx with certainty, for it demands a speedy therapeutic interference.

Treatment.—In the beginning of the affection we may try to reduce the inflammation by the internal and external application of ice or by leeches; but if stenosis of the larynx occurs, surgical interference is usually necessary, for only in very rare cases do we see the abscess open of itself and a subsidence of the dangerous symptoms follow. In the majority of cases the patient can be saved from suffocation only by the timely performance of tracheotomy. The laryngeal abscess has been repeatedly opened internally by laryngologists with favorable results. If a chronic stenosis of the larynx remains after a favorable termination of the disease, either the patient must wear a tracheal canula all his life, or the attempt may be made to dilate the stenosis gradually by the methods referred to in the preceding chapter.

CHAPTER IV.

ŒDEMA OF THE GLOTTIS.

The practical importance of the subject demands a brief special description of œdema of the glottis, by which name we mean œdema of the entrance of the larynx, especially of the ary-epiglottic ligaments. We have already learned to recognize laryngeal perichondritis as one of its most frequent causes. In less deeply seated inflammations in the larynx and its neighborhood, however, œdema of the glottis may sometimes develop as a dangerous complication, especially in cases of laryngitis occurring in the course of severe acute diseases, like typhoid, small-pox, or erysipelas, or in inflammations of the larynx arising from severe mechanical or chemical irritation, like hot steam or corrosive substances, or from wounds of the larynx, or, finally, from foreign bodies in the larynx. The collateral œdema in angina Ludovici, in intense inflammations of the parotid gland, or the tonsils, etc., may in rare cases extend to the ary-epiglottic ligaments. Finally, œdema of the glottis occurs in rare cases as a complication of general œdema of the body, as a result of Bright's disease, disease of the heart, emphysema of the lungs, etc. Œdema of the glottis has been repeatedly observed to come on quite suddenly, especially in Bright's disease.

The chief symptom of œdema of the glottis is dyspnœa, which comes on as a result of the stenosis of the entrance of the larynx, and is sometimes extreme. At first this is chiefly on inspiration, but it soon comes on with expiration also. Respiration, especially inspiration, is accompanied by a loud laryngeal stridor. As a result of the incomplete entrance of the air, the efforts at inspiration involve the neck, the epigastrium, and the sides of the thorax. We see with the laryngoscope, if the examination be successful, an œdematous swelling of the ary-epiglottic ligaments, and often a swelling of the epiglottis and the false vocal cords. Sometimes we succeed in feeling the swollen parts with the finger.

If the dyspnœa reaches a degree which threatens life, an operation is the only thing which can afford relief. Laryngologists attempt to reduce the swelling by long incisions in the œdematous parts. If this does not succeed, tracheotomy must be performed. If the immediate danger to life is thus averted, further treatment should be directed to the disease which has given rise to the œdema.

CHAPTER V.

TUBERCULOSIS OF THE LARYNX.

(*Laryngeal Phthisis. Consumption of the Larynx.*)

Ætiology.—Since tuberculosis of the larynx is in most cases combined with tuberculosis of other organs, especially of the lungs, we must refer to the description of tuberculosis of the lungs for the general ætiology and pathology of the disease. A particular description of the special appearances in laryngeal tuberculosis, is, however, justifiable, because tuberculosis may at times begin in the larynx and may remain isolated there, at least for a time; and, furthermore, in many cases of laryngeal tuberculosis, which are evidently combined with pulmonary tuberculosis, the laryngeal symptoms are predominant in the clinical picture of the disease. Many physicians have, wrongly as we think, disputed the fact that tuberculosis can begin in the larynx. Clinical experience not infrequently teaches us that men, who up to that time were apparently in good health, are attacked with hoarseness, the disease being at first thought to be a common laryngitis, but at last, by its later course, proving to be a tuberculosis. In spite of the most careful examination, there are not to be found at first the slightest physical signs of disease in the lungs, and not till later do the manifest signs of a pulmonary tuberculosis succeed the symptoms of a laryngeal affection. In such cases it seems to us an affectation to claim that there is a primary pulmonary tuberculosis which could not be made out at first. Everything is much more in favor of the opinion that the tubercular poison, the tubercle bacilli, may sometimes first fix upon the larynx, excite the first symptoms of tuberculosis there, and only later attack the lungs.

In the majority of cases of laryngeal tuberculosis the symptoms are developed, of course, secondarily in the course of chronic pulmonary phthisis. We shall see that in these cases the disease of the larynx is to be considered as the result of an infection of the mucous membrane of the larynx by the tuberculous sputum which passes over it. In about one fourth of all cases of pulmonary tuberculosis this complication occurs, if we include all the mild diseases of the larynx. Marked and extensive tuberculosis of the larynx is much rarer, however.

Pathological Anatomy.—In its anatomical appearances the laryngeal affection which complicates pulmonary phthisis or occurs primarily is at first usually a simple catarrh of the mucous membrane, which does not differ in any remarkable way from any other laryngeal catarrh. Shallow erosions, too, on the vocal cords or between the arytænoid cartilages have nothing characteristic in themselves. In fact, it is even hard to decide whether the simple laryngeal catarrh and superficial ulcers in the larynx, which often occur in phthisical patients, are really in every case specific tubercular affections. Perhaps they are often only the result of the mechanical irritation from the frequent cough or of the chemical irritation from the sputum. This question is to be decided finally only by the discovery of the special tubercle bacilli in the laryngeal affections of phthisical patients.

The more marked changes in the larynx in phthisical patients, however, are without doubt always of tubercular origin. In these we find a characteristic tubercular infiltration, with the formation of miliary tubercles in the mucous and submucous tissues. When the infiltrated parts break down, extensive ulcers are formed which always extend farther, and whose favorite seat is on the arytænoid cartilages, the vocal cords, and the epiglottis. From the latter the ulcers not infrequently extend to the back of the tongue. In severe cases we often find a marked collateral œdema in the neighboring parts accompanying the inflammation, and sometimes the tubercular perichondritis which has already been described.

Clinical Symptoms.—In the beginning of tuberculosis of the larynx the laryngoscope usually shows nothing but the appearances of a simple catarrh. In the later stages, however, most of the special signs of the destructive tubercular process, like ulcers, infiltration, etc., can be very satisfactorily made out. In fact, we often get in this way a better picture of the disease than we do at the autopsy, for the hyperæmia and swelling of the parts are much diminished in the cadaver.

The other clinical symptoms of tuberculosis of the larynx vary very much with the extent and intensity of the process. Sometimes they consist merely in moderate roughness and hoarseness of the voice, but in other cases they increase to the most painful condition which is ever seen in any variety of tuberculosis. This is especially apt to be the case if the ulceration involves the epiglottis and the arytænoid cartilages. Swallowing is then extremely painful, so that the nutrition is very often impaired, and painful attacks of coughing frequently occur. If severe ulcerations attack the vocal cords, and their free mobility is affected to a marked degree, the hoarseness increases, and finally reaches a complete aphonia. Death finally occurs from general inanition, or, exceptionally, from œdema of the glottis.

The **diagnosis of tuberculosis of the larynx** is not difficult if pulmonary phthisis is already known to be present. When attention has been called to it from the onset of hoarseness or from some disturbance in swallowing, we recognize the character and seat of the changes by the aid of the laryngoscope. The diagnosis, however, may present much difficulty in cases where we are not sure that an affection of the lungs co-exists. As has been said, the symptoms at first are not unlike those of a simple catarrh, and the suspicion of the existence of tuberculosis is first aroused from the stubbornness of the disease, the condition of the patient, some inherited taint, the onset of fever, and the remarkable emaciation. With the changes in the larynx which have been described the distinction between tuberculosis and syphilis may be very difficult. In syphilis of the larynx, however, we find that co-existing changes in the pharynx are much commoner than in tuberculosis, and the cicatricial formation which is usually visible furnishes, besides, a very characteristic evidence of syphilis. The diagnosis of tuberculosis of the larynx, however, is made perfectly certain in all doubtful cases by the discovery of tubercle bacilli in the patient's expectoration or in the secretion from the ulcer, which often can be easily obtained by the aid of a fine laryngeal brush. In regard to the laryngoscopic appearances, we may also say that a thick infiltration of the epiglottis with a partial ulceration of the same is an appearance which is almost exclusively confined to tuberculosis. The same is true with regard to a marked projecting infiltration of the inter-arytænoid region.

For the general *treatment* of tuberculosis the reader is referred to the consideration of pulmonary tuberculosis. We shall here discuss merely the local treatment. This is in the milder forms the same as for simple laryngeal catarrh. There is no doubt that even genuine tuberculous ulceration of the larynx may be healed. Nevertheless permanent cures of this sort are exceptional. Of course very much depends upon the general condition of the patient, and upon the co-existing state of the lungs. For local treatment insufflations of iodoform and iodol were for a time strongly recommended, but they are now for the most part abandoned. Of late the best results, comparatively speaking, have been reported from the local employment of lactic acid, in a solution of thirty per cent. to eighty per cent. and preceded by cocaine, and of menthol in a twenty-per-cent. solution in oil. But all these methods of treatment demand considerable dexterity. Inhalations are for the most part merely palliative. We have always obtained the best satisfaction in this regard from inhalations of Peruvian balsam. It is our conviction that for any true advance in the treatment of laryngeal tuberculosis we must look to surgery, and not to endo-laryngeal manipulations, which are always unsatisfactory even in the best hands, but to a complete excision of the diseased tissue by means of laryngotomy. In advanced cases it will usually be found advisable to employ merely palliative treatment.

The constant use of cracked ice, and especially a lavish employment of narcotics, form the best means of lessening the pain and the difficulty in swallowing. Subcutaneous injections of morphine a quarter of an hour before each meal often afford great relief. Besides this, we can paint the larynx with strong solutions of morphine, blow in powdered morphine, or let the patient inhale solutions of morphine or bromide of potassium. Cocaine, which is an excellent local anæsthetic, excels all these, however, in potency (von Anrep). If we paint the ulcerated mucous membrane at the entrance of the larynx with a ten- or twenty-per-cent. solution of cocaine, in a few minutes such an anæsthesia of the affected parts ensues that swallowing may take place without any pain. The following formula may be used:

℞ Cocaini muriatis........................	gr. xv–xxx	(1·0–2·0);	
Alcohol..................................	ʒ ss	(2·0);	
Aquæ destillatæ	ʒ ij	(8·0).	M.

Unfortunately, the action of cocaine is extremely transitory, so that the painting must be repeated over and over again.

CHAPTER VI.

PARALYSES OF THE LARYNGEAL MUSCLES.

1. **Paralyses in the Distribution of the Superior Laryngeal Nerve.**—The superior laryngeal nerve, arising from the vagus, is the sensory nerve for the mucous membrane of the upper portion of the larynx down to the glottis, and also for the mucous membrane of the epiglottis and its neighborhood. Besides this, it also supplies motor fibers to the crico-thyroid muscle. Clinical experience renders it probable that the superior laryngeal nerve also supplies the depressors of the epiglottis, the thyro-epiglottideus, and the arytæno-epiglottidei muscles, and perhaps also the arytænoideus muscle. The last three muscles mentioned, however, perhaps derive some motor fibers from the recurrent nerve also (the inferior laryngeal nerve).

Paralysis of the crico-thyroid muscles and of the depressors of the epiglottis is seen most frequently after recovery from diphtheria. It is usually a part of a more extensive paralysis, and, in addition, is frequently associated with anæsthesia of those parts of the mucous membrane which, as we have seen, derive their sensory fibers from the superior laryngeal nerve (von Ziemssen).

Paralysis of the thyro-epiglottideus and the arytæno-epiglottidei muscles is recognized by the immobility and the erect position of the epiglottis, which is directed toward the back of the tongue.

Paralysis of the crico-thyroid muscles makes the voice rough, and especially renders the production of high tones impossible, since for this purpose we need the action of this muscle as a tensor of the vocal cords. The detection of this paralysis by the laryngoscope is extremely difficult. Its chief signs are a concavity of the edges of the vocal cords, a lack of visible vibration in them, and perhaps, in unilateral paralysis, a higher position of the vocal cord on the sound side.

For paralysis of the arytænoideus muscle, *vide infra*.

2. **Paralyses in the Distribution of the Inferior Laryngeal or Recurrent Nerve.**—The recurrent nerve supplies with sensory fibers the mucous membrane of the inferior cavity of the larynx below the glottis, and it is the motor nerve for all the laryngeal muscles except the crico-thyroid, and except possibly the depressors of the epiglottis (*vide supra*). The muscles innervated by it are arranged according to their function in the three following groups:

a. The openers of the glottis—the posterior crico-arytænoid muscles alone.

b. The closers of the glottis—the lateral crico-arytænoids and the arytænoideus (transverse and oblique).

c. The tensors of the vocal cords—the thyro-arytænoids, which act usually as closers of the glottis, but which very often produce the fine differences in tension in the vocal cords which are necessary in singing and in modulations of speech. They accordingly have the same task as the coarser-working crico-thyroid muscles, which are innervated by the superior laryngeal nerve.

The motor fibers for all these muscles have their special origin in the accessory nerve, from which they pass into the trunk of the vagus, and from this into the laryngeal nerves.

Most of the paralyses of the recurrent nerve are of peripheral origin. Except in the pure muscular pareses (*vide supra*), which arise not infrequently in the course of other laryngeal affections, peripheral paralyses of the vocal cords occur with the greatest relative frequency from an abnormal pressure on the trunk of the recurrent nerve, especially in aneurism of the arch of the aorta, which may cause a left-sided paralysis. Tumors of the bronchial glands, cancer of the œsophagus, thyroid or mediastinal tumors, and, in rare cases, even large pericardial effusions, may also cause a paralysis of the recurrent on one side. Paralyses on the right side are seen quite frequently in contractions at the apex of the right lung and in the rare cases of aneurism of the subclavian artery. The paralyses of the laryngeal muscles, which are sometimes met with after recovery from diphtheria (*q. v.*), also belong to the peripheral paralyses of the recurrent nerve, and their cause is to be found in a degeneration of the branches of the affected nerves. In other cases the paralysis of the recurrent nerve is due to an affection of its fibers in the vagus or even in the accessorius. Excluding certain injuries from operations, these affections are usually due to new growths which cause a paralysis of conduction. Paralyses of the recurrent nerve also arise from affections of the nucleus of the accessory nerve in diseases of the medulla, in the different forms of acute bulbar paralysis, in chronic bulbar paralysis, in multiple sclerosis, etc. The frequent hysterical paralyses in the distribution of the recurrent nerve are to be regarded as cerebral. Finally, paralyses of the laryngeal muscles are sometimes observed for which we are not in a position to find any cause.

1. **Complete Paralysis of the Recurrent Nerve.**—Paralysis of all the laryngeal muscles supplied by the recurrent nerve occurs quite frequently in the pressure paralysis of the trunk of the recurrent, or of its fibers in the vagus. With the laryngoscope (see Fig. 14) we find the vocal cord on the paralyzed side in a middle position, often falsely called a "cadaveric position," and completely motionless on respiration, and also on phonation. On phonating as strongly as possible, the vocal cord on the sound side passes beyond the median line, the arytænoid cartilage also crosses the line, and consequently the glottis is put in an oblique position. The other symptoms are sometimes so slight that without a laryngoscopic examination we do not even think of a paralysis. The speech, however, is usually not pure; it often breaks into a falsetto, and the patient is easily tired by speaking. With bilateral paralysis of the recurrent nerve, which is very rare, we find both vocal cords motionless in a middle position. Complete aphonia exists, and it is impossible to cough, since in coughing we have to make at first a complete closure of the glottis. There is no dyspnœa, however, if the patient keeps quiet.

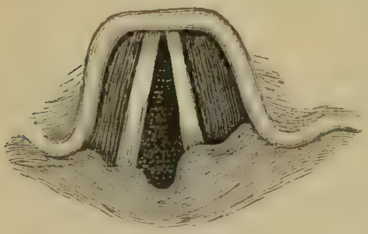

FIG. 14.—(From ZIEMSSEN.) Position on inspiration in paralysis of the left vocal cord, or paralysis of conduction in the recurrent nerve.

2. **Paralysis of the Dilators of the Glottis, the Posterior Crico-arytænoid Muscles.**—Bilateral paralysis of these muscles is a very rare phenomenon, but clinically it is of the utmost importance, since it results in a condition of most marked inspiratory dyspnœa. This condition develops gradually, and usually without any cause that has been satisfactorily determined. There is probably some affection of the nerves themselves which finally leads to the paralysis.* In most cases the disease lasts for years. The dyspnœa may increase, especially from external causes, to severe attacks of suffocation, and tracheotomy is frequently

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* It is remarkable, however, that a purely mechanical hindrance to the dilatation of the glottis may occur from the formation of anchylosis in the crico-arytænoid articulation.

necessary. In paralysis of the dilators of the glottis the respiration is so changed that inspiration only is difficult, protracted, and noisy, while expiration is free and unhindered. This depends on a valve-like action of the vocal cords. They are drawn together by the dilatation of the thorax on inspiration, while the current of air in expiration easily pushes them aside. Phonation is usually entirely undisturbed. With the laryngoscope (see Fig. 15) we find the glottis changed to a small slit, which grows narrower instead of wider on inspiration.

The prognosis is usually unfavorable. Only in the hysterical can these apparently severe conditions appear and disappear again in a short time.

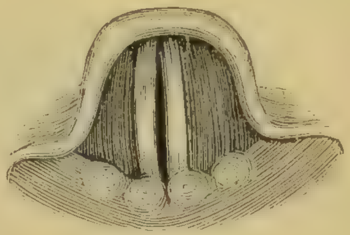


FIG 15.—(From ZIEMSSEN.)—Complete bilateral paralysis of the posticus at the moment of inspiration.

3. **Paralysis of the Thyro-arytænoid Muscles.**—The paralysis or paresis of these muscles, which run into the vocal cords, and which are their chief tensors, is one of the most frequent of the paralyses of the laryngeal mucous membrane, and is often the chief cause of the accompanying hoarseness. It also frequently develops as the result of an habitual over-exertion of the voice in singers and public speakers, and it is one of the commonest causes of hysterical aphonia.

Paralysis of the thyro-arytænoid muscles may be bilateral or unilateral. It is frequently associated with a paresis of the other closers of the glottis, the arytænoidei and the crico-thyroid muscles. With the laryngoscope (see Fig. 16), in the ordinary bilateral paresis of the thyro-arytænoid muscles, we see that on phonation the glottis does not close completely, but that an oval space is left between the vocal cords.

In unilateral paralysis the affected cord shows a concavity of its edge. The voice is always more or less hoarse and low and the speech is strained.

In many cases, after a cure of the original catarrh, a complete recovery from the paralysis may follow by taking good care of the voice. Hysterical paralyses are diagnosticated by their sudden disappearance and reappearance, usually after some psychical disturbance. They are quite common in children of the age of ten to fourteen years, especially in girls. (See the chapter on hysteria.)

4. **Paralysis of the arytænoideus muscle** is rarely an isolated phenomenon. It is sometimes seen in laryngeal catarrh or in hysterical aphonia. The voice is quite hoarse, and with the laryngoscope (see Fig. 17) we find on phonation that the whole anterior part of the vocal cords closes well, but that the cartilaginous glottis remains open as a triangular gap on account of the imperfect motion of the arytænoid cartilages toward each other. When the thyro-arytænoids are paralyzed with the arytænoideus, the glottis shows on phonation a narrow hour-glass opening (see Fig. 18). Both the anterior and the posterior portions of the glottis fail to close, while the vocal processes take their usual median position on phonation from the normal turning of the arytænoid cartilages inward by the action of the lateral crico-arytænoid muscles.

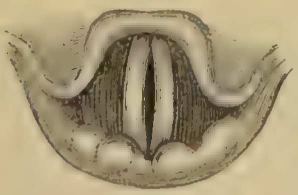


FIG. 16.—(From ZIEMSSEN.)—Paralysis of both internal thyro-arytænoid muscles in the course of an acute laryngitis.

5. **Paralysis of the lateral crico-arytænoid muscles,** as an uncomplicated condition, has never been observed with certainty. Some cases of a complete and simultaneous paralysis of all the closers of the glottis have been described, however, in which the vocal cords are immovable laterally and the glottis remains abnormally wide open.

We may expect success from the **treatment** of paralysis of the vocal cords only when the primary disease is capable of cure. If catarrhal or other diseases of the larynx co-exist, we must first treat these by the methods already mentioned. Paralysis from the compression of tumors, etc., may be relieved in rare cases by extirpation, or by partial resolution of the tumors when of strumous origin. In catarrhal, diphtheritic, and the so-called "rheumatic" pareses—that is, those which occur without any assignable cause—and also in all hysterical aphonias, electricity often works very well. A very rapid recovery sometimes occurs in hysterical paralyses, but it is not always permanent. We commonly employ external faradization of the neck or galvanization through the larynx, combined with frequent changes of the current. Ziemssen has made electrodes for the endolaryngeal irritation of single muscles. Internally we may prescribe preparations of iron and small doses of quinine, especially in anæmic patients. Subcutaneous injections of strychnine are also of advantage, in doses of gr. $\frac{1}{20}$ to gr. $\frac{1}{6}$ daily (grm. 0·003 to 0·01). Methodical efforts at speaking and breathing are of great service in hysterical aphonia.

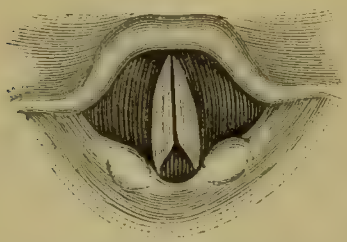


Fig. 17.—(From Ziemssen.) Paralysis of the arytænoideus in acute laryngitis.

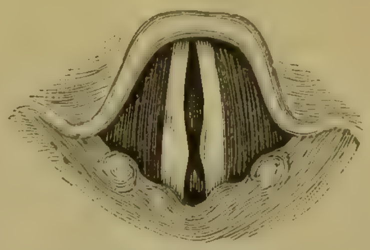


Fig. 18.—(From Ziemssen.) Bilateral paralysis of the thyro-arytænoids combined with paresis of the arytænoideus.

CHAPTER VII.

SPASM OF THE GLOTTIS.

(Millar's Asthma. Thymic Asthma.)

Ætiology.—Spasm of the glottis is a disease which occurs almost exclusively in children under three years of age, and which consists of attacks of spasmodic closure of the glottis, and consequently of most severe dyspnœa. Boys are more frequently attacked by this disease than girls, but the cause of this is wholly unknown. The old name of thymic asthma arose from the idea that the attacks were due to an increase in the size of the thymus gland, but this opinion is wholly unfounded. The relation between spasm of the glottis and rachitis is remarkable, but it is unexplained. Nearly two thirds of all the children who suffer from spasm of the glottis are rachitic, but the opinion which was once held that spasm of the glottis has a special relation to the rachitic craniotabes is not clearly proved. The fact that it is often combined with eclampsia, in that the attacks of spasm of the glottis are aggravated by eclamptic attacks, and that the two alternate with each other, is an argument in favor of a central origin for the disease. In the cases which come on, as they often do, at the time of dentition, we think it possible to assume a reflex origin for the spasm, just as we may in those cases which seem to follow a laryngitis due to taking cold.

Symptomatology.—The single attacks usually come on suddenly by day or by night, either without any cause or from some external influence, like crying, swallowing fluid, or some psychical disturbance. They usually begin with a deep inspiration, followed by complete cessation of respiration. The child becomes pale, cyanotic, looks anxiously about, rolls his eyes, and makes strained and labored efforts at respiration. In severe cases there is a temporary loss of consciousness, and tonic and clonic spasms in the muscles of the extremities and the trunk, as has been mentioned. The attack lasts from some seconds up to two minutes. In very severe cases the attack may be immediately fatal. As a rule, however, the spasm passes off, deep, noisy inspirations follow, and in a short time the child is completely well. The severity of the attacks varies, moreover, in different cases, and it varies very markedly, too, in the same child. Sometimes we have only one attack or a small number of them, while in other cases they may come on ten or twenty times a day, and even oftener, and may last with varying intensity for months. If the child reaches his third year the disease almost always disappears, but quite a large number of the children who suffer from spasm of the glottis die before that age, either in the attack itself or from other affections.

Pure spasm of the glottis hardly ever occurs in adults, but similar attacks are sometimes observed in hysteria.

The **treatment** must be especially directed to the child's general condition. The child is usually pale and emaciated, and if we succeed in improving its nutrition with iron and cod-liver oil, the attacks become less frequent, milder, and finally may wholly disappear. The child should also be kept in moderately warm air and guarded from any exposure to cold. Internal remedies to prevent the recurrence of the attacks are very uncertain in their action. We may employ bromide of potassium, ten to thirty grains daily (grm. 0·5–2·0); musk, ten drops of the tincture every hour or two; oxide of zinc, etc.

In the attack itself the child must be raised up. The face should be sprinkled with water, or, if the attack be of long duration, a cool shower-bath should be given. Friction should be applied to the skin, aided by mustard, or a mustard plaster to the chest and calves. If the attacks are very frequent and intense, we must use narcotics, either inhalations of chloroform or subcutaneous injections of morphine, with care, in doses for a child of $\frac{1}{60}$ to $\frac{1}{12}$ of a grain (grm. 0·001 to 0·005).

CHAPTER VIII.

DISTURBANCES OF SENSIBILITY IN THE LARYNX.

DISTURBANCES of sensibility in the laryngeal mucous membrane have been observed especially in the distribution of the superior laryngeal nerve, in the epiglottis, and in the superior cavity of the larynx above the glottis; but in rare cases they are also observed in the lower portion of the larynx, which is supplied with sensory fibers by the recurrent nerve. They are most frequently associated with motor disturbances, particularly with hysterical paralyses, but they are also quite often found in paralyses of diphtheritic origin. Anæsthesia of the larynx is recognized by the lack of sensation which the patient shows when we touch special parts of the larynx with the point of a sound. The choking and coughing reflexes are almost always absent, so that we can touch the whole entrance of the larynx with the finger without causing discomfort.

The absence of the reflexes may sometimes be dangerous, especially in severe diphtheritic and bulbar paralyses, for, as a result of it, small portions of saliva may reach the larynx in swallowing, and fail to be coughed up, but may be drawn down into the lungs, where they set up a bronchitis and a lobular pneumonia. This danger is especially great if at the same time the patient can not cough forcibly, as is frequently the case in imperfect closure of the glottis. Hysterical anæsthesia is the only form where there is no fear of the development of inhalation diseases in the lungs.

An effective prophylaxis against the dangerous condition just described is possible only by feeding patients, who have much weakness in swallowing and coughing, by means of the œsophageal tube.

CHAPTER IX.

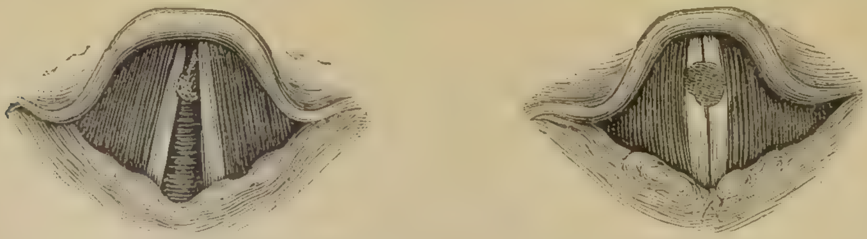
NEW GROWTHS IN THE LARYNX.

SINCE new growths in the larynx are of interest rather to specialists and surgeons, we will here only glance briefly at them. We must remember especially, however, that they can be recognized only by the aid of the laryngoscope. It unfortunately often happens that a patient is treated for a long time without success for a "chronic laryngeal catarrh," until the laryngoscope finally shows that a new growth is the cause of the hoarseness. It is of especial importance, however, to make a diagnosis as early as possible, particularly in carcinoma, since the earlier the operation is done the better is the chance for success (*vide infra*).

A. BENIGNANT NEW GROWTHS IN THE LARYNX.

1. Papilloma is one of the commonest new growths in the larynx. It forms glandular, cauliflower-like excrescences, which are usually situated on the anterior part of the vocal cords, rarely on the false cords. The base of the swelling is broad or pediculated. We do not know the special cause of their origin. They sometimes develop upon an existing chronic catarrh.

2. Fibroma in the larynx is comparatively common. The tumors known as "laryngeal polypi" are usually fibromata. They are generally situated on the vocal cords and form whitish or reddish-brown swellings, from the size of a pea to that of a cherry, and are usually pediculated (see Figs. 19 and 20). People who use their voices very much are especially liable to the formation of fibromata.



FIGS. 19 and 20.—(From ZIEMSSEN.) Pediculated fibromata.

3. Cysts and "mucous polypi" rarely occur. They are probably due to the retention of the secretion in a mucous gland from the stoppage of its orifice. We find them in the ventricles of Morgagni, on the epiglottis, etc.

The symptoms which are excited by benignant tumors in the larynx depend partly upon the situation and partly upon the size of the new growth. Small polypi may exist wholly without symptoms, and are found only by chance on laryngoscopic investigation. Usually, however, the presence of hoarseness, pressure, and itching in the larynx, or respiratory disturbances, when the tumor is a large one, are the symptoms which give occasion for an examination.

B. MALIGNANT NEW GROWTHS. CARCINOMA OF THE LARYNX.

Carcinomata develop usually in old people, either primarily in the larynx or secondarily from affection of the neighboring organs. In the first case the vocal cords or the ventricles of Morgagni are the points most frequently attacked. An extension of the disease to the larynx is seen especially in cancer of the tongue or pharynx, rarely in cancer of the œsophagus.

The symptoms of cancer of the larynx develop slowly. Hoarseness, disturbance in swallowing, pains in the larynx often shooting up into one ear, the appearance of respiratory symptoms, and finally the signs of general weakness and emaciation which are seen in almost all forms of carcinoma, form the picture of the disease. The diagnosis is possible only by the aid of the laryngoscope. Besides this, a digital examination may at times be of diagnostic value by the detection of the characteristic hardness about the entrance or in the neighborhood of the larynx. A general description of the laryngoscopic appearances can not be given on account of the diverse character of the cases. We see the uneven, injected new growth, covered with mucus and often ulcerated, and besides this at times the secondary appearances of catarrh, a developing perichondritis, etc. With a little care the diagnosis is usually tolerably easy. It may be difficult, however, at times, to distinguish it from tuberculosis or from syphilis. We may be aided in such cases by the discovery of the tubercle bacilli or by the results of anti-syphilitic treatment. All the other organs of the patient therefore must always be carefully examined.

Surgical **treatment** is the only one for all laryngeal new growths. We must refer to the special works for all the details. Laryngologists have devised numerous instruments for the removal of benignant polypi, by which, under the guidance of the laryngoscope, the new growth is cut, snared, squeezed, or torn off. The performance of the operation is made much easier by the advantage of the local anæsthesia of the laryngeal mucous membrane due to painting with cocaine (see p. 138). Nevertheless we firmly believe, about these growths as well as about tuberculosis of the larynx, that the "endolaryngeal" operations should be more and more superseded by laryngotomy.—Carcinoma of the larynx can be cured only through removal of the tumor by splitting the larynx or by its total extirpation. The former operation is comparatively free from danger, while total extirpation has met with success as yet in but few instances. If surgical interference is no longer practicable, we can only endeavor to mitigate the suffering of the patient by means of morphine, cocaine, and other narcotics.

SECTION III.

Diseases of the Trachea and the Bronchi.

CHAPTER I.

ACUTE CATARRH OF THE TRACHEA AND THE BRONCHI.

(*Tracheitis and Acute Catarrhal Bronchitis.*)

Ætiology.—Acute catarrh of the larger air-passages, of the trachea, and larger bronchi, is a frequent disease, and it may often arise from taking cold. It is conceivable that the inhalation of cold, damp air sometimes directly affects the mucous membrane of the upper air-passages. Bronchial catarrh is very often associated with a coincident catarrh of the larynx, and more rarely of the pharynx. In the ordinary mild forms the catarrh is usually confined to the trachea and the first large branches of the bronchi, while the finer bronchi remain healthy.

More intense inflammation of the bronchial mucous membrane is the result of active mechanical or chemical irritation. A severe bronchitis develops after the inhalation of noxious gases, nitrous and sulphurous oxides, chlorine, bromine, etc., as is often observed in operatives. The inhalation of smoke and dust, especially vegetable dust, works in the same injurious fashion, and the followers of many trades and employments, like millers, colliers, etc., are especially subject to disease from this cause. In this form of bronchitis the catarrh often extends to the finer bronchi.

The bronchitis which develops in the course of other acute and chronic diseases is still commoner than the primary forms already mentioned. It is often due to infectious causes, like certain infectious diseases, especially measles, whooping-cough, and influenza. In these diseases bronchitis is one of the most constant local affections, and is probably immediately dependent upon the primary infection. Bronchitis, however, develops secondarily in most of the other acute infectious diseases, and is largely due to the inhalation of noxious substances from the upper part of the air-passages. This is the explanation of the bronchitis in diphtheritic processes in the pharynx and larynx, in so far as it does not depend upon a direct extension of the disease, and also of the bronchitis in small-pox, etc. Bronchitis may also be met with in all other forms of severe disease, because retention of secretion, inflammation, thrush, etc., arise in the cavity of the mouth and pharynx, and from them chemical or organic irritants may easily be inhaled into the bronchi. The imperfect expectoration in all severe diseases is a still more harmful factor than this inhalation. The secretion remains in the bronchi, processes of decomposition arise in the stagnating mucus, bacteria collect and lead to a bronchitis, and finally to a lobular pneumonia which is so often found (*vide infra*). The swallowing and inhalation of portions of saliva, which easily decompose, is also a frequent cause of secondary bronchitis.

We do not know how far we may claim that infectious agents act as a cause of primary bronchitis, yet it is not improbable that many cases have such an ætiology. It is especially likely that many cases of bronchitis due to "catching cold" really have something infectious about them, and that the preceding exposure to cold has merely lowered the natural powers of resistance, and thus permitted, or at any rate promoted, infection.

Finally, we must mention that an acute bronchitis is sometimes merely an exacerbation of a previous chronic bronchitis.

The predisposition to acute bronchitis varies in different persons. We do not know definitely on what ground such an increased predisposition to bronchial disease rests, nor why we meet with it sometimes in the weak and anæmic, and at other times in the so-called "full-blooded" persons. Bronchitis is more frequent in children and old people than in those in middle life. Most of the cases occur in the spring and autumn.

Symptoms.—Pain in the chest may be present in some cases of simple catarrhal bronchitis, but usually only in a moderate degree. In severe tracheitis patients often have a painful feeling of soreness in the neck and behind the upper part of the sternum, and this is increased on coughing. The mucous membrane of the bronchi, apparently, has no nerve-fibers which are sensitive to pain, and the pains in the chest which are often present in bronchitis are, as a rule, muscular pains in the intercostal muscles, due to the severe paroxysms of coughing.

Cough is one of the most constant symptoms of bronchitis, and by it usually the attention of the patient or of the physician is first called to the existing thoracic affection. The cough may of course be due to a laryngitis, if that is also present. There is no doubt, however, but that a cough may be excited in a reflex manner from the mucous membrane of the trachea and of the larger as well as of the finer bronchi. Experiments have shown that the point of bifurcation of the trachea is especially irritable, and many severe paroxysms of coughing may be due to an irritation of this very spot from the accumulation of secretion. The intensity of the cough, moreover, is very different in individual cases, which is due in part to the degree and extent of the bronchitis and in part to the reflex irritability of the person affected.

The expectoration consists of the secretion from the inflamed mucous membrane. Its abundance and consistency vary very much in the different cases. We distinguish a catarrh with an abundant secretion, and the so-called "dry catarrh." In the latter only a little viscid sputum is expectorated, but in the former the expectoration is more abundant and muco-purulent. Very often in the beginning of the disease the expectoration is scanty and viscid—the *sputum crudum* of the old physicians; and later it becomes more abundant, more fluid, and more purulent—the *sputum coctum*. In catarrh of the finer bronchi the expectoration may contain little mucous or muco-purulent casts of the bronchi. A simple catarrhal expectoration shows nothing peculiar under the microscope. The pus-corpuscles are often swollen, and show more or less marked fatty degeneration. A slight admixture of blood may occasionally be present in severe bronchitis, but it usually has no special significance, being at times merely the result of severe fits of coughing. A more marked and persistent admixture of blood is seen in the catarrhal sputum in some cases of intense bronchitis in drunkards, so that we may even speak of a "hæmorrhagic bronchitis."

Dyspnœa is usually entirely absent in simple bronchitis, but marked shortness of breath may be noticed in extensive catarrh of the finer bronchi.

Physical Examination.—We may obtain direct evidence of the condition of the tracheal mucous membrane, with due practice, by the laryngoscope. We see a reddening of the membrane, and sometimes an abnormal abundance of secretion on it, if there is a tracheitis. Other methods of physical examination are at our service for judging of the changes in the bronchi.

Inspection of the thorax shows nothing abnormal in the milder forms of bronchitis. The respiration is somewhat accelerated and the expiration prolonged in severe bronchitis, especially if the finer bronchi are affected. Percussion in uncomplicated bronchitis shows nothing abnormal in the pulmonary resonance. Auscultation, too, shows nothing unusual in many cases of mild catarrh limited to the trachea and large bronchi, but in the cases where the smaller bronchi are the seat of the catarrh and there is a marked accumulation of secretion in them, we hear, besides the vesicular respiration, the so-called rhonchi which almost wholly hide it. In dry bronchitis we speak of humming or buzzing sounds, sonorous rhonchi, or shrill, whistling sounds, sibilant rhonchi, according to their pitch. These sounds are probably due to stenosis, and are caused by the passage of the air through narrow portions of the bronchi. The narrowing occurs in part from the swelling of the mucous membrane, in part from the accumulation of secretion. The masses of secretion themselves, if they are set in vibration at the same time, may possibly take part in the production of the humming noises. If the amount of secretion collected in the bronchi is more abundant and of a more fluid consistency, it gives rise to "moist râles" on the passage of the air. These are distinguished as "medium" or "small moist râles," according as they occur in the larger or smaller bronchi.

Other symptoms of disease are often present besides those already mentioned as being directly due to the bronchitis. The general health is usually disturbed in a severe bronchial catarrh. The patient does not feel well, and has less appetite than usual. A moderate amount of fever is often present, especially toward evening. An increase of temperature above 102° or 103° (39° C.) is rarely seen except in children. The patient sometimes complains of headache, which is increased by severe coughing.

The separate forms of bronchitis are distinguished chiefly by the degree of extension of the catarrh.

1. **The Milder Forms of Acute Bronchitis.**—In most cases of simple primary bronchitis, as well as in many milder attacks of secondary bronchitis, the catarrh is limited to the mucous membrane of the larger bronchi. Exposure to cold and other injurious influences are frequent causes of the primary form. The symptoms are moderate. The cough, however, may be quite troublesome. Often fever is absent or but slight. Upon auscultation, particularly over the lower lobes, but sometimes over the entire lungs, and usually with some symmetry of distribution, are heard numerous rather coarse wheezing or rattling sounds; but in many cases, as we have said, there may be nothing abnormal heard, so that the diagnosis will have to rest merely upon the subjective discomfort in the chest, the cough, and the expectoration. With proper care, simple primary bronchitis runs its course in a few days, or at the most in a few weeks, and ends in complete recovery. If the patient exposes himself recklessly, or the ætiological factors continue to be active, the disease may however prove to be very tedious, and finally develop into chronic bronchitis.

2. **The Severer Febrile Forms of Acute Bronchitis.**—Sometimes acute bronchitis assumes a severer form, whether because the influences which give rise to it are unusually violent, or because it is due to some special and as yet little known cause (perhaps infectious). In such cases the symptoms are more marked, the bronchial râles more abundant, the general condition of the patient worse. Not infrequently there is fever for several days, or even for one or two weeks, the type being irregularly remittent, but the temperature seldom exceeds 102° or 103° (39° to 39·5° C.). The author has not infrequently noticed that there is more liability in the severer forms of acute bronchitis to have the disease mainly limited to one lobe, or at least to one lung, but the disease may be diffuse. This form of the disease also has a favorable prognosis except in feeble or elderly persons.

3. **Catarrh of the Finer Bronchi—Capillary Bronchitis.**—A simple primary bronchial catarrh rarely extends to the finer bronchi in adults. The secondary bronchitis, however, which develops in other severe diseases (*vide supra*), often extends into the ultimate divisions of the bronchi, and finally leads to the formation of nodules of lobular pneumonia—"catarrhal pneumonia" (*vide infra*). We recognize the implication of the finer bronchi by hearing the high, shrill, whist-

ling rhonchi [sibilant rhonchi], or the abundant small, moist râles. Respiratory symptoms may be quite marked in extensive catarrh of the finer bronchi. Respiration is evidently accelerated, and expiration is usually prolonged. There is often quite a severe cough. The expectoration is muco-purulent and usually not very abundant.

Capillary bronchitis in children is of great practical importance. Every bronchitis in young children has, as experience tells us, a tendency to attack the smaller bronchi. Extensive bronchitis is seen especially in weak children who are rachitic or predisposed to tuberculosis. Children have an especial predisposition to be attacked with bronchitis at the time of the first dentition, but it is also seen at an even earlier age.

The parents' attention is usually called to the disease by the appearance of a cough, which is excited especially by the child's crying. Small children never expectorate, for they swallow the secretion which is coughed up into the pharynx. The rapidity of respiration is very striking, it being increased to sixty or eighty, or even more, in a minute. The respiration is also labored, but it is usually superficial, and in severe cases irregular. There is generally a distinct respiratory play of the alæ nasi. We often see a retraction of the lower lateral portions of the thorax on inspiration as a result of the imperfect entrance of air into the smaller bronchi. The expiration is frequently noisy and groaning in children. We hear extensive small, moist râles over the lungs. In severe cases the child becomes restless, anxious, perhaps markedly pale and cyanotic, and finally apathetic and stupid. In such cases, however, we have no longer to deal with simple bronchitis, but catarrhal pneumonia has already developed. The disease almost always runs its course with fever, the temperature rising to 104° (40° C.) and over. The pulse is increased to 120 or 140 or more per minute. The duration of the disease is seldom less than two or three weeks, and it may last much longer. Death may ensue, especially in ill-nourished children, partly as a result of general weakness, and also directly from the imperfect respiration. In such cases we find at the autopsy not only diffuse bronchitis, but also almost always lobular pneumonia. In many cases a gradual recovery finally takes place in spite of the most severe symptoms.

The secondary bronchitis in children complicating measles, whooping-cough, diphtheria, etc., has the same tendency to involve the finer bronchi and to lead to lobular pneumonia.

In conclusion, we must mention that acute bronchitis in old people also readily attacks the finer bronchi, and may be dangerous partly from the general exhaustion, partly from the occurrence of respiratory symptoms, as in lobular pneumonia.

Diagnosis.—The diagnosis of bronchitis presents no special difficulty. It is obtained directly by the discovery of rhonchi on auscultation. If these fail, we conclude that there is a mild catarrh of the larger bronchi from the presence of cough and expectoration, if no cause for the cough is to be found in an affection of the larynx. The question is more difficult, but it must always be considered, whether a given bronchitis is a common primary catarrh or secondary to some other affection. This question naturally can be decided only by a very careful examination of the body. We must always remember, furthermore, that severe pulmonary affections may be at first quite latent and show objectively merely the signs of simple bronchitis, while later pneumonia, a tubercular affection, or something similar, develops. A bronchitis which is unilateral, or in which the signs are to be found in circumscribed localities, must therefore be regarded as suspicious. It has long been known that bronchitis in the apices of the lungs, the "apex-catarrh," is often the first objective change to be met with in pulmonary phthisis. We can only conjecture, and not pronounce with certainty on objective evidence, whether nodules of lobular pneumonia are present or not in diffuse bronchitis affecting the finer bronchi.

From what has been said, it is clear that we should be cautious in our prognosis regarding every severe bronchitis, especially in children and old people. The prognosis in the milder forms of bronchitis is of course always very favorable.

Treatment.—The prophylaxis of primary bronchial catarrh consists in the removal of all the injurious influences mentioned which, as experience shows, may give rise to a bronchitis. A careful hardening of the skin to the effects of a change in temperature is of service in persons, particularly children, who have a special tendency to bronchitis, as we have already said in regard to the prophylaxis of laryngitis. It is very important to remember, in this connection, that we can also be successful in our prophylactic measures against secondary bronchitis. Keeping the mouth and pharynx clean, urging deep inspirations, and aiding expectoration by the timely use of tepid baths and shower-baths, may often prevent a bronchitis or keep it within bounds, while it would surely develop if the patient were neglected.

Simple hygienic measures suffice in the treatment of mild cases of acute bronchitis. The patient should be kept warm, should remain in his room, or, if there be any fever, in bed. Diaphoretic remedies have long been praised as especially potent in the treatment of acute bronchial catarrh. The patient, therefore, should drink hot tea, pectoral tea* (Brustthee), or elder tea, etc., or hot milk mixed with Seltzer, a remedy whose efficacy is frequently praised by the patient. Local treatment of the mucous membrane by inhalations is usually illusory, for only the smallest part of the inhaled fluid reaches the bronchi. We may, however, always prescribe inhalations of warm steam, or a one- or two-per-cent. solution of common salt, especially with a dry cough and a secretion which is hard to loosen.

Otherwise we must employ symptomatic treatment. A mustard plaster or a cold, wet compress about the chest does good service with severe subjective thoracic symptoms. In bad cases a few dry cups may be very useful in adults, but local abstractions of blood are never necessary in simple bronchitis. If there is troublesome irritation on coughing, so as to disturb the rest, we may prescribe small doses of morphine, five to ten grains of Dover's powder (grm. 0·3–0·5), fifteen to twenty drops of cherry-laurel water, codeine, etc. When expectoration is difficult, we may use the so-called expectorants—ipecac, chloride of ammonium, apomorphine, etc.

We have already repeatedly mentioned the use of tepid baths and shower-baths, indicated in severe diffuse bronchitis developing secondarily in the course of other acute diseases.

Tepid baths with shower-baths, two or three times a day, are also to be used as a most powerful remedy in severe cases of capillary bronchitis in children. The baths assist expectoration and guard against the possibility of the development of lobular pneumonia. Wet packs applied to the thorax or over the whole body are serviceable. Children are wrapped to the neck in a sheet which has been previously dipped in water at a temperature varying with the degree of the fever from 68° to 77° (16° to 20° R.) and well wrung out. It is well to leave the arms free. A dry woolen blanket may be wrapped around the moist sheet. This procedure must be repeated three or four times a day. As to other remedies, we use the same as in adults. With weak children our care must be to keep up the strength by furnishing the most nourishing food possible and giving small amounts of wine. An emetic is sometimes indicated in cases with an abundant accumulation of mucous in the bronchi, and is of good service. As experience has shown, we should use opiates for small children only with the greatest care. Senega and benzoin may be used as expectorants.

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* A favorite German household remedy, consisting of an infusion of eight parts of althæa, three parts of licorice, one part of orris-root, four parts of colt's-foot, and two parts each of mullein and aniseseed.—Trans.

In the bronchitis of old people our chief aim should be to keep up and improve the patient's strength. We prescribe liquor ammonii anisatus, infusion of senega, etc., to aid expectoration, which is usually difficult, since the cough is feeble. Tepid baths may be of advantage, but they must be used with care.

CHAPTER II.

CHRONIC BRONCHITIS.

(Chronic Bronchial Catarrh.)

Ætiology.—Chronic bronchial catarrh may develop gradually from external causes, or in rare cases it may follow an acute bronchitis. The same noxious influences which excite an acute bronchitis may, by the frequent repetition of their action, result in a chronic bronchitis.

In a large number of cases severe chronic bronchial catarrh is not an independent disease, but occurs as a complication or a result of other diseased conditions. The combination of chronic bronchitis with emphysema of the lungs (*vide infra*) is the most common. A large number of cases also are the result of some form of heart disease, like valvular disease or myocarditis, or of disease of the vessels, leading to stasis in the pulmonary circulation, and finally to a chronic catarrh of the bronchi. Chronic bronchial catarrh in renal diseases also depends, in part at least, upon circulatory disturbances. Finally, we find a more or less extensive chronic catarrh of the bronchi in other chronic affections of the lungs and pleura, as in tuberculosis or pleurisy.

Chronic bronchitis is seen especially in adults and old people, and more frequently in men than in women.

Pathological Anatomy.—Chronic bronchitis is characterized anatomically by a marked venous hyperæmia of the bronchial mucous membrane. The whole tissue itself is often thickened, and the surface of the membrane is swollen. In old cases, however, we finally meet with an atrophy of all the layers of the mucous membrane. One of the most frequent results of a chronic bronchitis is a cylindrical dilatation of the middle and lesser bronchi—bronchiectasis. This arises gradually from the loss of elasticity of the diseased bronchial walls, increasing their tendency to give way, as well as from the pressure of the stagnating secretions.

Symptoms and Course of the Disease.—The symptoms which are due to chronic bronchitis are disturbances of respiration, cough, and expectoration. To these should be added the results of a physical examination.

The cough is of very different severity in different cases. Usually it is worse early in the morning, in the evening, and at night, than in the daytime. The amount of expectoration is also subject to great variations. In many cases there is a dry cough (*catarrhe sec*, *vide infra*), in which only small amounts of tough, viscid sputum are expectorated. In other cases the expectoration is more abundant and muco-purulent, and sometimes excessive and quite thin. Microscopically, it has no special characteristic appearances, but it contains only the usual elements of sputum—pus-corpuscles mixed with pavement epithelium, often many bacteria, sometimes needles of fat acids, and rarely a few pointed octahedral crystals, the so-called asthma crystals (*vide infra*). Small amounts of blood may be seen in severe chronic bronchitis, but they do not have any bad significance.

Dyspnœa of moderate degree may also be present in uncomplicated and extensive bronchitis. In the cases in which it is severe, however, it is usually due to other conditions affecting the heart or lungs.

Physical Examination.—The percussion in bronchitis shows no special change. At most the resonance may be somewhat tympanitic from the relaxation of the lung-tissue, especially in the lower and posterior portions of the lungs, or, with an abundant retention of secretion in the bronchi, it may be a little diminished. Auscultation may give either rhonchi, whistling, hissing, humming, etc., or moist râles, according to the extent of the catarrh and the amount and consistency of the secretion. The sounds are usually to be heard over the whole lung, or especially over the lower lobes, because here the catarrh is usually most marked, and retention of secretion is most apt to occur. The respiratory murmur in some places may be quite obscured by the râles. Otherwise it is vesicular, sometimes exaggerated, sometimes rough and indefinite. Expiration is usually prolonged. The respiratory murmur may be much diminished, or even entirely suppressed in places where the bronchi are stopped by secretion, which happens most frequently in the lower lobes.

Except in mild cases, we usually distinguish several different forms of chronic bronchial catarrh, which may run into one another.

1. The dry chronic catarrh (*catarrhe sec* of Laennec) is the form in which the mucous membrane has only a very slight secretion. The cough is usually very troublesome and labored, but the patient raises merely a little tough sputum, or none at all. On auscultation we hear sibilant rhonchi, but no moist râles. This form of catarrh is usually associated with pulmonary emphysema, and asthmatic attacks are also frequent. The disease is stubborn, and usually lasts for years.

2. The so-called bronchial blennorrhœa is that form of chronic bronchitis in which we find a very copious secretion from the mucous membrane. The cough is therefore associated with a very abundant and quite thin expectoration, the amount of which in the twenty-four hours may exceed a pint (half a litre). The expectoration runs together in the sputa-cup and usually separates on standing, the more purulent portion sinking to the bottom, and the sero-mucous portion, which is usually frothy on the surface, remaining at the top. Numerous moist râles are heard in the lungs, especially in the lower portions. These diminish if large amounts of sputum are coughed up. Anatomically, the bronchi are almost always found dilated in this form of chronic bronchitis.

3. The so called serous bronchorrhœa ("*catarrhe pituiteux*" of Laennec) is quite rare but very interesting. It is characterized by the expectoration of a very large amount of frothy, purely serous, thin sputum. The cough usually comes on in very violent paroxysms which last from half an hour to an hour or more. The respiratory symptoms are quite severe, especially during these attacks, and have given rise to the old and useful term "*asthma humidum.*" The expectoration collected in twenty-four hours may amount to one or two quarts (litres). Examination of the lungs usually gives very abundant and extensive moist râles. The resonance on percussion is normal or a little diminished, from the accumulation of secretion.

The special cause of this peculiar disease is quite obscure. It is either an independent, very chronic trouble, which may last for years with a varying course, or it may occur secondarily in other affections, especially in chronic contraction of the kidney. We once saw a very severe case of the independent and apparently quite uncomplicated form in a young woman who had high fever at times, and who became much broken down physically.

Course of the Disease.—The course of most chronic bronchial catarrhs is very protracted. The disease usually has frequent remissions and fresh exacerbations. The patient is tolerably well in the pleasanter time of the year if he takes good care of himself, but in autumn and winter, or after exposure to various noxious influences, the catarrh grows worse and the patient's symptoms increase. If the disease has lasted for years, we usually find symptoms in the lungs, like emphysema or chronic tuberculosis, or in the heart, like secondary dilatation and hypertrophy of the right ventricle, which symptoms gradually become more severe. The details of these conditions are to be found in the appropriate sections.

Diagnosis.—The diagnosis of chronic bronchitis is not difficult in itself, and may easily be made by considering the patient's symptoms and by judging of the result of the physical examination. We must always consider, however, whether the bronchitis is not a result or a complication of some other chronic disease. Therefore in every case of chronic bronchitis the heart and the urine must be carefully examined, as well as the lungs.

Prognosis.—Chronic bronchitis is in most cases a very stubborn affection, which frequently shows improvement, but from which complete recovery is rare. The prognosis also depends greatly upon the patient's circumstances, and upon the possibility of his taking care of himself and avoiding all harmful exposure. In secondary bronchitis the question whether the bronchitis is capable of material improvement or not of course depends mainly upon the prognosis of the primary disease.

The danger in primary chronic bronchitis comes from the final development of its sequelæ, especially from the gradual appearance of pulmonary emphysema, dilatation of the heart, etc.

Treatment.—The only hope of success in severe cases in any method of treating chronic bronchitis is in removing the patient completely, at least for a time, from the action of injurious influences. The favorable result of the baths and health resorts that are employed depends largely upon this, that patients enjoy in them complete bodily rest, and are far better protected from dust and the changes in the weather than at home. We must make the patient comprehend the necessity of this condition as the basis of any treatment. If he can not go to a suitable climate during the cold season, he must keep his room in all unpleasant weather, but at other times he may be permitted to stay in the open air. Furthermore, the patient must be warned to avoid as completely as possible those harmful influences which his calling and manner of life entail, and among which especially is the bad air in our inns and restaurants. Food should be easily digestible, and, in people inclined to corpulence, sparingly taken. Alcohol is to be permitted only in a moderate degree. We combat the tendency to constipation, which is often present, by dietetic remedies, by taking fruit, especially grapes, prunes, etc., honey, Graham bread, or by mild laxatives, like the bitter waters, Friedrichshall, Ofner, etc.

If the circumstances of the patient permit and his condition requires it, we should send him south in the autumn in order to avoid the evils of a northern winter. The rule is to send patients with a bronchial catarrh when there is much secretion to health-resorts with a dry climate—for example, to the western Riviera, San Remo, Bordighera, Mentone, Cannes, etc. The somewhat dry yet cooler climate of Meran, Gries, or Arco is suitable for patients with a stronger constitution. Patients with dry bronchitis usually find themselves at their best in a warm but not too dry climate. If we wish to be sure of avoiding the winter's cold, we must choose Sicily, Egypt, or Madeira for a residence. Of the more northern winter resorts we may mention here places on the eastern Riviera, Nervii, Spezzia, etc., except Venice, Pisa, or Rome.

We must recommend especially, in bronchitis, a suitable summer residence outside of large and dusty cities. Any private country residence in a well-wooded and protected place is of advantage. If we wish to send patients to a bath, Marienbad, Kissingen, or Homburg are proper places for corpulent people who also suffer from digestive disturbances, while we may send weaker patients to Ems, Soden, Badenweiler, Ischl, or Reichenhall. Milk cures, whey cures, and grape cures are also prescribed in many cases of chronic bronchitis, the milk cure in particular for weak and anæmic individuals. A summer residence on the sea, best on the Baltic, is very serviceable for many patients with bronchitis.

The inhalation treatment is much employed in chronic bronchitis, but we should not cherish too high hopes about its use. The best inhalations in dry catarrhs are simple steam, a two-per-cent. solution of common salt or bicarbonate of sodium, Ems water, etc. In cases with marked secretion, inhalations of oil of turpentine are most to be praised. The simplest way is to pour a teaspoonful of oil of turpentine into hot water and inhale the vapor as it arises. The so-called turpentine-pipe, however, is more convenient and more efficacious. This consists of a flask, which is filled to the height of several inches with water and then with a layer of oil of turpentine or of *oleum pini pumilionis* (P. G.), some two centimetres thick. Two glass tubes, open at both ends, are passed through the cork. One, straight, tube extends down into the layer of water; the lower end of the other is free in the upper part of the flask. The outer portion of this last tube is long enough to be bent at an angle and forms the mouth-piece of the pipe which the patient sucks. He thus breathes the air which is filled with turpentine vapor. We have treated many patients in this way, who, for a number of hours a day, "smoked" their turpentine-pipes.

In treating chronic bronchitis the pneumatic method* was considerably employed for a time; that is, the patient was made to breathe artificially compressed air, or to expire into air of a less than atmospheric tension, by means of a movable pneumatic apparatus, as proposed by Waldenburg and others. Of late, however, this method of treatment has received less favor, inasmuch as actual results have fallen decidedly short of the benefit promised. In Ems, Reichenhall, and other places special pneumatic cabinets have been arranged, filled with compressed air, in which patients remain for varying lengths of time.

The different alkaline mineral waters—Seltzer, Ems, Victoria, etc.—are next to be mentioned among internal remedies, which may also be used efficaciously at home. The numerous expectorants, like ipecac and apomorphine, are especially valuable in dry bronchitis. In bronchial blennorrhœa we know empirically that the internal use of balsams causes a distinct diminution of the secretion. Oil of turpentine is the most active, and may be given internally in gelatine capsules, two or three capsules a day, or mixed with milk, in doses of ten or fifteen drops two or three times a day. Lépine, G. Sée, and other French physicians recommend terpine as still more effectual. This is a derivative of turpentine, and is best employed in pills containing one and a half grains (0·1), of which two or even more are to be taken three times a day. It may also be given in solution as follows: Terpine, 2½ drachms (grm. 10); alcohol, q. s. ad sol. faciundam; aq. dest., 6 ounces, 6 drachms (200 grm). Misce. Sig: Two or three tablespoonfuls daily. Balsam of copaiba and balsam of Peru are also used internally. We should be very sparing of narcotics at first, but in severe cases we can not wholly dispense with them.

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* Details of the pneumatic treatment may be found in the following works: R. v. Vivenot, Jr., "Zur Kenntniss der physiologischen Wirkungen und der therapeutischen Anwendung der verdichteten Luft," Erlangen, 1868. Waldenburg, "Die pneumatische Behandlung der Respirations- und Circulationskrankheiten," Berlin, 1880. Knauthe, "Handbuch der pneumatischen Therapie," Leipzig, Wigand, 1876. Schnitzler, "Die pneumatische Behandlung d. Lungen- u. Herzkrankheiten," Wien, 1877 (40 Seiten). Oertel, "Handbuch d. respiratorischen Therapie" (v. Ziemssen's "Allg. Therapie," ii, 4), Leipzig, Vogel, 1881.

[The iodide of potassium in doses of five to ten grains thrice daily is sometimes distinctly curative. An out-door life, free diet, moderate alcoholic stimulus, tonics, and woolen clothing do much to promote recovery.]

Local applications to the chest in the form of embrocations, mustard plasters, dry cups, or cold wet compresses are to be used, especially with severe dyspnœa, or with pain and a feeling of oppression in the chest. Regular cold sponging of the chest serves to harden and strengthen the patient.

Warm baths are very well borne by many patients with chronic bronchitis. Sometimes, too, vapor baths, if taken with caution, may be of service, especially in strong and corpulent patients.

In all secondary chronic catarrhs our chief attention, beyond the symptomatic treatment of the bronchitis, must be directed to the treatment of the underlying disease. If we succeed in once more regulating the heart's action where there is uncompensated heart disease, or in establishing diuresis where there is renal disease, we may also in that way cause improvement in the existing bronchial catarrh.

CHAPTER III.

FŒTID BRONCHITIS.

(*Putrid Bronchitis.*)

Ætiology.—By putrid or fœtid bronchitis we mean that form of bronchitis in which the secretion of the mucous membrane undergoes a putrid decomposition, and in which, consequently, the expectoration takes on a peculiar and extremely foul odor. The cause of fœtid bronchitis is usually the entrance of the bacteria of putrefaction into the bronchi by means of the inspired air. Only in rare cases does it arise from a pulmonary gangrene of embolic origin (*vide infra*).

The opportunity for the agents of putrefaction to enter the bronchi with the inspired air is of course often given, but a fœtid bronchitis naturally is excited only when they can remain there and increase. Their retention and their further development is chiefly favored, as we know, by diseased conditions which already exist in the bronchi. A great number of cases of fœtid bronchial catarrh therefore develop secondarily upon other pulmonary affections of longer standing. Thus the expectoration may quite suddenly change and take on a fœtid character in the course of a chronic or rarely of an acute bronchitis or of phthisis. Bronchiectasis (*vide infra*) greatly favors the development of this putrid change, for in it the retention and stagnation of large amounts of secretion furnish the aid and occasion for it. If a putrid decomposition of the secretion begins in one part of the bronchial system, the further extension of the process follows from direct infection.

In rare cases putrid bronchitis also develops in lungs which were previously apparently sound—primary fœtid bronchitis.

Symptoms and Course; Anatomical Changes.—If a fœtid bronchitis arises in the course of some other chronic pulmonary disease, its appearance may be marked by a sudden impairment of the general condition, by high fever, often associated with numerous chills, and by an increase of the thoracic symptoms, like pain and cough. The change in the expectoration, the peculiarity of which was first accurately described by Traube, is characteristic. There is a repulsive, sweetish, putrid smell. The expectoration is usually quite abundant; the consistency is rather thin. On standing, the sputum shows a very marked division into three layers. The upper layer consists of a very frothy, muco-purulent stratum, consisting in part of individual masses, from which a number of coarser or finer fibers float down into the middle layer. This middle layer consists of a dirty-green muco-serous fluid. At the bottom of the vessel is found the third layer, which is often the thickest, and is composed entirely of pus. It consists of pus-corpuscles which have sunk to the bottom, and is of a rather thin, greasy consistency. With the naked eye we generally recognize a number of little whitish-gray plugs and particles in it. These so-called "Dittrich's plugs," which are easily crushed under a cover-glass, are quite characteristic. Microscopically, they consist of decomposed pus-corpuscles, detritus, and bacteria, and usually contain very many needles of fat acids arranged in bundles (see Fig. 21).

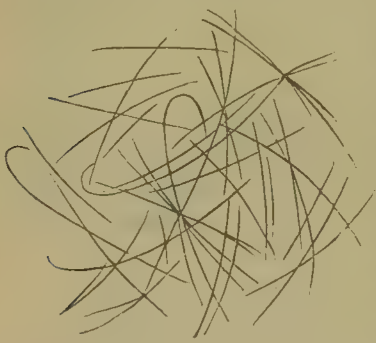
FIG. 21.—Crystals of fat acids.

We often find also in the sputum large masses of fungi, especially great bunches of twisted leptothrix fibers, which, by an unpracticed eye, may readily be mistaken for elastic fibers. The latter are, of course, never found in the expectoration of a simple fœtid bronchitis, but only in the deep-seated, destructive processes in the lung, like gangrene. On chemical examination of the sputum. the ordinary products of putrefaction may be found—volatile fat acids, especially butyric and valerianic acids. also sulphuretted hydrogen, leucine, tyrosine, etc.

The breath of the patient, as well as the sputum, is very often foul-smelling, and so offensive that he becomes a burden to his associates.

The signs which fœtid bronchitis gives on physical examination are those of an ordinary bronchitis. In a great number of cases we also find signs of consolidation and contraction of the lung, of pleurisy. etc., which do not belong to fœtid bronchitis as such, but are due to complications or sequelæ.

The most frequent of these sequelæ is the development of a "reactive" lobular inflammation, a pure pneumonia, which follows a catarrh which has attacked the finer bronchi. These pneumonias frequently run into gangrene, so that we very often find a number of larger or smaller nodules of pure gangrene besides the extensive fœtid bronchitis in the lungs. In many of these cases the fœtid bronchitis is certainly the primary process, and the development of the nodules of gangrene is secondary; yet we shall see later that the reverse may also be true. Fœtid bronchitis and gangrene of the lungs run into each other so often, both clinically and anatomically, that there is no sharp line to be drawn between them. If the nodules are superficial, and reach the pleura, the infection attacks this, and we have a purulent or even an ichorous pleurisy.

The smaller and medium-sized bronchi are almost always found in a condition of cylindrical dilatation in old fœtid bronchitis. Their mucous membrane is intensely inflamed, and often ulcerated superficially. On its surface we see in the cadaver the greasy purulent masses and the plugs which we find in the expectoration during life.

Whatever may be the case with the general course of fœtid bronchitis, its beginning is often quite sudden and acute, both in the primary and in the secondary forms, as we have said. The patient is attacked with fever, which may often be quite high, and with a stitch in the side, and cough, and expectoration. Later, the characteristic peculiarities described above appear. The further course of the disease is almost always chronic, lasting for years, but subject to many variations. Very often manifest improvement, and even apparent recovery, takes place, until suddenly there is a new attack of fever and thoracic symptoms. The general condition and nourishment of the patient may be quite good for a long time, except during the periods of marked exacerbation of the disease. Patients with chronic fœtid bronchitis often appear somewhat bloated, but also pale and slightly cyanotic. Peculiar clubbed thickenings of the terminal phalanges of the fingers gradually develop, as in many cases of bronchiectasis. Slight œdema of the lower extremities is also sometimes present.

Symptoms referable to other organs may be wholly absent. We see most frequently disturbances of the stomach, loss of appetite, and nausea, which probably comes from swallowing the fœtid sputum. Patients also complain of occasional rheumatic pains in the muscles and joints, which may perhaps be due to an absorption of septic matter. In conclusion it must be mentioned that in fortunately rare cases of fœtid pulmonary disease pyogenic germs reach the brain by metastasis, and here give rise to purulent meningitis or cerebral abscess.

The danger of the disease, apart from the exceptional occurrence just mentioned, lies in the possible extension of the process to the lungs and the development of pulmonary gangrene and its sequelæ. We hardly ever find a simple fœtid bronchitis in the cadaver, but we almost always see other processes besides, which have been mentioned above—reactive pneumonia, pulmonary gangrene, etc. These processes develop very readily, and make rapid progress in old, decrepit persons, who live under bad external conditions, in whom putrid processes in the lungs are frequent.

The **diagnosis** of fœtid bronchitis is not difficult in itself, for the diagnosis of a putrid process in the lung may be made from the stinking sputum alone. It may be difficult to decide whether we have to do merely with a fœtid bronchitis, or with a pulmonary gangrene also. Decisive indications of gangrene are derived from physical examination—dullness, bronchial respiration, and large, moist râles—and also the discovery of elastic fibers and fragments of parenchyma in the expectoration.

The **prognosis** must be made with care in every case of fœtid bronchitis. If the external circumstances of the patient are favorable, he may remain in tolerable health for years. We must always be prepared for the appearance of new exacerbations of the disease and of affections of the lung itself.

Treatment.—The chief aim of treatment must be to bring the putrid processes in the bronchi to a stand-still by the death of the agents of putrefaction. The difficulty of fulfilling this task lies in the impossibility of getting the disinfecting material to act on the bronchial mucous membrane in the necessary amount and concentration. Nevertheless, we can, without doubt, at least relieve a fœtid bronchitis and keep it in check by the judicious use of inhalations. Inhalations of a two-per-cent. solution of carbolic acid are most useful, given for five or ten minutes several times a day. These, however, are sometimes not well borne if long continued, and they may excite mild symptoms of carbolic poisoning—like headache, malaise, and dark carbolic urine. We have often used with good results the "carbolic mask" recommended by Curschmann, a kind of respirator fastened in front of the nose and mouth, containing cotton in a special receptacle impregnated with carbolic acid, equal parts of carbolic acid and alcohol, or other remedies like turpentine or creasote. Many patients can wear these masks, with occasional interruptions, for many hours a day. Turpentine is most used next to carbolic acid. Turpentine can be used by inhalation and also internally. Likewise terpine and myrtol are often of decided benefit. We have very recently, in the clinique at Erlangen, had good results from inhalations of *oleum pini pumilionis* (employed as described on p. 154 for the inhalation of turpentine). Both inhalations and the internal exhibition of turpentine are of distinct value. We may also try acetate of lead internally, one or two grains (grm. 0·05–0·10) in powder every two hours.

In other respects all the general hygienic and symptomatic methods of treatment recommended for common chronic bronchitis are also useful in fœtid bronchitis. The sputum should be disinfected by putting strong carbolic acid, etc., into the sputa-cup to lessen the bad odor. It is a very good plan to keep the carbolic spray at work in the patient's room as often and as long as possible, or the air may be impregnated with *oleum pini pumilionis.*

CHAPTER IV.

CROUPOUS BRONCHITIS.

(*Fibrinous or Pseudo-membranous Bronchitis.*)

CROUPOUS bronchitis is a peculiar form of disease of the bronchial mucous membrane, of very rare occurrence, in which there is a formation of extensive fibrinous patches in the bronchi. Only that form of croupous bronchitis which occurs primarily in the bronchi is to be considered here, and not the secondary form, which on the one side is associated with diphtheria in the pharynx and larynx, and on the other with croupous pneumonia.

The **ætiology** of the disease is as yet wholly unknown. From analogy with other well-known croupous inflammations of mucous membranes, we must look here for some noxious influence which destroys the epithelium, but up to this time we are entirely ignorant of its character. Individuals in youth and middle age, somewhere between ten and thirty years old, are the chief victims. Men are attacked somewhat more frequently than women. The disease comes on either in persons who were previously healthy—the essential croupous bronchitis—or in those who have already suffered from some other disease, especially some chronic pulmonary affection—the symptomatic, secondary croupous bronchitis. It is not certain whether the last-named cases can have the same ætiological relation as the cases of pure primary fibrinous bronchitis. Fibrinous bronchitis has been observed in the course of typhoid fever.

Symptoms and Course.—Primary fibrinous bronchitis occurs in two forms, acute and chronic. The acute form begins quite suddenly, with fever, cough, pain in the chest, and severe dyspnœa which speedily develops. The fibrinous coagula, which alone render the diagnosis possible, appear in the expectoration either at once, or after the existence for some days of what is apparently simple catarrhal bronchitis.

These coagula form complete casts of the bronchi, and are more or less branching. They are of a whitish color and of quite a dense, elastic consistency. The main stem may be a centimetre thick, and from it the further ramifications branch, dividing dichotomously. The largest casts are ten or fifteen centimetres long. On section, we usually find a free lumen within, and generally recognize a definite laminated structure in the membrane. In many places they are enlarged and swollen. Microscopically, we find white blood-corpuscles in and upon the hyaline ground-substance of the casts, and also red blood-corpuscles, sometimes epithelial cells, and quite often the peculiar pointed octahedral crystals which are also found in the expectoration in bronchial asthma (*vide infra*). The so-called "spirals" (*vide infra*) have also been found in the expectoration of fibrinous bronchitis. Chemically the casts consist of coagulated albumen. Their solubility in alkalies, especially in lime-water, is of therapeutic importance.

On coughing, the patient usually raises a simple mucous or muco-purulent expectoration beside the casts, and in this sputum the casts are imbedded. They are often first discovered by pouring the whole amount of sputum into water, when they unfold and spread out. The expectoration also contains not infrequently a slight admixture of blood.

The subjective symptoms of the patient may be very violent. The dyspnœa sometimes attains a high and alarming degree. It ceases when a large cast is expectorated after a severe paroxysm of coughing. Such attacks may recur every day or two. In other cases, however, the subjective symptoms are comparatively slight.

Physical examination of the lungs gives little that is characteristic. In uncomplicated cases percussion gives nothing abnormal, or at most the signs of an acute emphysema. Auscultation gives the ordinary signs of bronchitis, not characteristic in themselves, such as rhonchi, or moist râles. If a large bronchus is plugged, the respiratory excursions and the respiratory murmur are almost entirely absent in the corresponding portion of the lung, but after the expectoration of a cast the murmur once more becomes audible.

The duration of acute cases is sometimes only a few days, at most a few weeks. In favorable cases the fever, which at times is quite high, soon disappears, the respiratory symptoms grow milder, the expectoration of the casts ceases, and there is a complete and permanent recovery. In severe cases, however, death often ensues with all the symptoms of suffocation. The acute form sometimes becomes chronic, but this is rare.

The chronic form of fibrinous bronchitis may last for years. Usually the condition grows worse periodically, at varying intervals of time, and at each exacerbation casts are expectorated, while in the interval there is apparently merely a simple bronchial catarrh. Some observations are also recorded in medical literature of people who have expectorated these casts at intervals for years without any special disturbance of their health or their nutrition. In some cases other chronic pulmonary affections, like tuberculosis, finally develop.

The pathological anatomy of fibrinous bronchitis is not yet satisfactorily known on account of the rarity of the affection. The changes in the lungs found at the autopsy of fatal cases have usually been complications, like pneumonia, pleurisy, or tuberculosis, which stood in no direct relation to the fibrinous bronchitis. A loss of epithelium has been discovered in some cases in the parts of the bronchial mucous membrane that were attacked.

Prognosis.—In all acute cases the prognosis should be guarded, for we know that about one fourth of the cases terminate fatally. The chronic cases, as has been said, are usually very protracted and are subject to frequent recurrences, but they differ from the acute cases in being much less dangerous.

Treatment.—We make special use for inhalations of those remedies which, as we have said, are able to dissolve the casts. We usually employ a two- to-five-per-cent. solution of carbonate or bicarbonate of sodium, and above all lime-water, either pure or diluted with an equal volume of water. The internal administration of iodide of potassium, in doses of twenty to forty-five grains (grm. 1·5–3·0) a day, proves of advantage in many cases. Energetic inunction with mercurial ointment is sometimes of service. Expectoration of the casts may be aided in many cases by such expectorants as senega and benzoic acid, or by the timely use of emetics. We do not know any remedies which are able to prevent a return of the attacks in the chronic form. The treatment, except at the time of the attacks, is the same as in ordinary chronic bronchial catarrh.

CHAPTER V.

WHOOPING-COUGH.

(*Pertussis. Tussis convulsiva.*)

Ætiology.—By the name "whooping-cough" we mean a specific disease of the mucous membrane of the air-passages, which is chiefly seen in children, and is characterized by a peculiar violent and paroxysmal cough. Sporadic cases are of almost constant occurrence in large cities, but the disease often appears in epidemic outbreaks. Epidemics of whooping-cough follow epidemics of measles with remarkable frequency.

Whooping-cough is without doubt contagious, and therefore often attacks one child after another in the same family. Kindergartens, orphan asylums, and nurseries aid very much in extending the disease. The contagious element seems to be connected with the air expired by the patient, particularly with the secretion from the mucous membrane expectorated after coughing. Children are most subject to an attack up to the age of six years; from that age the disposition to the disease decreases rapidly with increasing years. Whooping-cough is seen, indeed, in adults, but it is quite rare, and almost always comparatively mild and rudimentary.

The epidemic onset, the contagiousness, and the whole course of the disease favor the theory of its infectious nature. The presence of the organisms which are supposed to be the poison of the disease has not yet been certainly demonstrated, although many have claimed to discover characteristic organisms in the sputa of patients. These statements, however, all conflict, and lack well-attested and methodical proof. If a patient has once had the disease, he is almost invariably secure against a new attack.

Symptoms and Course of the Disease.—Whooping-cough begins with the symptoms of a catarrh of the trachea and bronchi, which develops more or less rapidly, and which at first often shows nothing characteristic. We can at this period make a tolerably probable diagnosis only at a time when an epidemic is prevailing, or in case the child's associates have already been attacked with the disease. The cough is often quite severe at the beginning, but it does not yet come on in distinct paroxysms. Examination of the chest shows nothing peculiar except a few rhonchi. There is often a coryza, with frequent sneezing, and there is sometimes a mild conjunctivitis. The child is restless and feverish, especially toward night. The temperature may repeatedly reach 103° or 104° (39°–40° C.) in this initial fever. The duration of this first so-called catarrhal stage varies, but it usually lasts a week or ten days.

The catarrhal stage gradually passes into the second, convulsive stage, without any sharp boundary. The cough becomes more violent, and comes on in the separate paroxysms of whooping-cough which are characteristic of the disease. We do not know the particular reason why the cough has this paroxysmal character, but a nervous factor probably plays the chief part in it.

The peculiarity of the attack consists in the violent, paroxysmal fits of coughing, which are from time to time interrupted by deep, long-drawn, loud, and shrill inspirations, due to the occurrence of a spasmodic contraction of the glottis. Exceptionally there are cases without this loud whistling inspiration. The child becomes markedly cyanotic during the attack, the veins in the neck swell, and tears come into the eyes. Hæmorrhage into the conjunctiva, nose-bleed, and in some cases hæmorrhages into other organs, like the ear, the skin, and the brain,* often come on as a result of this stasis. Vomiting very often occurs either during a paroxysm or at its close. Involuntary evacuations of urine and fæces may also follow from the violent contraction of the abdominal muscles. Exceptionally we observe still more severe symptoms with a paroxysm: a complete spasmodic cessation of respiration with imminent danger of suffocation, or sometimes general convulsions.

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* In one instance we observed the very rare occurrence of a hemiplegia during a paroxysm of whooping-cough.

The paroxysms vary with the severity of the disease, frequently appearing ten or fifteen times in twenty-four hours; sometimes with greater frequency—fifty times or more. They also occur at night as often or even oftener than in the daytime. They come on either spontaneously or from some special predisposing cause. We may, for example, excite a paroxysm artificially by pressing on the larynx or by making the child cry. If there are several children with whooping-cough in the same room and a paroxysm attacks one of them, the others, as a rule, soon begin to cough too. Some prodromal symptoms often precede the peculiar paroxysm, such as general uneasiness, rapid respiration, or vomiting. At the end of a paroxysm many children remain very feeble and exhausted, but others recover very rapidly, and are playing again quite briskly a few minutes after.

In general the child feels quite well in the interval between the paroxysms, but the effects of the violent attacks of coughing may of course often be seen. Besides the occasional hæmorrhages into the conjunctiva, we find the eyelids somewhat swollen, their veins dilated and blue, and showing through the skin. A small ulcer is quite frequently formed on the frænum of the tongue, the origin of which is to be referred to mechanical causes. The tongue is violently protruded in the severe paroxysms of coughing, and the frænum is thus pulled or torn, or injured by the sharp lower incisors.

Physical examination of the lungs shows nothing abnormal in uncomplicated cases except a few moist râles or rhonchi. Sometimes the rhonchi are wanting, or are present in small numbers only a short time before a paroxysm, but in other cases an intense diffuse bronchitis is developed, which often leads to the development of a lobular pneumonia (*vide infra*). Sometimes, but not always, there is an acute catarrhal inflammation of the bronchi, and especially of the posterior wall of the larynx.

The fever, which is usually present in the first or catarrhal stage, is absent in the convulsive stage. The child is free from fever for the most part. We often find a slight rise of temperature up to 100° or 101° (38°–38·5° C.), but only toward night. Higher and more persistent fever points to the development of complications, especially on the part of the lungs.

The convulsive stage seldom lasts less than three or four weeks, and often much longer, up to three or four months. The paroxysms gradually become less frequent and less violent (*stadium decrementi*), until they finally disappear entirely; but relapses and fresh exacerbations also occur in this stage. Finally, however, the disease, in uncomplicated cases, goes on to a permanent and complete recovery.

Complications and Sequelæ.—The severe results which sometimes follow whooping-cough are probably partly due to the direct action of the specific causes of the disease, and partly to complications of a secondary nature whose origin is merely favored by the whooping-cough. The most important are complications in the lungs. A lobular, catarrhal pneumonia often develops after a severe bronchitis which involves the finer bronchi. In such cases the respiration becomes hurried and superficial, the fever higher, and the general condition bad even in the times between the paroxysms. On examination of the lungs, we hear numerous moist râles, especially over the lower lobes, and we can sometimes make out dullness on one or both sides if there is extensive pneumonic infiltration. Such cases are always very protracted, and many children succumb, partly from the disturbance of respiration and partly from general weakness and inanition.

Complications in other organs are much rarer. Among the most frequent are attacks of diarrhœa which exhaust the child's nutrition. Many observers have also mentioned the quite frequent occurrence of a croupous or diphtheritic inflammation in the pharynx and larynx in the course of whooping-cough. Finally, a case under our own observation may here be mentioned, in which death occurred with severe nervous symptoms, convulsions, and coma. At the autopsy very numerous capillary hæmorrhages were found in the brain.

Pulmonary emphysema is the first thing to be mentioned among the sequelæ of whooping-cough. From the marked pressure which the severe and frequent outbursts of coughing exert from within upon the alveoli of the lungs, they gradually become dilated. An acute lobular emphysema ("acute pulmonary inflation") is set up which sometimes passes into a typical chronic pulmonary emphysema (*vide infra*). Chronic bronchial catarrh may also remain for a long time after an attack of whooping-cough.

A third important sequel of whooping-cough is pulmonary tuberculosis. The bronchitis and lobular pneumonia which occur during a whooping-cough sometimes do not improve, especially in weak children with a tubercular tendency. The fever continues high, the child grows thin, and constantly becomes more and more miserable. At the autopsy we find cheesy nodules in the lungs, cheesy bronchial glands, and at times tuberculosis of other organs. These cases signify that when a tubercular infection is present, but is still latent, the whooping-cough acts as an exciting cause for the outbreak of the disease, or that a greater receptivity to infection with tubercular poison is created by the whooping-cough. Möbius has lately reported the occurrence in a few cases of paralysis as a sequel of whooping-cough. This usually begins in the lower and extends to the upper extremities, and is apparently due to neuritis.

The **diagnosis** of whooping-cough can not be made with certainty, as we have said, until the second or convulsive stage. It is easy then, however, since the characteristic attacks occur in no other affection of the lungs in like manner and with like frequency and duration. If we have no opportunity to observe the attack itself, and have to depend upon the description of the friends, the diagnosis is sometimes more uncertain. In such cases, however, certain signs are often present: the child has a bloated aspect, or we may find slight hæmorrhages into the conjunctiva, or ulcers on the frænum of the tongue, which make the diagnosis highly probable. Under some circumstances we may also make the attempt to bring on the paroxysm artificially (*vide supra*).

The **prognosis** is favorable with the majority of children if they are previously strong and healthy. Very young children are in more danger than older ones. There is danger if secondary pneumonia develops, and if the general nutrition and strength of the child suffer. As soon as the diagnosis is certain we must call the attention of the parents to the probable long duration of the disease. Regard must also be paid to the possibility of the development of sequelæ, especially in weak children suspected of tuberculosis.

Treatment.—Since the disease is protracted and is not devoid of danger, it is our duty, when an epidemic of whooping-cough prevails, to guard children from it as far as possible. If one child in a family is taken ill, the other children must be rigorously kept away from him. If circumstances permit, we should prefer to send them away to another place free from whooping-cough.

With regard to the treatment of the disease, we must first endeavor to fulfill general dietetic and hygienic indications. The child should breathe good, pure air, and for this reason it is often advisable to transfer the patient to a larger room, with as much air and sunlight as possible. The atmosphere should not be too dry, and it is advisable to employ a spray of carbolized water occasionally, or to hang up sheets moistened with the same in the room. In good weather the child should be out of doors a large part of the time, provided fever has ceased. City children are to be sent, if possible, into the country. The food should be good and nourishing, but dry and crumbly articles should be avoided, being apt to excite cough. Warm or lukewarm baths frequently prove very beneficial, particularly when there is considerable bronchitis, as they lessen the danger of a lobular pneumonia.

The medicinal treatment of whooping-cough has not yet shown brilliant results despite the large number of remedies recommended. Internally, quinine, belladonna, and bromide of potash are most worthy of a trial. Quinine is given in powders of one and a half to eight grains (0·1 to 0·5) several times a day, either in capsules or, in the case of smaller children, with chocolate. The earlier this remedy is employed the more prompt is said to be its beneficial influence. Lately the treatment of whooping-cough with antipyrine has been much recommended. Belladonna is prescribed in powders containing one twelfth to one sixth grain (0·005 to 0·01) of the extract of belladonna, giving three to five such powders a day. This remedy has often seemed to the author to diminish the number and violence of the paroxysms. The largest daily dose of sulphate of atropine to be given to children is one sixtieth of a grain (0·001), and even this amount demands in every case caution and watchfulness for the possibility of symptoms of poisoning, such as enlargement of the pupils and dryness of the mouth. Bromide of potash is employed in an aqueous solution in the dose of fifteen to forty-five grains per diem (1 to 3 grm.). Its benefit is probably due to its power to diminish reflex excitability. The same drug employed in an atomizer often has a palliative effect.

If the paroxysms are very violent we may cautiously administer small doses of morphine or codeia. Inhalations of chloroform and ether have also been recommended. The following mixture is a suitable one:

℞ Chloroformi.................................... ℥ j (grm. 30);
Ætheris.. ℥ ij (grm. 60);
Ol. terebinthinæ................................. ʒ ijss. (grm. 10). M.

Sig. One or two teaspoonfuls to be poured upon a pocket-handkerchief for inhalation.

Finally, considerable success has attended the application of cocaine to the throat and larynx by means of a brush. A solution of ten to fifteen per cent. has a considerable influence in modifying the frequency and violence of the paroxysms. Michael advocates the daily insufflation into the nostrils of powdered benzoin.

Inhalations of various antiseptic remedies have been frequently employed because of the infectious nature of the disease. The practitioner must not expect too much from them, however, although they sometimes act well. A one-per-cent. or two-per-cent. solution of carbolic acid is most frequently employed for inhalation. It may be given several times a day for periods of two or three minutes at a time. Next to this, turpentine and benzine are most to be recommended; of these, twenty or thirty drops are poured upon a sponge previously moistened with hot water. [Farlow and others report marked success from spraying the upper air-passages with a two-per-cent. solution of resorcin.]

For the treatment of the complications and sequelæ of whooping-cough the reader is referred to the appropriate chapters of this book.

CHAPTER VI.

BRONCHIECTASIS.

(Bronchial Dilatation.)

Dilatation of the bronchi is not a separate disease, but it is a result of various affections of the lungs and bronchi. Nevertheless, we will speak of it briefly in this connection since many cases of bronchiectasis present the appearance of quite a characteristic disease.

We distinguish anatomically the cylindrical and saccular bronchiectases.

Cylindrical bronchiectasis consists of a uniform dilatation of a bronchial tube, and occurs most frequently in the medium-sized, or rarely in the finer bronchi of one or more lobes of the lung. It is usually due to a long-continued bronchitis, and develops most frequently in cases of emphysema, and also in whooping-cough, measles, and sometimes in phthisis, etc. The primary process is probably always the atrophy which follows the catarrh, and the diminished resistance of the bronchial walls thus occasioned. The dilatation of the lumen of the bronches is produced gradually, partly by the traction of the thorax during inspiration, and still more by the increased pressure in the bronchi due to the frequent and violent fits of coughing, and finally, perhaps, by the constant pressure of the stagnating secretion.

The diagnosis of cylindrical dilatation of the bronchi is only a probable one. We suspect that a bronchiectasis has formed if the conditions are fulfilled which we know lead to it. In the chronic bronchial catarrh of emphysema we judge that there is cylindrical dilatation of the bronchi if the secretion is very abundant and comparatively thin, and separates on standing in a sputa-cup. The dilatation is usually emptied by a severe paroxysm of coughing, such as is apt to occur in the morning if the secretion collects in great quantity during the night. Physical examination usually gives numerous small and medium moist râles, especially in the lower lobes. The respiratory murmur sometimes loses its vesicular character in marked cylindrical bronchiectasis, and has a more indefinite and tubular quality.

Saccular bronchiectases are spherical or oval dilatations which are confined to a definite portion of the bronchial tube. They may attain a diameter of several centimetres. The bronchus passes suddenly or gradually into the dilatation, and it is often obliterated so that the bronchiectasis forms a completely closed cavity. The wall of a saccular bronchiectasis loses in great measure the character of a normal bronchial wall. As a rule it is atrophied to a high degree, the atrophy involving not only the mucous glands, but also the muscular fibers, the elastic elements, and even the cartilages, so that the bronchiectasic cavities seem lined with nothing but a thin membrane. In other cases, however, we find hypertrophic processes, which involve the connective tissue of the mucous membrane, and lead to band-like projections and swellings. Finally, ulcerative processes may develop on the inner surface of a bronchiectasis and attack the surrounding lung-tissue, and change the bronchiectasis to a typical ulcerating cavity.

Only rarely, for example in emphysema, do we find a single saccular bronchiectasis surrounded by tolerably normal lung-tissue. Its origin, then, is to be referred to causes like those which have been given above for the much commoner cylindrical bronchiectases. In the great majority of cases we find saccular bronchiectases, singly or in large numbers, surrounded by indurated and contracted lung-tissue. They form one of the complications of "pulmonary contraction" [fibroid phthisis], which is almost always associated with contraction of the pleura. Since Corrigan's day we have with good reason looked upon this contraction as the chief cause for their origin. By the gradual shrinking and retraction of the lung, which as a rule has become adherent to the costal pleura, a traction is exerted upon the bronchial walls from without to which they gradually yield. Thus arises the frequent combination of pulmonary contraction with the formation of bronchiectases. This combination is usually unilateral, and involves the whole lung or only one of the upper or lower lobes. This form has been described from a histological stand-point as a chronic interstitial pneumonia, and it has been believed possible to make a sharp distinction between it and the chronic tubercular processes in the lung.

We often see the form of pulmonary contraction in question developing as a result of pleurisy. Laennec first advanced the opinion that in such cases the pleurisy was the primary trouble, and that from it an interstitial inflammatory process attacked the connective tissue of the underlying lung and led to contraction and then to the formation of bronchiectases. In our opinion we must indeed recognize the manifold anatomical and clinical peculiarities of the combination of pulmonary contraction and formation of bronchiectases in question, but ætiologically we are unable to separate it from pulmonary tuberculosis (*vide infra*), at least in the great majority of cases. But on the other hand it can not be denied that extensive bronchiectasis is sometimes observed entirely independent of any tuberculous process. In these rare cases the bronchial dilatation seems to be a sequel in most instances of a severe antecedent chronic bronchitis. It is seen, for example, in workmen who are obliged to inhale a great deal of dust. There can be no doubt, however, that the process here finally extends to the lung-tissue, for upon autopsy the parenchyma between the various bronchiectasic cavities is usually not normal, but transformed into a firm indurated tissue. As a rule the disease is mainly limited to one side, involving perhaps the whole lung, or, if only a single lobe, usually the lower one.

The symptoms caused by saccular bronchiectasis alone are derived in part from the result of physical examination and in part from definite peculiarities of the sputum. If great bronchiectasic cavities lie near the chest-wall, they may give the same physical signs that we shall learn to recognize later in the description of tubercular cavities. Bronchiectases lying within the lung, however, are often devoid of definite physical signs, so that at most we may suspect them from other symptoms, like the peculiarities of the sputum. The more abundant the formation of bronchiectases the more does the respiration lose its vesicular character and become harsh and finally bronchial. Inasmuch as there is usually a very considerable secretion of mucus, we generally find, upon auscultation, abundant medium and even coarse moist râles.

The expectoration is, as a rule, very abundant, and it is often raised "by mouthfuls." On standing, it exhibits a distinct division into an upper layer of serum and a lower of pus. It usually has a stale, sweetish odor, but it may be fœtid. The diagnosis of the extensive formation of bronchiectases in the lungs is usually quite easy. In order to avoid confounding it with chronic tuberculosis, the following should be especially considered: In bronchiectasis the patient does not present a true cachexia; he is usually somewhat cyanotic and often pale at the same time. The terminal phalanges of the fingers are often clubbed, as in fœtid bronchitis. Fever is usually absent, unless there are some special complications. The expectoration is more abundant and more distinctly stratified than is often seen in tuberculosis; and, most important of all, it contains, of course, no tubercle bacilli.

Since bronchiectasis may give rise to a fœtid bronchitis, and since, on the other hand, as we have said, fœtid bronchitis itself often leads to the formation of bronchiectasis, we can understand the manifold relations and changes which the two forms of disease described may furnish. If ulcerative processes arise in the wall of a bronchiectasis, they may give rise to hæmoptysis.

The further course of bronchiectases depends, of course, upon the nature of the primary affection. The cylindrical dilatations which arise after a severe bronchitis, as happens in whooping-cough, measles, or typhoid, may in many cases gradually get well; but recovery from saccular bronchiectasis by a process of obliteration, if it occurs at all, is extremely rare. Nevertheless, the course of the disease may be comparatively benign, since the affection often remains circumscribed, and the patient's general strength and nutrition suffer comparatively little from it. Finally, of course, severe symptoms arise, either from insufficiency of the heart, when there is cyanosis, dyspnœa, or œdema, or as a result of emphysema, tuberculosis, gangrene, or extensive lobular pneumonia.

The **treatment** is never directed against the bronchial dilatation as such, but toward its causes or sequelæ. The treatment of bronchiectasis, therefore, coincides with the treatment of chronic bronchitis, emphysema, fœtid bronchitis, chronic tuberculosis, etc.

CHAPTER VII.

STENOSIS OF THE TRACHEA AND BRONCHI.

1. Tracheal Stenosis.

Ætiology.—Stenosis of the trachea may be caused either by diseases in the vicinity of the trachea, or by diseases of the trachea itself. The first-named mode of origin is the more frequent. To this are due all the stenoses of the trachea from compression. Enlargements of the thyroid gland from simple struma and new growths, aneurisms of the arch of the aorta and of the innominate artery, tumors and abscesses in the anterior mediastinum, swelling of the lymph-glands at the bifurcation of the trachea, abscesses on the anterior surface of the cervical vertebræ, etc., may exert so great a pressure on the trachea from without that its lumen is made narrower. Besides the direct action of pressure, in most cases, a gradual atrophy from the pressure and a softening of the rings of cartilage sometimes plays an important part, according to Rose, in the occurrence of stenosis. A collapse of the trachea may arise from this "flaccid softening," which may come on quite suddenly, and may cause many of the cases of sudden "scrofula death."

Changes in the trachea itself leading to stenosis are quite rare. Cicatricial stenosis as a result of syphilitic ulcerations is relatively the most frequent. New growths in the trachea are also to be mentioned, such as polypi and carcinomata, the latter almost always having invaded the trachea from the adjacent parts. Very rarely acute and chronic inflammatory processes like perichondritis lead to a swelling of the mucous membrane sufficient to cause stenosis. In conclusion, we may mention that stenosis of the trachea may be due to the presence of foreign bodies.

Symptoms.—If the stenosis is so extreme that there is a real hindrance to respiration, a very striking modification of the breathing occurs. It is difficult and labored, and is performed only by the help of the accessory muscles. Both expiration and inspiration are protracted, long drawn, and accompanied by a loud stridor. In many cases inspiration is more difficult than expiration, so that there is accordingly a preponderating inspiratory dyspnœa, and the number of respirations a minute is diminished. If the entrance of air into the lungs is incomplete in spite of the lengthening of the respirations, we see an inspiratory retraction of the lower part of the thorax, and sometimes of the throat and the supra-clavicular fossæ. In tracheal stenosis the larynx, however, shows little or no to-and-fro movement on respiration. This fact is of value in diagnosis in distinguishing tracheal from laryngeal stenosis, for in the latter the respiratory movements of the larynx are quite well marked.

We sometimes notice in the pulse during inspiration a marked fall in tension and in the height of the pulse-wave, the *pulsus paradoxus*. With the sphygmograph we can show still more plainly the changes in blood-pressure, which vary quite markedly with the respiration. The frequency of the pulse is usually a little increased, but sometimes it is diminished.

The symptoms of the disease just described may form so characteristic a picture that we can recognize it at the first glance. More precise information as to the seat of the stenosis, or the accurate differentiation of tracheal stenosis from the very similar picture presented by laryngeal stenosis, demands a direct laryngoscopic examination of the larynx and trachea, which, of course, is hardly practicable in a patient with a high degree of dyspnœa.

2. Bronchial Stenosis.

Narrowing of a primary bronchus, which is the only form to be mentioned here, arises most frequently as a result of the presence of foreign bodies. These may enter the air-passages by means of a deep inspiration while eating, or during sleep. We know that foreign bodies get into the right bronchus, which is wider, somewhat more frequently than they do into the left. Stenosis of the main bronchi from pressure also arises from aneurisms of the aorta, mediastinal tumors, enlarged bronchial lymph-glands, etc. Stenosis of the left bronchus from the pressure of the greatly dilated left auricle has been observed in mitral stenosis.

The symptoms are not equally distinct in all cases, and they depend upon the shutting off of the corresponding part of the lung. The dyspnœa is usually very evident, especially in acute cases. The respiratory excursions are much less on the affected side than on the sound side. The percussion-note, indeed, remains clear, but the vesicular respiratory murmur disappears, and instead of it we sometimes hear over the whole side a loud whistling or humming sound, the vibration of which can in some cases be felt by the hand applied to the chest-wall. The vocal fremitus is diminished on the affected side. A vicarious emphysema soon develops in the other lung.

Lobular pneumonia frequently develops as a result of the entrance of foreign bodies into a bronchus, because the agents of inflammation have entered at the same time with these bodies, and, as the expectoration can be evacuated only with difficulty, and hence is more or less stagnant, these irritants can readily establish themselves in it. In stenosis from pressure the character of the disease may of course be modified in many ways by the primary disease.

The **prognosis** and **treatment** of tracheal and bronchial stenosis depend entirely upon the nature of the primary disease. General statements as to treatment, therefore, need not be given here. A direct mechanical treatment of tracheal stenosis in appropriate cases, such as cicatricial stenosis, may be undertaken according to the different modes of dilatation above enumerated. The methods for removing foreign bodies from the larger air-passages belong to the domain of surgery. The employment of an emetic has met with distinct success in such cases, but it is not without danger, for the foreign body may wedge itself into the glottis during the act of vomiting and result in the danger of instant suffocation.

CHAPTER VIII.

BRONCHIAL ASTHMA.

(*Nervous Asthma.*)

BRONCHIAL asthma is a disease clinically well characterized, but ætiologically it is probably not quite a distinct affection. Its chief symptom consists of marked paroxysmal attacks of dyspnœa. The cause of the dyspnœa is not to be sought in any coarse factor that can be demonstrated anatomically, but it is probably due, at least in part, to some abnormal condition of nervous irritation. The chief theories as to the origin of the asthmatic attacks will be given further on. The disease is decidedly more common in men than in women, and it is not very rare even in children.

Symptoms and Course of the Disease.—"Nervous" bronchial asthma consists, in its purest form, of attacks of shortness of breath, which come on in persons who are otherwise quite well, with varying frequency and varying duration, partly from some special cause, and partly without any discoverable reason. In the intervals between the attacks the patients are completely well, and do not show the slightest signs of any disease of the respiratory or circulatory organs.

The asthmatic attack either begins quite suddenly, or it is preceded for a shorter or longer period by prodromata. These consist in a general feeling of discomfort, in abnormal sensations in the larynx or epigastrium, sometimes in remarkably frequent gaping, and often in a marked coryza associated with a good deal of secretion and frequent sneezing (compare the relation between many attacks of asthma and diseases of the nose, given below). The attack begins in most cases at night. The patient wakes up with an intense feeling of pressure and anxiety. Sometimes he complains of a feeling of pain in the chest. He has to sit up straight, and in severe cases even to get out of bed. He often hurries to an open window in order to "get air." His expression is anxious; his skin becomes pale and cyanotic, and sometimes is covered with a cold sweat. The respiration, too is altered in a very peculiar and characteristic way. Both inspiration and expiration are almost always accompanied by a high-pitched whistling sound, audible at a distance. Both respiratory acts are labored, requiring the aid of the accessory muscles. On inspiration, only the upper part of the thorax is elevated to any extent. We see in the neck the inspiratory contraction of the sterno-cleido-mastoids, the scaleni, etc. Still more striking, however, is the labored, panting, long-protracted expiration, during which the abdominal muscles are contracted to a board-like hardness. We therefore recognize the disturbance of respiration in asthma as essentially an expiratory dyspnœa. The frequency of respiration is in many cases normal, or even somewhat diminished, yet we have repeatedly counted thirty or forty respirations a minute.

On physical examination of the lungs during the paroxysm, we find the percussion-note over them normal or even strikingly loud and deep—the "box-tone." The lower boundary of the lung is usually found one or two intercostal spaces lower than normal. We accordingly have to do with an abnormally low position of the diaphragm, with an acute emphysema. On auscultation, whistling and creaking sounds, which quite obscure the vesicular murmur, are heard over most of the lung, especially during the long expirations. In many places, indeed, the respiratory murmur is entirely absent, or we hear only a low whistle on expiration. Toward the end of the paroxysm the noises become deeper and more booming, and sometimes we hear a few moist râles.

In brief paroxysms there may be scarcely any cough or expectoration. In most, particularly in the tedious, cases, there is, however, a scanty tough mucous expectoration. In this are found, beside the ordinary constituents of simple bronchitic sputum, larger or smaller numbers of very characteristic clumps. These may be yellow or greenish-yellow, or, on the other hand, gray. The yellowish clumps, which are usually very tough, and often consist of a clump of thready matter, represent swollen and fatty-degenerated pus corpuscles, between which are very frequently interspersed a considerable number of pointed octahedral crystals. These crystals were first described by Leyden in the sputum of asthmatic patients, and are usually termed asthma crystals (see Fig. 22). Chemically they are identical with "Charcot's crystals," which are found in the leukæmic spleen, the bone marrow, and the semen, and they probably represent the phosphoric-acid salt of a peculiar base (Schreiner's base, C_2H_5N). As soon as the paroxysms cease the number of crystals in the sputum usually begins to diminish rapidly, and it is often possible to observe in them evident tokens of disintegration. Often, also, numerous ciliated epithelial cells are found, in addition to the crystals, in the yellow masses. The gray plugs in the sputum of asthmatic patients consist mainly of clumps of thready mucus, and contain the peculiar "spirals" which were first described by Ungar and by Curschmann. Many of these spiral threads are visible to the naked eye, but others demand the microscope for their recognition, through which they are seen as brilliant forms composed entirely of various sized bands and threads collected in spirals (see Fig. 22). Sometimes a brilliant central thread of small diameter is seen in the midst of the spiral. Around the spirals are found round cells, drops of fat and myeline, epithelium, and, according to Lewy, frequently also numerous epithelial cells from the pulmonary alveoli. As to the precise way in which the spirals and their central thread develop, the question is not yet settled, but it is certain that the spirals represent casts of the minutest branches of the bronchi, and therefore indicate the existence of a peculiar disease of the terminal bronchial twigs (see below). It should be added that sometimes also the microscope reveals in the sputum crystals of oxalate of lime (Ungar) and of phosphate of lime (Lewy).

The pulse is usually accelerated during the asthmatic paroxysm; the bodily temperature is normal, or sometimes even subnormal. In asthmatic patients who have protracted attacks we have repeatedly seen a slight febrile movement up to about 102° (39° C.).

The duration of the asthmatic paroxysm is very different in individual cases, as has already been said. Sometimes it lasts only a few hours, but sometimes it lasts several days, and even weeks. The attacks of protracted asthma are not very rare. Marked exacerbations and remissions of the disease usually alternate

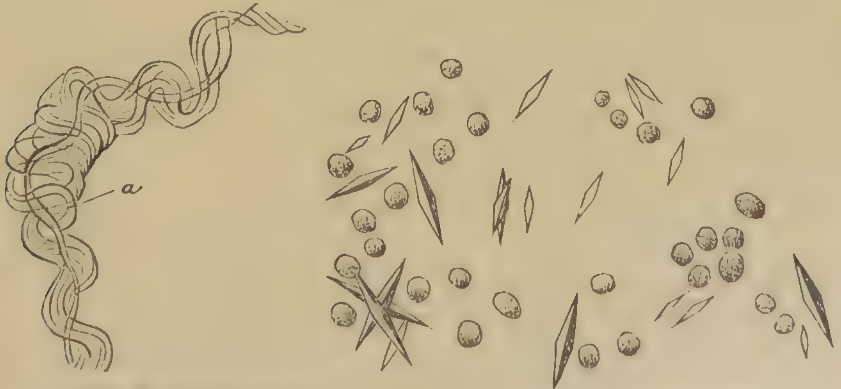

FIG. 22.—Asthma crystals and Curschmann's spirals (*a*, central fiber).

in them. The frequency of the paroxysms in ordinary asthma also varies exceedingly. Sometimes they come on almost every night, and then there are long pauses of months and years, so that we can not make any general statements as to the course of the disease. Definite recoveries are quite rare; they are most frequent in children.

Although patients with the form of pure essential asthma which we have so far described seem perfectly well in the intervals between the attacks, there is also a symptomatic asthma. This is seen chiefly in patients with emphysema and chronic bronchitis. The term "symptomatic asthma," however, can be used only when the attacks actually show the symptoms of pure asthma, and when the dyspnœa which occurs in them has no relation to the anatomical lesions present. In such cases it is often hard to decide whether the existing emphysema and chronic bronchitis are really the primary disease, or the result of the asthma. There is no doubt but that a secondary emphysema of the lungs may develop as a result of frequent and protracted asthmatic attacks. The attacks of dyspnœa, which come on in chronic affections of the heart and blood-vessels—cardiac asthma (*vide infra*)—depend upon other causes than the peculiar bronchial asthma, and should not be classed with it.

Theories as to the Origin of Asthma.—Ætiology.—The peculiarity of the asthmatic symptoms has given rise to numerous theories as to the origin of asthma, yet none of them have obtained general recognition up to the present time. Many authors, like Weber, Störck, and Fräntzel, seek the underlying cause of asthma in an acute swelling of the bronchial mucous membrane, as a result either of a sudden dilatation of the blood-vessels arising from nervous influences or of a very acute catarrh. Wintrich and Bamberger have advanced the theory that asthma consists in a tonic spasm of the diaphragm, by which the diaphragm is kept motionless in a fixed inspiratory position; but it is at once plain that such a condition can at least not play the chief part in the occurrence of asthma, for we can usually feel the respiratory movements of the diaphragm quite plainly during the paroxysm. It should be stated that Riegel, who is the latest champion of the theory of diaphragmatic spasm, is of the opinion that there is not a complete tetanic spasm of the diaphragm, but merely a superexcitation of the phrenic nerve, and that the excursions of the diaphragm are not completely inhibited.

The theory long ago advanced by Trousseau, the chief advocate of which of late is Biermer, is the most probable one, and is now generally accepted, namely, that the spastic nervous element, which is not wholly to be disregarded in any explanation of bronchial asthma, consists of a tonic spasm of the muscles of the smaller bronchi. The tonic contraction of the smaller bronchi explains the whistling sounds that are heard. A marked hindrance to respiration is set up which can be more easily overcome by the inspiratory suction of the thorax than by the expiratory pressure. Since the latter acts not only upon the alveoli, but also upon the lesser bronchi themselves, the closure of the bronchi upon expiration is still more marked. The air which is drawn into the alveoli can consequently get out again only imperfectly, and this explains the expiratory dyspnœa, the emphysema that soon occurs, and the low position of the diaphragm. The acceptance of this theory of bronchial spasm also readily explains the often sudden onset, and just as sudden cessation, of the asthmatic attack.

If we inquire further, however, into the cause of the occurrence of the bronchial spasm, only a very indefinite answer can be given; for little is said by answering that asthma is a neurosis of the vagus. Many facts make it very probable that the spasm is of reflex origin, at least in many cases. Leyden has expressed the suspicion that the irritation of the mucous membrane by the pointed crystals, which he discovered, gives rise to the spasm. It may be said in opposition to this, however, that the "asthma crystals" are sometimes found in the sputum of patients with emphysema who have no asthmatic symptoms, and also that in asthmatic patients the severity and duration of the attacks stand in no constant relation to the number of the crystals. Nor can the above-pictured spirals be regarded as the cause of the paroxysms of asthma, since they likewise occur in other diseases of the minutest branches of the bronchi—for example, they are not very rare in croupous pneumonia.

The fact lately corroborated by numerous observations by Voltilini, B. Fränkel, Hack, and others, is very important—namely, that the asthmatic paroxysm is sometimes of reflex origin, starting from some disease of the nasal mucous membrane. We find quite often, for instance, that asthmatic patients are suffering from chronic diseases of the nose, like chronic catarrh, nasal polypi, and especially the enlargement of the so-called erectile bodies of one or more turbinated bones, and that after their removal the asthma disappears. In this connection may be cited the noteworthy fact that many asthmatic patients have an attack brought on by certain odors, for example, at the smell of freshly roasted coffee, or of ipecacuanha. Trousseau, who suffered from asthma himself, always had an attack on smelling violets. It is doubtful whether a pure bronchial asthma can have a reflex origin from other distant organs. The connection between asthma and diseases of the pharynx, or hypertrophy of the tonsils, is extremely probable in some cases, but the statements as to the occurrence of asthmatic paroxysms in diseases of the stomach ("dyspeptic asthma"), of the intestine, or of the female sexual organs, are to be taken only with great reserve. We usually have to do here with a confusion between pure asthma and other conditions of dyspnœa—nervous dyspnœa, conditions of cardiac weakness, etc.

In a large number of cases—which seem to us to be most characteristic—the disease can be explained, in our opinion, only by the hypothesis of a peculiar primary disease of the bronchial mucous membrane, particularly affecting the terminal twigs of the smallest bronchi, and whose special feature, somewhat like the spasm of the glottis in whooping-cough, consists in the occurrence of a reflex bronchial spasm. The whole type of the disease and the peculiarities of the expectoration, the spirals, and crystals, furnish unequivocal testimony for this theory of asthma. Curschmann therefore claims that the anatomical basis of these cases is an exudative bronchiolitis. We think that "asthmatic bronchiolitis" is the most fitting name for such cases of asthma as are not found to be dependent upon disorders of the nose or similar causes.

The observation, often made, that many asthmatic patients have attacks only when in certain places, and are quite free from them in others, is very remarkable. They sometimes have an attack at every change of place. In conclusion, it may be mentioned that in some cases a distinct hereditary predisposition to asthma has been observed, and that asthma sometimes occurs in families with a general neurotic tendency.

Diagnosis.—This is usually easy if we pay careful attention to all the symptoms and to the whole course of the disease. Other conditions which may lead to dyspnœa are of course to be excluded by a careful examination of the chest. Attacks of spasm of the glottis and of paralysis of the openers of the glottis are to be differentiated from bronchial asthma by the predominance of inspiratory dyspnœa as well as by other signs. And in cardiac asthma and the manifold varieties of respiratory spasm and neurotic angina, such as are seen especially in hysteria, not only are the other attendant symptoms different from those seen in genuine bronchial asthma, but the very dyspnœa itself is usually of quite another variety.

Prognosis.—There is hardly ever any immediate danger to life even in the most intense asthmatic paroxysms, but permanent recovery is rare, since even after long intervals the attacks may finally return. The chief danger in severe and protracted cases lies in the development of a pulmonary emphysema with its further consequences.

Treatment.—In every case of asthma the first thing to be thought of is whether there is not a definite cause whose removal may cure the disease. In this connection we should examine the nose carefully, since numerous observations have recently shown that a previously existing asthma may permanently disappear after the treatment of some nasal disease which may be present, like the removal of polypi, the destruction of the erectile bodies by the galvano-cautery, etc.

If we can not satisfy the causal indication in this way, we should always try next a remedy which must pass for a direct specific against certain forms of asthma—iodide of potassium. In doses of twenty to forty-five grains a day (grm. 1·5–3·0), which can be increased if necessary, this usually causes a rapid improvement, which of course is not always, although it is frequently, permanent. If iodide of potassium has been used in vain, we must turn to the other remedies which have been employed against asthma, although their action is often quite uncertain. We may mention here the nitrite of sodium (two parts in one hundred and twenty of water, two to three teaspoonfuls a day), and nitro-glycerine, which has an analogous action (twenty drops of a one-per-cent. alcoholic solution in six and a half ounces (grm. 200) of water, a tablespoonful two or three times a day); also quinine, bromide of potassium, belladonna, atropine, arsenic, etc. In some cases pneumatic treatment, such as the inhalation of compressed air, has been successful, and sometimes, too, electricity (galvanization and faradization of the neck), or hydrotherapy, has been claimed to give relief. Change of climate may be of distinct service. Many patients bear the sea-air well, while with others mountain traveling exerts a favorable influence.

In severe cases a special symptomatic treatment of the attack itself is often necessary. Narcotics are without doubt the most effective, especially chloral and morphine. In severe attacks we can not avoid injections of morphine, but we must always be cautious in order that the patient may not form the habit of using this to excess. Germain Sée has lately strongly advocated inhalations of pyridin. Of this, about one drachm (4 to 5 grm.) is shaken into a saucer and the fumes are inhaled three times a day for fifteen to thirty minutes. Inhalations of chloroform and ether are also much employed. Among other useful remedies and devices we may mention mustard plasters to the chest and calves, putting the hands and feet into hot water, inhalations of nitrite of amyl, inhalations of turpentine or ammonia vapor; also the often-used fumigation with saltpeter paper—unsized paper dipped in a concentrated solution of nitrate of potassium and dried. The stramonium cigarettes to be had in most drug-stores are much praised; or the patient may smoke stramonium or belladonna leaves which have previously been dipped in a solution of saltpeter and then dried. Among internal remedies we may mention tincture of lobelia, formerly much used, and also the remedy lately employed by Penzoldt, the tincture of quebracho, a tablespoonful pure or in some mucilaginous vehicle.

[Potassic iodide is more likely to prevent recurrence if it is given continuously, for several months at least, and it should not be thrown aside as useless until it has been pushed to the limit of toleration without avail. A convenient form of administration is in saturated aqueous solution, a minim of which represents about a grain of the drug.

The syrup of hydriodic acid may be substituted for potassic iodide in cases of intolerance of the latter. Grindelia robusta, a drachm of the fluid extract three or four times a day, serves sometimes to prevent recurrence of attacks. Marked alleviation of the paroxysms is often obtained from the inhalation of fifteen to thirty drops of the iodide of ethyl.]

SECTION IV.

DISEASES OF THE LUNGS.

CHAPTER I.

PULMONARY EMPHYSEMA.

(*Alveolar Ectasis. Increased Volume of the Lungs.*)

Nature and Ætiology of the Disease.—Pulmonary emphysema, the abnormal inflation of the lungs, is one of the commonest pulmonary affections. It either develops in separate parts of the lung, in which case it is subordinate to other pathological changes which co-exist in the lungs, or it involves almost the whole extent of both lungs, and then presents the symptoms of a characteristic affection, which it is usually easy to recognize.

The essence of pulmonary emphysema, the condition from which most symptoms are immediately derived, is the loss of elasticity in the lungs. If we compare the sound lung with its normal elastic force to a new and very tense rubber band, the emphysematous lung must be compared to an old and lax band that is stretched and pulled out. We therefore see why the emphysematous lung takes up a greater space than the sound one, for, on account of its lack of elasticity, it can no longer contract to its former volume. We may thus call emphysema a permanent inspiratory distention of the lung from which it can no longer return to its expiratory condition. If we open the thorax of a subject with normal lungs, they contract, as is well known, but the emphysematous lungs remain in their inflated condition after the thorax has been opened.

If we inquire into the factors which cause this loss of elasticity in the lung, we find that they are the same kind of influences which tend to diminish the elasticity of any other elastic body. As a rubber band, by much pulling and stretching, gradually gets longer and less elastic, so the lungs, as a result of their abnormally frequent and severe distention, gradually become inelastic and emphysematous. The normal traction of inspiration, which is continually making new demands on the elastic powers of the lungs, finally leads to a loss of elasticity in them. In advanced age most lungs become more or less inelastic. The lungs of an old man are like an elastic band, which has done its work for years but which has finally become yielding. We therefore class the emphysema of the lungs in old age rather among the states of involution such as develop in almost all organs in advanced life, than among special pathological changes. We distinguish, moreover, most of the lungs with senile emphysema from other emphysematous lungs by the fact that their volume as a whole is not increased, but is rather diminished below that of the healthy lung, since we find in them the extensive atrophic processes of old age.

The condition becomes pathological, however, if the elasticity of the lung is deficient in earlier years and without exposure to the action of the special injurious influences which will soon be mentioned. In such cases of emphysema, developing in middle life or even in youth, the idea of a congenital weakness of the elastic elements in the lungs can not be set aside. It probably consists in a quantitative or a qualitative defect of the elastic tissue. Some observations seem to corroborate the statement that a disposition to emphysema may be present in several members of the same family.

If a lung whose elasticity is previously subnormal can not persistently satisfy the ordinary demands upon it, a normal lung, on the other hand, also loses its elasticity if the demands made upon it are greater than it can perform. This is the reason why pulmonary emphysema is in part considered a disease arising from the occupation. We mean here not only those influences which lead to chronic bronchitis and thus later to emphysema (*vide infra*), but more especially the abnormal demands upon the lungs in all those callings which necessitate severe physical labor. We must not only regard the deeper and more rapid respirations, but also the increased pressure on expiration to which the lungs are often exposed in the raising of heavy weights, etc. This explains the great frequency of emphysema in the laboring classes, and also its greater frequency in men than in women. Beside this, we must add that in certain callings, like glass-blowing and horn-blowing, the overstraining of the lungs is much more direct. In all such cases emphysema may be termed simply a premature exhaustion of the lungs.

In very many cases emphysema develops as a result of other diseases of the lung, and especially as a result of chronic bronchitis. Dry catarrh of the middle and finer bronchi when of long duration leads, as a rule, to pulmonary emphysema. The abnormal mechanical influences to which the lungs are thus exposed act both in inspiration and in expiration. Since the entrance of air to the alveoli is rendered more difficult by the swelling of the mucous membrane in the smaller bronchi, abnormally deep and strong inspirations are necessary, with a marked expansion of the alveoli, in order to draw a sufficient quantity of air into the alveoli. The alveolar walls are therefore exposed to an abnormal traction at each inspiration. On expiration, a pressure from within, which is perhaps even more injurious, acts on the alveoli. The ordinary expiration, which usually needs only the elastic power of the lungs, is not sufficient in chronic bronchitis to drive the air out of the alveoli through the narrowed bronchi. Thus arise the difficulty and delay in expiration which are present in chronic bronchitis, and which lead to the active participation of the muscles of expiration, the abdominal group of muscles. On forced expiration, however, the pressure does not act simply upon the contents of the alveoli, but much more upon the smaller bronchi themselves. The channel of exit for the air from the alveoli, therefore, becomes still narrower. Since the air can not escape, the pressure within the alveoli is raised by the efforts at expiration, and the alveolar wall is thus again abnormally expanded. The cough, which is often present in chronic bronchitis, is a further factor, which acts in a precisely similar injurious fashion. The attacks of coughing begin with a forced contraction of the muscles of expiration, which follows the closure of the glottis. Until the glottis opens, therefore, the lower parts of the lung especially are put under strong pressure. The air in them, which can not escape outward, is driven into the upper parts of the lung, and there leads to expansion of the alveoli, and finally to emphysema.

We accordingly see that a number of injurious influences co-operate in the gradual development of emphysema from chronic bronchitis, and that, sooner or later, these influences have as their result the gradual dilatation of the lungs. Here, too, we must bear in mind the individual differences in the resisting power of the lungs.

Conditions precisely similar to those in chronic bronchitis occur in other diseases, and lead in like manner to pulmonary emphysema. We very often see the development of emphysema in severe and persistent whooping-cough. The worst factor here, besides the existing bronchitis, is the frequent paroxysms of coughing. We have already mentioned, in the description of bronchial asthma, both the acute emphysema which occurs during the attacks, and the final development of a permanent emphysema.

In conclusion, we must here consider a theory advanced by Freund, which would make the development of an emphysema dependent upon a "primary rigid dilatation of the thorax." It is indeed conceivable that from certain pathological changes in the costal cartilages, as Freund claims, a thorax, which had become rigid in the position of inspiration, might exert a constant abnormal traction on the lungs and so give rise to an emphysema. The occurrence of this hypothetical primary disease of the cartilages, however, has up to the present time not been established. It is rather considered by the majority of authors as a secondary change, developing as a result of emphysema or else simultaneously with it. On the other hand, it is certainly remarkable that we sometimes observe in children the "emphysematous habit" of the thorax and neck, which will be more fully described further on, and that in fact in such children we can often discover early in life a beginning emphysema.

Besides the already described essential or substantial emphysema, which is a special disease attacking both lungs uniformly, we distinguish a so-called vicarious or complementary emphysema. If, by any disease, certain portions of the lungs are incapacitated in their functions, the parts which remain healthy must then assume the whole business of respiration. They become excessively expanded on inspiration, and as a result they become emphysematous. Thus we see emphysema of the upper lobes in affections of the lower lobes. Emphysema of one lung is most frequently observed clinically when the other lung is extensively diseased, especially in unilateral chronic contractions of the lungs and pleuræ, usually seen in tuberculosis. Vicarious emphysema may also be confined to quite small portions of the lung, but then it is merely of pathological and not of clinical interest.

Pathological Anatomy.—As we have seen, the actual abnormality of the lung in emphysema is not due to a pathological change, but to a change in its physical conditions. The loss of elasticity of the lung is shown in its greater volume, in its lack of contractility, and in its persistence in a position of inspiration.

The single alveoli are of course just as much expanded as the whole lung, but their walls show at first no histological changes. We have here, then, a condition which Traube has called "increased volume of the lungs," and has distinguished from the "pulmonary emphysema" proper. This distinction is without doubt justified anatomically, but clinically it can not well be maintained. As the distention is constant, the alveolar walls can not withstand the constant traction and pressure. This leads to progressive atrophy of their tissue from pressure—that is, it leads to a real disappearance of the elastic elements of the lung. The atrophy begins quite gradually. The partition-walls of the alveoli are first perforated, and then they partly or wholly break down. The neighboring alveoli run more and more into one another, and thus finally arise alveolar ectasis and infundibular ectasis, which can be made out with the naked eye, and which may attain a diameter of five or ten millimetres or more. If single air-bubbles enter the interlobular, interstitial, or subpleural connective tissue, which may happen perhaps in severe fits of coughing, we speak of an interstitial or interlobular emphysema, in distinction from the ordinary vesicular or alveolar emphysema.

The tissue atrophy in the septa of the alveoli affects not only the elastic tissue, however, but it also affects the branches of the pulmonary capillaries in the alveolar walls. The affection of the elastic tissue adds no new conditions to the disturbed functions of the emphysematous lung, which we have just described. The destruction and final atrophy of the pulmonary capillaries, however, is the second important factor in the pathology of pulmonary emphysema, for, with the destruction of so great a part of the vascular area in the lungs, the outflow from the right side of the heart is considerably lessened. There must therefore necessarily be a stasis in the pulmonary arteries and the right side of the heart, and the right side of the heart can overcome the increased resistance only by increased work, and thus in every chronic pulmonary emphysema there finally arise a dilatation and consecutive hypertrophy of the right ventricle with their further consequences.

SYMPTOMS AND COURSE OF THE DISEASE.

General Course of the Disease.—Although a pulmonary emphysema may sometimes, as in whooping-cough, develop in a comparatively short time, still its course is always very chronic. In most cases the origin of the disease is quite gradual, as in all those cases in which an emphysema develops from a chronic bronchitis, an asthma, or as a result of some injurious occupation. The symptoms gradually and insidiously associate themselves with those of the chronic bronchitis.

The symptoms of emphysema usually begin in middle or advanced life, but marked emphysema may occur in youth and childhood. The disease always lasts for years, unless some [fatal] intercurrent disease arises.

The objective and subjective symptoms are due either to the chronic bronchitis, which very often co-exists, or to the emphysema itself. Not only is the bronchitis, as we have seen above, very often the cause of emphysema, but, on the other hand, the development of a chronic bronchitis is greatly favored by the circulatory disturbances in the lung associated with emphysema. Thus the two diseases, emphysema and chronic bronchitis, are closely connected clinically.

Bronchitis causes its well-known symptoms—cough, expectoration, moderate dyspnœa, and a feeling of pressure in the chest. The bronchiectases, which are often gradually formed, especially in the lower lobes, may lend a peculiar stamp to the cough and expectoration (see page 165). Emphysema increases the patient's dyspnœa to a degree which can never be caused by chronic bronchitis alone. The emphysematous lungs soon become incapable of satisfying any extraordinary demands of respiration. Many patients are only slightly conscious of the difficulty in breathing so long as they keep quiet, but whenever they make a trifling physical exertion, go up-stairs, or take a little longer walk than usual, the dyspnœa comes on.

The variations in the intensity and extent of the bronchitis correspond to the frequent and quite marked variations in the patient's feelings. These variations depend upon the condition of the patient, his external circumstances, and the possibility of his taking care of himself; the change of seasons, too, has an influence on him. In pleasant weather many patients live in tolerable comfort, but autumn and winter bring an increase of all their symptoms with the increase in their bronchitis.

The last stage of the disease is characterized by the appearance of a disturbance of compensation in the heart. We have seen above that the cause of the impairment of the pulmonary circulation, and of the resulting hypertrophy of the right ventricle, is the closure of numerous pulmonary capillaries. A further reason for the impairment of the circulation comes from the disturbance of respiration itself, since the influence of the respiratory movements on the circulation is well known. The appearance of a marked disturbance of the circulation may be deferred for some time by the increased efforts of the right ventricle. The cyanosis of most patients is due solely to incomplete oxidation and to the blood-stasis which extends backward from the right side of the heart into the veins of the body. Finally, however, the right ventricle becomes more and more feeble, the stasis in the veins increases, œdema of the extremities and transudation into the various cavities of the body ensue, and after long suffering the patient succumbs to dropsy.

Emphysema is frequently combined in its later stages with other chronic diseases. Pulmonary emphysema with its sequelæ is seldom found at the autopsy as a single lesion, but we discover in the cadaver co-existing disease of the heart, the blood-vessels, or the kidneys. Pulmonary tuberculosis is often a final development in emphysema, but it is usually of the chronic indurated form, and is not very extensive.

Physical Examination. 1. *Inspection.*—In many patients we can detect the disease with considerable confidence at the first glance; we are therefore justified in speaking of an emphysematous habit. The patients are usually quite well nourished, at least in the early stages of the disease, and are often rather corpulent. They appear plump or even somewhat bloated, and their faces are more or less markedly cyanotic. The configuration of the neck and thorax is especially characteristic. The neck is usually short and compressed; the sterno-cleido-mastoid muscles, which have to act as auxiliaries in inspiration, are tense and hypertrophied. The inspiratory contraction of the scaleni may also be seen and felt. The veins in the neck are visibly dilated, and in severe cases are swollen to thick blue cords, and we sometimes see in them evident undulating or pulsating movements. The thorax is rather short, but broad and strikingly deep—the "barrel-shaped thorax." The intercostal spaces are narrow, and the lower ribs move only a little downward. The epigastric angle is therefore obtuse, and sometimes becomes almost a straight line. The respiratory movements are almost always accelerated in severe cases. Inspiration becomes short and labored. The excursions of single ribs are therefore slight, and the thorax is raised rigidly and more as a whole. Expiration is visibly prolonged. There may be a noticeable retraction of the intercostal spaces on inspiration in the lower and lateral portions of the thorax.

This characteristic form of the thorax in emphysema is regarded as a constant inspiratory position of the ribs, and corresponds to the permanent inspiratory dilatation of the lungs. The peculiar rigidity of the thorax is probably due to the changes in the costal cartilages already described, which, according to Freund, are primary. In many cases the emphysematous form of the thorax gradually develops in the course of the disease, but in other cases it seems to depend on some original predisposition (*vide supra*) to the disease.

In conclusion, we must state that the above description corresponds to the typical form of emphysema, from which we may have many deviations. In the paralyzed thorax, for instance, we may meet with a high degree of essential emphysema of the lungs, which has often given rise to errors in diagnosis.

2. *Percussion.*—Percussion gives very decided results in the diagnosis of pulmonary emphysema. We find the inferior border of the lungs one or two intercostal spaces lower than under normal conditions, corresponding to their permanent inspiratory inflation. Clear pulmonary resonance on the right front in the line of the nipple extends to the lower border of the seventh, and sometimes of the eighth rib. On the left front it extends to the fifth or sixth ribs, so that the cardiac dullness is lessened. The area of cardiac dullness can often not be made out at all; or at most, on strong percussion, it is made out in a limited extent as relative dullness. The pulmonary resonance extends on both sides in the back to the first or second lumbar vertebra. This condition on percussion in emphysema,

however, is frequently altered, because other conditions, like passive congestion of the liver, meteorism, and ascites, may be present at the same time, and push up the diaphragm. Thus the detection of emphysema by percussion is made decidedly difficult.

Qualitative changes in the percussion-note may be entirely wanting in emphysema. The pitch is sometimes remarkably loud and deep—the "box-tone" [tympanitic resonance]; but in other cases, especially in the back, we find it somewhat raised. This may depend in part upon the poor vibratory conditions in the rigid chest-walls, but in other cases it is caused by the retention of an abundant secretion in the lower lobes.

The detection of dilatation and hypertrophy of the right ventricle by percussion is in many cases uncertain, because the lungs cover the heart. A positive result can be obtained only by carefully defining the relative cardiac dullness. The frequent epigastric pulsations in emphysema, and also the marked undulating and pulsating movements in the jugular veins, are to be regarded as quite certain signs of dilatation of the right side of the heart.

3. *Auscultation.*—The characteristic auscultatory sign of emphysema is the prolonged expiration. As a flabby rubber band, when it is stretched and then let loose, no longer snaps back quickly and strongly, so the emphysematous lung, when it has been stretched in inspiration, comes back again only slowly. We hear with it a somewhat aspirated, sonorous sound, which plainly exceeds the vesicular inspiratory sound in duration. The vesicular murmur itself often undergoes a modification in pulmonary emphysema. It sounds exaggerated, and very shuffling, or in other cases it is rougher and more indefinite. In a high degree of emphysema the vesicular respiration is sometimes very low and obscure, because the inspiratory current of air is reduced to a small amount in the lungs, which are already excessively dilated. In many cases we hear rhonchi beside the respiratory murmur, dry whistling, buzzing, and creaking sounds on inspiration and expiration. If cylindrical bronchiectases have already formed, we hear, especially over the lower lobes, numerous small and medium moist râles, but no sonorous rhonchi. The rhonchi may wholly conceal the respiratory sounds. With a marked retention of secretion we sometimes hear nothing but a low, suppressed, rattling sound.

In the heart the sounds are usually rather low, because it is covered by the lung. The "accidental systolic sound in emphysema" at the apex, described by some writers, we have heard much less frequently when the valves were intact than we should expect after the statements relating to it. The pulmonic second sound is, as a rule, markedly accentuated, as a result of the stasis in the pulmonary circulation.

The diminution of the expiratory pressure in emphysema may be measured with the manometer, or with Waldenburg's "pneumatometer." The normal expiratory pressure of 110 to 130 millimetres sinks in emphysema to 100 or 80 millimetres. As we should expect, the spirometer shows a diminution of the vital lung capacity, which can be readily explained. The normal lung capacity of about 3,500 cubic centimetres falls to 2,000 or 1,000 cubic centimetres.

OTHER SYMPTOMS IN THE LUNGS AND IN OTHER ORGANS.

In regard to the other symptoms in the lungs we have only a little to add to what has already been said. The intensity of the cough naturally varies in individual cases according to the degree of the existing bronchial catarrh. Many patients are troubled by a dry cough, while others have abundant expectoration. There is nothing characteristic of emphysema in the composition of the latter.

All the kinds of sputa which are found in the different forms of chronic bronchitis are also found in pulmonary emphysema. The dyspnœa, whose predominant expiratory character we have already mentioned, increases in advanced cases to a most marked degree. Sometimes the increase shows itself by the appearance of distinct asthmatic attacks. These are often really to be regarded as a symptomatic bronchial asthma, of nervous origin, but, on the other hand, we must not overlook the fact that a temporary increase of the bronchitis, retention of secretion, and cardiac failure, may also excite attacks of dyspnœa, which can not properly be termed asthma.

The important changes in the heart resulting from emphysema have already been described. The exhausted right ventricle can no longer overcome the increased resistance in the pulmonary circulation. The difficulty of respiration is still greater, from the passive congestion of the pulmonary vessels. The skin becomes still more cyanotic, and finally œdema and general dropsy develop. The failure of compensation is evident by the lessening of the pulse, its increased frequency, and sometimes by its irregularity. The difficulty of an objective examination of the heart in emphysema has been spoken of above.

The appearances of blood stasis in the internal organs are shown especially in the liver and the kidneys. The liver is swollen, and its increase in size (the liver of passive congestion) can frequently be made out by percussion or palpation. The pains in the region of the liver, of which many patients complain, are perhaps sometimes due to the stretching of the capsule of the liver, but they are probably more often muscular pains excited by the frequent coughing.

In the kidneys the effect of stasis is first shown by a diminished excretion of urine. The urine is more scanty in amount, more concentrated, of a higher specific gravity, and of a darker color. It generally gives an abundant sediment of urates, and may contain a small amount of albumen. Microscopically, are discovered a few hyaline casts, and a few red and white blood-corpuscles. It is evident that this diminished activity of the kidneys favors the development of dropsy.

The spleen is not infrequently found congested at the autopsy. The evidence of this, however, is often uncertain during life, for percussion of the spleen is difficult on account of the emphysema, and palpation is difficult from the swelling of the body.

Gastro-intestinal symptoms may be present in emphysema. The appetite seldom remains good throughout the disease. Many patients suffer from chronic constipation; and more rarely there is a tendency to diarrhœa.

Fever is not present in simple pulmonary emphysema. Every fever which exists for a long time depends on other complications, like severe bronchitis, pneumonia, or tuberculosis.

Complications of emphysema with other chronic diseases are frequent. The old opinion that emphysema and tuberculosis, and emphysema and chronic heart disease, were antagonistic to each other is entirely false. These complications are not very rare. We may also mention the complication with general arteriosclerosis and with chronic nephritis, especially the contracted kidney. Among acute diseases we must mention croupous pneumonia, which is not very rare in emphysema, where it must always be regarded as a dangerous combination.

The **diagnosis** of emphysema can be made directly from the results of the physical examination, and usually presents no difficulties. It is obscure only when the patient is not examined till the final stage of dropsy. Then it may be very hard to avoid confusing it with forms of heart disease, like primary hypertrophy, myocarditis, or mitral stenosis, or with contraction of the kidney.

Prognosis.—Pulmonary emphysema of acute origin, like that resulting from whooping-cough and analogous affections, may be recovered from in many cases, but otherwise, as regards the final curability of the disease, the prognosis is wholly bad. The duration of the disease and the intensity of the symptoms are of course very different in individual cases. Here almost everything depends upon the circumstances in which the patient is placed. With sufficient care the disease may be tolerably well borne for many years, but without it the first symptoms of respiratory and cardiac insufficiency appear much earlier.

Treatment.—Since emphysema itself is only slightly amenable to treatment, most of our therapeutic remedies are directed to that accompanying condition upon which the greater part of the symptoms depend—to the chronic bronchitis. If we succeed in improving this, or even in wholly removing it, we always obtain a distinct improvement in all the patient's symptoms. The therapeutic remedies mentioned in the description of chronic bronchitis are therefore of frequent use in emphysema.

In the first place, we must seek the best hygienic conditions for the patient, and remove him from all injurious influences, such as dust, bad air, and work requiring physical exertion. In dry catarrh we should use the alkaline mineral waters, and when there is abundant mucous secretion the balsams, such as turpentine internally and by inhalation. The most valuable expectorants are apomorphine, liquor ammonii anisatus, and senega. Their action, of course, too often fails of the desired result, so that we frequently have to change our remedies. When there is a troublesome cough, disturbing the sleep, we can not dispense with narcotics, like morphine or Dover's powder. If severe dyspnœa comes on, we may try to obtain relief by mustard plasters to the chest, or by immersing the hands and feet in hot water. With asthmatic attacks we may try iodide of potassium, beside the other remedies mentioned for asthma. Here, too, we must finally resort to narcotics.

We must carefully watch the condition of the heart, and use digitalis when there are signs of beginning disturbance of compensation and the pulse grows small and irregular, and this drug may prove very useful. If symptoms of dropsy set in, we may sometimes prescribe diuretic remedies, like juniper-tea, or acetate of potassium, besides digitalis. We also try to strengthen the heart by wine, camphor, benzoic acid, or other stimulants.

Besides the purely symptomatic treatment thus described, the attempt has been made to meet the causal indications in emphysema, and especially to aid the patient in expiration, and thus to improve the power of the lung to contract, where it is possible. To this end Gerhardt has recommended assisting expiration mechanically by compression of the thorax. This compression must be done methodically by another person,* about five or ten minutes every day, by the aid of both hands laid flat on the lower lateral portions of the thorax. The effect of this manipulation in diminishing the dyspnœa and making expectoration easier is in many cases very satisfactory.

The employment of the pneumatic treatment has also become quite general, especially since the introduction of Waldenburg's portable apparatus. The expiration into rarefied air, which meets the causal indication, may procure great relief for the patient in many cases, and sometimes, too, result in an improvement of the emphysema which can be demonstrated on physical examination. Inhalations of compressed air are also employed when there is severe bronchial catarrh. Still, too much must not be anticipated from pneumatic treatment.

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* One of my patients at the policlinic in Leipsic made himself a very simple but very effective apparatus for producing this compression of the thorax on himself by the aid of two small boards, which are firmly fastened together at one end by a long cord. These boards, which are furnished with a piece of wood at this end fitted to the wall of the chest, are laid flat on the two sides of the thorax so that their free ends can project forward some six inches or a foot, and serve as a one-armed lever. By pressing them together the patient himself can thus, without any strain, exert a considerable pressure on his thorax with each expiration.

CHAPTER II.

PULMONARY ATELECTASIS.

(*Compression of the Lungs. Aplasia of the Lungs in Cases of Kyphoscoliosis.*)

Ætiology.—Atelectasis of the lungs is a condition directly opposed to emphysema. While in the latter the lungs are abnormally inflated, in the former they are abnormally collapsed. The air has disappeared from the alveoli and lesser bronchi, and in the most advanced cases even from the larger bronchi. The atelectatic portions of the lung are not altered histologically, but are changed to a firm tissue, deprived of air—the so-called splenization or carnefaction.

The atelectasis of the new-born is due simply to deficient respiration and to the consequent imperfect entrance of air into the lungs. In weak children, who die soon after birth, we often find the lower lobes wholly or in part in a fœtal, uninflated condition—that is, atelectatic. By artificial inflation we can readily expand the lungs to their normal extent.

Acquired atelectasis occurs in two ways. We may mention, as the first and most frequent ætiological factor, the plugging of the smaller bronchi. If a complete closure of a bronchus arises from the accumulation of secretion, as may easily happen in the narrow bronchi of children, the air can no longer enter, on inspiration, into that portion of lung supplied by the plugged bronchus. The air which is shut up in it is gradually absorbed by the blood. The adjacent parts of the lung expand, and the portion that is excluded from respiration collapses, leaving a circumscribed pulmonary atelectasis, usually rich in blood but devoid of air. Such atelectases, in greater or less number and extent, are very often found in the bodies of children who have suffered from severe bronchitis, especially after measles, whooping-cough, or diphtheria. Beside the direct action of the plugging of the bronchus, the weakness of the respiratory movements and the cough, conditional upon the general state of the disease, play a significant part.

The second very frequent and important cause of pulmonary atelectasis is compression of the lung. In all the diseases which diminish the space for the expansion of the lungs, the lungs are pressed together from without to a greater or less extent, whereby the air is pressed out of them. Thus arise the atelectases from pressure in pleuritic effusion, hydrothorax, pneumothorax, in marked cardiac hypertrophy, pericardial effusion, and aneurism of the aorta. Atelectasis of the lower lobes also arises in the same way from great upward pressure on the diaphragm by ascites, meteorism, abdominal tumors, etc.

That form of pulmonary atelectasis which arises from deformities of the thorax is of great practical importance. In severe kyphoscoliosis, the half of the thorax corresponding to the convexity of the vertebral column is much narrowed. The lungs are materially hindered in their expansion, and even in their growth, if the deformity occurs in youth. This is called "aplasia of the lungs," a condition which may give rise to grave results (*vide infra*).

Symptoms.—In the majority of cases the appearances in atelectasis are subordinate to the symptoms caused by the primary disease. This is especially the case in most of the atelectases from pressure, although the most dangerous factor lies in the compression of the lung.

The atelectasis of the lungs developing as a result of diffuse capillary bronchitis, especially in children, can of course not be detected by physical examination until it is of great extent. The respiration, in extensive formation of atelectasis, often shows a very striking and characteristic deviation from the ordinary type, especially when the atelectasis develops in the lower lobes. It is accelerated and labored, and is performed chiefly by the upper and anterior portions of the thorax. In the lower portions we see marked inspiratory retractions, which are caused in part by the external pressure of the air, and in part correspond to the forced contraction of the diaphragm.

Physical examination can, of course, reveal abnormal conditions, especially dullness on percussion, only when the atelectasis is extensive. Dullness, however, is usually hard to make out in children. Auscultation gives signs of existing bronchitis ; and sometimes, too, with more extensive consolidation, there is bronchial respiration. In other cases, as may be easily seen, the respiratory murmur is much diminished or wholly absent. As we can perceive, the physical signs of atelectasis are not really distinguishable from those of pneumonia, especially of lobular pneumonia. In fact, a sharp boundary between atelectatic nodules and nodules of lobular pneumonia in the lung can not be drawn clinically.

Aplasia of the lungs in kyphoscoliosis demands a special description, because it is of great practical significance. Many patients with kyphoscoliosis may live for years without special respiratory disturbance. More careful observation, of course, usually shows a somewhat labored and hurried respiration, but the patients have not paid much attention to it. In other cases the difficulty in breathing is more noticeable. The person affected is incapable of any severe physical exertion; he always feels short of breath, and often suffers from cough and expectoration. In the cases first mentioned, however, which for years have had little or no trouble, disturbances in respiration sometimes come on quite suddenly. They may develop as a result of a mild bronchial catarrh, and they also frequently arise without any special cause, and may attain a very threatening degree. The condition may improve, or it may lead to comparatively speedy death. Examination of the lungs during life usually shows nothing but the signs of an extensive bronchitis. By careful percussion we may quite frequently detect an increased area of cardiac dullness to the right. Sometimes a moderate œdema develops. In such cases the autopsy shows nothing as the cause of death but the changes in the lungs. The lungs are abnormally poor in air, small, and compressed, but in circumscribed portions, on the contrary, emphysematous and expanded. The right side of the heart in the great majority of cases is dilated and hypertrophied. There can scarcely be a doubt, therefore, that the cause of the onset of severe symptoms and the final cause of death is to be sought in the cardiac failure.

Finally, it is worthy of mention that there is a frequent form of mild atelectasis in the lower lobes, which occurs in very sick and bed-ridden patients who usually keep in one position—on the back—as in typhoid fever. On making such patients sit up we hear during the first inspirations exquisite crepitant râles over the lower lobes, which sometimes disappear after a few deep inspirations. Here we have to do with a mild atelectatic condition, with a temporary adhesion of the walls of the alveoli and smallest bronchi.

The **treatment** of atelectasis coincides in great measure with the treatment of the primary disease, and is therefore to be looked for in the corresponding chapters. The prophylaxis of atelectasis, by constant attention to the respiration, is of great practical importance. We should try to keep the patient from lying continually on his back, and we should make him take deep inspirations. The timely use of tepid baths, with shower-baths, is a special preventive of the development of atelectasis, and it may bring about a recovery when atelectasis is already present.

Tepid baths may also be used with care in the treatment of dyspnœa caused by kyphoscoliosis. The condition of the heart, however, deserves especial attention (stimulants and digitalis). The reader is referred to the consideration of the general treatment of circulatory disturbances under Heart Disease. In other respects the symptomatic treatment by expectorants, etc., is the same as in other chronic pulmonary affections.

CHAPTER III.

PULMONARY ŒDEMA.

Ætiology and General Pathology.—We have in pulmonary œdema the exudation of a highly albuminous fluid, usually somewhat hæmorrhagic, not only into the interstitial tissue, but also into the alveoli themselves. The danger of the condition is easily understood from the high degree of dyspnœa which immediately ensues from it. In fact, pulmonary œdema is in many cases a terminal symptom, which comes on in all forms of acute and chronic disease. Many patients are said to die with the signs of pulmonary œdema, especially patients with heart disease, pulmonary and renal disease, and also with other affections of the most different kinds.

In rare cases pulmonary œdema is a transitory symptom. Repeated attacks of it may occur, especially in heart disease and chronic renal disease, and for a time at least, the patient recover from them.

Many erroneous notions formerly prevailed as to the particular cause of pulmonary œdema. The theory was especially wide-spread that arterial congestion in the lungs could excite an œdema, but through the experiments of Cohnheim and his pupils we now know that pulmonary œdema is to be considered the result of stasis. It takes place when the outflow of venous blood in the lung meets an obstacle which can no longer be overcome by the mechanical force of the right ventricle. The obstacle which plays the most significant part here, and which may occur in all possible forms of disease—of course more readily in those mentioned above than in others—is the paralysis of the left ventricle. If the further progress of the blood is much hindered by this, the overfilling of the pulmonary circulation and a consequent pulmonary œdema will necessarily follow, in spite of the most vigorous action of the right ventricle. Every terminal pulmonary œdema depends upon this fact, that the left ventricle is paralyzed in its action sooner than the right.

Inflammatory pulmonary œdema must be distinguished from the pure œdema from stasis just described. It is found in the vicinity of portions of lung infiltrated with pneumonia, it is usually of limited extent, and therefore it is of subordinate importance as a cause of disturbances in respiration compared with the general œdema of stasis.

In very rare cases, as we have seen, an apparently primary acute pulmonary œdema, with a speedily fatal termination, develops in men who were before that apparently perfectly healthy, and the autopsy gives no further explanation of its origin. We probably have to do in these cases with the sudden failure of the left ventricle.

Symptoms.—Marked dyspnœa is the most striking symptom in pulmonary œdema. It is subordinate only when the patient is found in the death agony and is no longer fully conscious.

In pulmonary œdema the respiration is hurried, labored, and rattling. All the accessory muscles of respiration are called into play. The patient usually sits upright in bed. We see on his lips and cheeks a gradually and constantly increasing cyanosis, and we often hear at a distance the moist râles in the larger bronchi.

On examination of the lungs, the percussion is essentially normal, if there is no other disease of the lungs. Sometimes the percussion-note is a little higher in pitch, and often it is slightly tympanitic. On auscultation, we hear everywhere many small and medium moist râles. If the patient can still expectorate, he raises a large amount of frothy, sero-hæmorrhagic sputum. The whole picture of the disease is so characteristic that the condition can scarcely be mistaken.

Treatment.—Since in most cases pulmonary œdema is not so much the cause as a symptom of approaching death, our remedies against it are apt to prove powerless, but it must always be our duty, at least in all cases that are not absolutely hopeless, to try to relieve the pulmonary circulation. From the pathogenesis of pulmonary œdema it follows that we must pay particular attention to the condition of the heart, especially of the left ventricle. Hence we should use energetic stimulants, especially subcutaneous injections of camphor or ether, every half hour or hour. Internally we give camphor, musk, wine, and strong *café noir*. Beside that, we apply strong irritants to the chest, such as large mustard plasters or hot sponges. Sometimes an actual improvement of the respiration, when it has already stopped, may be obtained by a bath with cold douching, where there is marked general cyanosis. If the patient is on the whole strong and well nourished, venesection is sometimes of manifest benefit. Emetics, however, accomplish little, and are even dangerous on account of the collapse which may readily come on after them. An energetic "derivation to the intestines," however, by senna, calomel, or enemata of vinegar, seems sometimes to be really of service. Acetate of lead in large doses, one or two grains (grm. 0·05–0·10), in powder, every hour, employed empirically by Traube, is deserving of trial.

In this way, especially in acute diseases like typhoid and pneumonia, we in fact sometimes succeed in averting the danger of pulmonary œdema by rapid and energetic action. In the cases of œdema occurring in incurable chronic diseases of the heart and kidneys, the remedies employed are of course unfortunately incapable of preventing death.

CHAPTER IV.

CATARRHAL PNEUMONIA.

(*Broncho-pneumonia. Lobular Pneumonia.*)

Ætiology.—Catarrhal pneumonia is not, like croupous pneumonia, a distinct and independent disease, but in the great majority of cases it is a secondary phenomenon, which may develop in the course of acute and chronic diseases of various kinds. It almost always follows bronchitis. The same process which produces catarrh of the bronchial mucous membrane, in its further course invades the bronchioles and the alveoli, and here leads to catarrhal pneumonia.

In every acute or chronic disease, of any severity, the conditions are favorable for the development of an inflammation in the bronchi, and subsequently in the pulmonary alveoli. Everywhere in the air-passages, as well as in the cavities of the mouth and pharynx, saliva, mucus, etc., readily collect if the patient is very ill. Expectoration is imperfect, and the constant dorsal decubitus favors the accumulation of secretion, especially in the lower lobes. The mouth and pharynx are harder to keep clean than under normal conditions. Fungi and bacteria collect in the secretion itself, as well as in the epithelium and particles of food which are left in the mouth, and these excite and keep up processes of decomposition. The inflammatory agents, which are carried into the air-passages with the inspired air, find everywhere favorable conditions for settling and further development. From the upper portions they are drawn farther downward. From the larger bronchi the process invades the finer bronchi, and finally leads to catarrhal pneumonia. We must also bear in mind that many patients who are very ill have difficulty in swallowing. They get choked, and particles of food, with the germs of inflammation clinging to them, are carried into the air-passages. That which a healthy person could easily cough up again remains there, is decomposed, and gives rise to bronchitis and lobular pneumonia.

This is the explanation of the frequent development of lobular pneumonia in the course of diseases which are entirely dissimilar. We observe it especially in all patients with stupor, in severe typhoid, in meningitis, and also in cases of nervous disease, where coughing and deglutition are impaired, as a result of bulbar affections. In all such cases lobular pneumonia is to be considered a complication, and with reference to its origin deserves the name of inhalation pneumonia or deglutition pneumonia. We shall soon see that this form, under some circumstances, may pass into circumscribed gangrene.

Although the ætiological factors just described, which come into notice in the development of lobular pneumonia, have nothing to do with the nature of the primary disease as such, there are, on the other hand, certain infectious diseases which from the beginning are exclusively, or at least mainly, localized in the air-passages. Among these are measles, whooping-cough, and also, to a certain degree, diphtheria, small-pox, etc. In these diseases we very often see lobular pneumonia following bronchitis. In individual cases, of course, it is scarcely possible to decide how far the bronchitis is directly dependent upon the specific cause of the disease, or whether it is merely a complication such as might also occur in any other disease. Lobular pneumonia in diphtheria, and in severe small-pox, is probably for the most part a deglutition or an inhalation pneumonia, the occurrence of which in these diseases may be readily understood. In measles and whooping-cough, however, we may consider that the pneumonia is directly dependent upon the specific agents of the disease, although here, too, the other causes for the development of lobular pneumonia should be borne in mind.

The development of lobular pneumonia from bronchitis is most frequent, as we know, in children and old people. The frequency of catarrhal pneumonia in childhood depends in part upon the limited dimensions of the bronchi. Besides that, however, the diseases in which it is especially frequent—namely, measles and whooping-cough—are children's diseases. In old people its comparatively easy development is due to imperfect expectoration.

The mild cases of primary bronchitis scarcely ever lead to lobular pneumonia, but sometimes in children, and less often in adults, a severe febrile bronchitis may occasion the formation of pneumonic foci. Here, in Erlangen, the author has seen cases which can not be regarded otherwise than as primary catarrhal pneumonia. The inhalation of irritating chemicals may occasion lobular pneumonia as well as bronchitis.

Pathological Anatomy.—It is characteristic of catarrhal pneumonia that the inflammation is circumscribed, being limited to the territory of a small bronchus. Hence the name of "lobular" pneumonia, in distinction from or croupous lobar pneumonia. An atelectasis (*vide supra*) of the affected lobule, arising from the plugging of the bronchus leading to it, often, but not always, precedes the inflammation. The inflammatory process itself consists of the exudation of a scanty fluid, which does not coagulate, and of numerous pus-corpuscles (white blood-corpuscles) into the lumen of the alveoli. The alveoli and smallest bronchi are completely filled by the pus-corpuscles. There are also more or less abundant red blood-corpuscles. The vessels of the alveolar walls are very hyperæmic. The alveolar epithelium is much swollen, and is often thrown off in quite large amounts, the "desquamative pneumonia." It is doubtful whether it also takes an active part in these changes by processes of division.

The inflamed lobules are readily apparent to the eye and the touch by their firm consistence, being devoid of air. Their color at first, from the blood contained in the inflamed part, is a dark red, but later it becomes more grayish. Their lobular boundary is usually easily recognized, but, by confluence of adjacent nodules, large portions of the lung, and even whole lobes, may become infiltrated throughout—generalized lobular pneumonia.

Symptoms.—The primary catarrhal pneumonia which not infrequently occurs in adults usually begins with the same phenomena as a severe attack of acute bronchitis. The patient feels prostrated, has cough, dyspnœa, and pain upon that side which is chiefly affected. The fever is not very high, say 101° to 103° (38·5° to 39·5° C.), and it does not follow any regular course. The expectoration is simply catarrhal, not bloody as in croupous pneumonia. Upon physical examination are found moist râles, seldom marked bronchial breathing, and a dullness upon percussion which is usually only moderate, or else a tympanitic percussion-note over the diseased area. The illness may last two or three weeks, or even longer. There is never a crisis, the fever ending gradually by lysis.

Most of the cases of catarrhal pneumonia develop, as we have already said, secondarily in the course of other affections, hence the symptoms are frequently overshadowed by those of the other disease. There are often found at autopsy a few foci of lobular pneumonia in the lower lobes which gave rise to no clinical symptoms whatever.

In other cases, however, the development of extensive lobular pneumonia is of the greatest clinical significance. The disturbance of respiration, during the patient's life, forms the most striking symptom of the disease, and lobular pneumonia is shown at the autopsy to be the immediate cause of death. The largest part of the fatal cases of measles and whooping-cough, and no very small part of those of diphtheria, scarlet fever, typhoid, or small-pox, are due, in the last instance, to the disturbance of respiration dependent upon lobular pneumonia.

Since a diffuse bronchitis, extending into the finer bronchi, almost always precedes the development of lobular pneumonia, and since it may also give rise in itself to marked disturbance in respiration, there is no sharp boundary to be drawn clinically between diffuse capillary bronchitis and lobular pneumonia. Only the experience, a hundred times repeated, that every extensive capillary bronchitis readily leads to lobular pneumonia, permits us to suspect the latter, with considerable certainty, even if there is no direct clinical evidence of it.

The type of lobular pneumonia seen in childhood is the most characteristic and the most important clinically. It is observed in measles and whooping-cough, and also in weak, atrophic, and rachitic children. The increased frequency of respiration is most striking. The breathing is superficial, but labored, as is shown by the contraction of the auxiliary muscles of inspiration and the play of the nostrils. We also notice inspiratory retraction of the lower lateral portions of the thorax as a result of the incomplete entrance of air. The number of respirations a minute increases in children to sixty or eighty, or even more. In most cases the child has a frequent and apparently painful cough. Expectoration is entirely absent in small children. When it is present it shows no characteristic peculiarities different from ordinary catarrhal sputum. The general condition is always bad. The child is restless, apathetic, and more or less stupid. Its face is usually pale, but often quite cyanotic. The pulse is very rapid, and in small children may attain a frequency of 140 to 180 a minute. Fever is almost always present. It shows no typical course, it is now remitting and now intermitting, and toward evening it perhaps rises to 104° or 105° (39·5°–40·5° C.). The occurrence of such a high rise in temperature is not without value in the diagnosis of catarrhal pneumonia. If in diffuse capillary bronchitis a high fever is present for some time, we may assume with considerable certainty that the formation of lobular nodules has already begun.

Physical examination furnishes direct evidence of the affection of the lungs, but its results are for the most part to be referred to the diffuse bronchitis and not to the lobular infiltration. Auscultation gives the most valuable signs. We hear over the lungs, in a greater or less extent, numerous small and medium moist râles, often quite high-pitched. From these signs, strictly interpreted, we can diagnosticate merely bronchitis, but we may suspect pneumonia with the greatest probability. With very confluent broncho-pneumonia auscultation sometimes gives bronchial breathing and bronchophony beside the râles.

It goes without saying that little lobular nodules, surrounded by normal lung-tissue containing air, give no special signs on percussion. With numerous nodules running into one another, the percussion-note is duller, and there is sometimes tympanitic resonance. The dullness is often first to be made out over a stripe extending along the vertebral column—the so-called "stripe-pneumonia."

Course and Termination.—An attack of extensive lobular pneumonia is usually quite protracted. Even in favorable cases the disease rarely lasts less than two or three weeks, and it may persist much longer. The course of the disease is apt to be irregular, relapses succeeding improvement. The chief danger of the disease lies in this tendency to a protracted course, extending over weeks and months. Many children finally die, not of the lobular pneumonia itself, but from the general weakness and emaciation following the tedious febrile disease. We must remember, however, that complete recovery may sometimes take place quite late in the disease.

The "transition of catarrhal pneumonia to caseation and tuberculosis" is a clinical fact with which physicians have long been conversant. In fact, we often find true tubercular changes in the lungs of children who have died after a tedious illness, as a result of measles, whooping-cough, etc. There can, of course, be no real question, however, of an actual transition from one disease to the other. In such cases we have to do either with an acquired tubercular infection, which has found a favorable soil in an already diseased lung, or (what is probably more frequently the case) the disease of the lung has promoted the development of a previously existing tuberculosis. It is usually weak children, with a hereditary predisposition to tubercle, who succumb to tuberculosis as a result of the above-named diseases. The diagnosis of a developing tuberculosis is not always easy, since it is only rarely that marked phthisical changes—like dullness at the apex, cavities, etc., which can be made out by a physical examination—are found in the lungs. We can usually suspect tuberculosis only from the general conditions—emaciation, persistent hectic fever, hereditary predisposition, or some secondary tubercular disease like meningitis, etc.—especially as absolute proof, from the detection of tubercle bacilli in the sputum, is only rarely possible in children.

The transition of inflammatory lobular nodules to purulent foci (abscesses), or nodules of gangrene, which sometimes happens, especially in small-pox, depends upon the specific malignant property of the agents of inflammation which have entered the bronchi.

If the lobular nodules extend to the pleura, a secondary sero-fibrinous or even purulent pleurisy may develop.

Treatment.—Since we have already mentioned the proper treatment, in our description of the various diseases in which secondary pneumonia is especially prone to develop, we can now be brief. We have also laid repeated stress upon the possibility and the great practical importance of prophylaxis, which is self-evident from a just comprehension of the origin of lobular pneumonia. Besides keeping the cavities of the nose, the mouth, and the pharynx as clean as possible, tepid baths, with cool douching later, are the best means of preventing the development of lobular pneumonia, or of checking its further extension if possible. Cold packs are often used with advantage, but they are more disagreeable to many patients than baths. It is an advantage, which is indeed to be considered in the second rank in comparison with the improvement in respiration, that by both the bath and the pack the febrile temperature is at the same time reduced.

In the treatment of the lobular pneumonia of children a wet pack including the whole body is the best remedy. A sheet is dipped in water, wrung out, and wrapped around the whole of the patient except his head and arms. Outside of this is to be placed a dry woolen blanket or a layer of oiled muslin. The temperature of the water employed should be 68° to 77° (16° to 20° R.). The higher the fever the colder should the water be, and the oftener, say every hour, must the pack be renewed. In milder cases and at night it may be allowed to remain for three or four hours. The beneficial influence of the pack is shown not only by the temperature, but still more by the respiration. If the breathing, despite this remedy, remains unsatisfactory, and the patient becomes more and more stupefied, the treatment must be changed to lukewarm baths of a temperature of 77° to 86° (20° to 24° R.), with douchings of colder water. It is sometimes advisable in severe cases to add to the water employed for bathing or for the wet pack a few handfuls of mustard. The stimulation thus exerted upon the skin is quite marked.

Among external applications to the chest, beside mustard plasters and poultices, dry cups are to be mentioned, which often do very good service in strong, older children, and especially in adults. We never need to use local blood-lettings, however, in catarrhal pneumonia.

Of internal remedies, expectorants are most used. Chief among these are ipecac, senega, and benzoic acid. This last is particularly useful in the lobular pneumonia of children. In strong children the abundant collection of mucus in the bronchi may sometimes be relieved by the administration of an emetic. We should be cautious in the use of narcotics. Stimulants (camphor, wine) must be used in severe cases. Inhalations are quite valueless in lobular pneumonia, yet it is recommended to keep the air in the sick-chamber rather moist by hanging up wet towels, or by sprinkling with water. The room should also be as large and as well ventilated as possible. The general hygienic treatment is of the greatest importance. One of the most important duties, of which the physician must always be mindful, is to keep up the patient's strength by sufficient and proper food. When convalescence sets in, complete restoration to health may be furthered by a suitable residence in the country.

CHAPTER V.

CROUPOUS PNEUMONIA.

(*Lung Fever. Lobar Pneumonia. Fibrinous Pneumonia. Pleuro-pneumonia.*)

CROUPOUS pneumonia is an acute febrile disease of the lungs, very sharply defined both anatomically and clinically. It is one of the most important and most common of the severe acute diseases. It is generally known among the laity by the name of "lung fever." Since secondary pneumonia may develop in the course of various other diseases, like typhoid, small-pox, or diphtheria, and may anatomically have all the signs of croupous pneumonia, but ætiologically be quite distinct from it, we speak of this pneumonia as primary, genuine pneumonia, in opposition to the other forms. The physical signs and the disturbances of respiration are of course the same in primary pneumonia as in secondary. The whole typical picture of the disease, which is so striking, is seen, however, only in genuine croupous pneumonia, of which we will make exclusive mention in what follows.

Ætiology.—The majority of pathologists have now become convinced, from a series of clinical observations, that the cause of pneumonia is to be sought in an infectious agent which enters the lungs and there gives rise to the development of an inflammatory process. This conception of croupous pneumonia as an acute infectious disease, with which alone all the pathological appearances readily coincide, has been more and more verified by all the recent investigations. The precise germ of the disease has not yet, however, been fully determined. Friedländer, indeed, has almost invariably found in the pneumonic lung a special kind of micrococcus, or, to speak more correctly, bacillus, which, singly or in larger numbers, is inclosed in a characteristic shell or capsule—"capsule coccus"—and on cultivation shows a peculiar "nail-like" growth in the culture gelatine; but since quite similar cocci are also found under other conditions, there is no definite proof at present that they are really the pathogenic agents in pneumonia, particularly as the results of experiments upon animals have not been unambiguous.

[Friedländer himself has now agreed that the capsule is simply accidental, probably due to imperfect staining or decolorization. Talamon has produced pneumonia in animals by the injection of the ovoid coccus in pure culture, but has obtained also a similar result with round cocci. Thus the question of the coccus of pneumonia is still undetermined.]

A. Fränkel has lately obtained by means of pure cultivation from the lungs in pneumonia a lancet-shaped diplococcus, or, more correctly, bacillus, to which he ascribes an ætiological significance. This bacillus appears to be identical with that sometimes seen in the saliva, even in that of healthy persons, and it is the cause of the so-called "sputum septicæmia" which can be artificially produced in animals, particularly in rabbits. Fränkel's statements have been confirmed by Weichselbaum, so that the very frequent occurrence of Fränkel's bacillus in lungs affected with pneumonia is indubitable, but an absolute proof that these bacilli are the genuine cause of croupous pneumonia has not yet been furnished. Supposing the infectious nature of pneumonia to be certain, all the other alleged causes may of course be regarded as at most "predisposing causes." The old opinion, which is yet current, that pneumonia is due to catching cold, is to be received with great limitations, for croupous pneumonia is very frequently seen independently of any such influence. In a good many cases it will be found that an exposure to cold immediately preceded the commencement of the disease; but in these instances the cold is probably to be regarded merely as that circumstance which promoted the occurrence of the infection, possibly because of the resultant injury to the bronchial and pulmonary epithelium. This explains the fact that pneumonia is especially frequent in certain classes, for instance, among day-laborers and soldiers. With regard to the so-called "traumatic pneumonia," the state of the case is similar to that of pneumonia due to cold. Patients from the classes who work hard physically sometimes assert that they were taken ill as a result of heavy lifting or of a blow on the chest, but in such cases the subsequent stitch in the side was probably not the result of the injury, but a symptom of the disease which had previously begun to develop.

It is a noteworthy fact in favor of our conception of pneumonia as an acute infectious disease that it may be endemic; which sometimes, though rarely, seems to be quite certain. Extensive endemics of pneumonia, usually of quite a malignant character, have been repeatedly observed in single buildings, especially in barracks or prisons, as well as in tenement houses and other localities.

Pneumonia does not show a decided epidemic character. In a large population sporadic cases occur at any season, but, on the other hand, we may notice a striking increase of pneumonia at many times. Most attacks occur in the winter or spring months, without any necessary relation, however, between the frequency of pneumonia and the occurrence of especially bad, wet, or cold weather.

Individual predisposition plays an unmistakable part in the disease, as we must suppose that it does in all infectious diseases. Like facial erysipelas and acute articular rheumatism, pneumonia is one of those diseases which is prone to attack the same individual several times. There are persons who have had acute pneumonia four or five or even more times in their lives.

We can not affirm with certainty that the liability to pneumonia is due to a special bodily constitution. The strongest and most robust often fall ill with it, and, on the other hand, weak and delicate people, with a tendency to phthisis, are frequently attacked. Drunkards seem to have a special predisposition to the disease, but of course it is exceedingly hard to give any definite statistics upon this point.

Pneumonia occurs at any time of life, most frequently in youth or middle age; but it is by no means rare in early childhood, and also in more advanced years up to sixty or seventy. In general it is observed rather more often in men than in women.

[Defective house drainage seems to be a predisposing cause of pneumonia in some cases. A careful inspection of the local sanitary conditions is desirable, especially where more than one case occurs in a house.]

Pathological Anatomy.—The anatomical process in croupous pneumonia consists in the formation of a hæmorrhagic, coagulable "fibrinous" or "croupous" exudation into the pulmonary alveoli and the smallest bronchi. The development of the exudation usually extends over one or more lobes to their whole extent, and, as the alveoli and fine bronchi are completely filled by the tough exudation, the spongy lung, filled with air, is changed to a firm tissue, devoid of air, except as it is penetrated by the large bronchi.

Since Laennec's day we distinguish three stages in the development of the process. In the first stage (stage of inflammatory engorgement, *engouement*) the lung is very hyperæmic, dark red, and the air contained in it is even now much diminished, but not entirely absent. The alveoli are filled with an abundant exudation, already hæmorrhagic, but still fluid and not coagulated.

In the second stage (stage of red hepatization) the coagulation of the exudation is complete, and the lung has become throughout of the consistency of liver. The hepatized lung shows a somewhat increased volume, and is strikingly hard. The surface of the section has a red and manifestly granular appearance, which is due to the projection of the numerous little fibrinous plugs situated in the alveoli. With the knife we can scrape off a tenacious, creamy, grayish-red fluid from the surface of the section. In the small bronchi, divided by the section of the lung, we find characteristic tubular bronchial casts.

In the third stage (stage of yellow or gray hepatization), which gradually develops from the second, the red color of the surface of the section changes to a yellowish gray, while the contents of the exudation grow poorer in red but richer in white blood-corpuscles. The consistency of the lung is still dense but more boggy. The fluid scraped from the surface of the section is more abundant, milky, and more like pus. We also speak, therefore, of a "stage of purulent infiltration."

The recovery from the process begins as the exudation becomes fluid. The fluid is in part absorbed and in part coughed up.

It is not necessary for every pneumonia to go through all three stages completely. In mild cases the process may stop sooner and go on to recovery.

Concerning the finer histological processes in croupous pneumonia, the primary change is probably to be found in the injury and partial destruction of the epithelium in the alveoli and smallest bronchi, produced by inflammation due to the specific causes of the disease. As in every croupous inflammation of a mucous membrane (see the chapter on diphtheria), a coagulable exudation is formed on the surface of the alveoli and smaller bronchi after the destruction of the epithelium. With the microscope we see the fibrinous net-work of the exudation filling the alveoli. Between its meshes lie numerous red blood-corpuscles—red hepatization. Where there is any of the alveolar epithelium left, we often notice active proliferation—increase and growth of cells. Later on the white blood-corpuscles increase, migrating from the vessels into the exudation—yellow hepatization. The red blood-corpuscles are dissolved unless they are removed by expectoration. The fibrinous exudation is also gradually dissolved as the result of chemical changes not yet clearly understood (peptonization of the albuminous substances ?), and is absorbed like the cells. The regeneration of the missing epithelium comes from the epithelium that has remained intact, and with that follows a gradual and complete *restitutio ad integrum.*

The whole process is comparatively brief, usually running its course in a week or ten days. The most frequent termination is in complete recovery. The other methods of termination, as well as the complications in other organs, will be spoken of in connection with the clinical symptoms. We may here mention simply that the pleura over the affected portion of the lung takes part in the inflammation, without exception, as soon as the disease reaches the periphery, and a fibrinous pleurisy, which is not very intense, may then be recognized; hence the former use of the terms "pleuro-pneumonia" and "peripneumonia."

Croupous pneumonia usually spreads rapidly over a great part of the lung. It is very often quite sharply limited to a single lobe—"lobar pneumonia"—so that the septum of connective tissue between two lobes also forms a strict boundary between pneumonic infiltration and healthy lung tissue; but this boundary is by no means insurmountable, and quite frequently several lobes are wholly or in part attacked by pneumonia. According to all statistics, the lower lobes are more frequently affected than the upper. Isolated disease of the right middle lobe may occur, but it is much rarer than pneumonia of the upper lobes. Of the two lungs, the right is attacked with decidedly greater frequency than the left. We have ourselves seen, in 244 cases, 137 on the right, 86 on the left, and 21 in which both lungs were attacked to a great extent. Simultaneous affection of the lower lobe on one side and the upper lobe on the other—quite a rare occurrence—is termed "crossed pneumonia."

General Course of the Disease.—In spite of the numerous modifications which the course of pneumonia may undergo in individual instances, we can still call pneumonia a typical disease, considering the great majority of cases. The subjective and objective symptoms dependent upon the local affection of the lung usually, but not always, take the chief place among the clinical appearances. In this, pneumonia differs from many other infectious diseases, like typhoid, in which the local affection is subordinated to the general infection.

Pneumonia usually begins quite suddenly. In the majority of cases it starts with a pronounced chill of half an hour to an hour's duration, or at least with a marked and prolonged chilliness. The initial chill may attack the patient while in the best of health. It comes on in the day-time, in the evening, or even in the middle of the night, after a previously quiet sleep. At the same time the patient almost always feels as if a severe illness were beginning.

In other and somewhat rarer cases the beginning of pneumonia is more gradual. A prodromal stage of a few days, or even longer, precedes the severe illness. The symptoms are either of quite a general and indefinite nature, consisting of malaise, dullness, loss of appetite, and headache, or the signs of a pulmonary affection follow the prodromal symptoms more closely. The patient always complains, for some days or weeks before the special severe illness, of cough, some pain in the chest, or slight trouble with breathing. We can not usually be sure whether these prodromata belong to pneumonia or not: a pre-existing simple bronchitis may often furnish the most favorable soil for the development of pneumonia.

In the cases where the disease begins more slowly, the onset of severe symptoms is sometimes marked distinctly by a chill or by sudden thoracic distress. In other cases the patient grows worse gradually, without any abrupt change.

The subjective symptoms in the chest begin, as a rule, a short time after the onset of the disease, on the first day or only a little later. The patient feels on every deep inspiration a stabbing pain in one side. The breathing is shallow, accelerated, and often somewhat irregular. Later on, in severe cases, there is very marked dyspnœa and rapidity of respiration. There is usually an irritating cough from the beginning of the disease. The cough is painful, and hence short, half suppressed, and quite frequent and troublesome. From the second day the expectoration may assume its characteristic viscid, rusty, hæmorrhagic appearance. Physical examination gives on percussion and auscultation the signs to be described more fully below. These are rarely to be found on the first day, but more frequently on the second, and sometimes not till later.

Among the appearances in other organs we may mention, as the most important in diagnosis, the very frequent occurrence of herpes on the lips or on the alæ of the nose. In severe cases there are sometimes marked symptoms on the part of the nervous system—headache, sleeplessness, and delirium. The appetite is usually completely lost. Vomiting is not infrequent, especially in the beginning of the disease. The bowels are usually constipated, but diarrhœa may sometimes be present.

Pneumonia is almost always associated with high fever. The typical character of the disease may be best demonstrated by the behavior of the temperature curve. A corresponding increase in the frequency of the pulse is seen with the increase of the temperature.

The course varies greatly according to the previous individual circumstances, the severity of the disease, and the existence of complications. In the majority of cases, after a comparatively short duration, the disease takes a favorable turn. The beginning of improvement is often sudden, like the onset of the disease. After the symptoms have lasted for some five to seven days, or in rarer cases a shorter or a longer time, at a constant height or with increasing intensity, there occurs in the regular course of the disease a critical decline of the fever, frequently associated with quite a copious perspiration, and with that a very rapid improvement of all the other symptoms. In a short time complete recovery follows.

In other cases, however, the course is not so favorable. The disease may have a fatal termination. In a third small class of cases the disease finally takes a protracted course, which is usually due to the occurrence of abnormal sequelæ in the lungs.

Description of Single Symptoms and Complications.

1. **Symptoms on the Part of the Lungs.**—The chief subjective symptom in pneumonic patients is the characteristic painful feeling in the affected side—the "stitch in the side." This probably always has its origin in the dry pleurisy which accompanies the pneumonia. It is therefore absent in the cases of central pneumonia (*vide infra*). In pneumonia of the lower and right middle lobes the pain is usually more severe than in pneumonia of the upper lobes. One result of the stitch in the side is the difficulty, or even the impossibility, of deep inspiration. Hence the patient's dyspnœa is considerably increased, and this explains the incongruity between the shortness of breath and the relatively slight extent of the pneumonia in many cases. The subjective feeling of difficulty of breathing is present in the majority of cases, and it may become most distressing.

Cough is one of the most constant symptoms in pneumonia, and is usually very painful; hence the patient often tries to suppress it. Expectoration is apt to be very difficult at the onset of the disease, from the viscidity and scanty amount of the sputum; hence very severe and distressing paroxysms of coughing are sometimes observed. The cause of the cough is probably not to be found in the affection of the alveoli, but in the co-existing bronchitis. The irritation of the pleura may also set up a reflex cough. In rare cases cough is entirely absent in pneumonia. Except in the cases of limited or late localization (*vide infra*), we observe this absence of cough chiefly in the pneumonia of old or very weak people, and also, what is of practical importance, in the drunkard's pneumonia associated with delirium tremens.

The pneumonic expectoration is so characteristic that we can often make the diagnosis of croupous pneumonia from this alone. It consists of a very tough mucus, which sticks fast to the bottom of the vessel, and is mixed with blood, and therefore has a more or less intense red or yellow hæmorrhagic color. In individual cases there are numerous gradations. We usually call the pneumonic sputum "rusty," or "brick-red," or of a "prune-juice color," etc. Sometimes it has only a slight reddish or yellowish tint, and sometimes it consists almost entirely of blood. In some cases it assumes a peculiar grass-green color, which is due to a change in the blood coloring-matter, or to a mixture with bile pigment in "bilious pneumonia."

The red color of the sputum, as microscopic examination shows, is due to numerous red blood-corpuscles, still well preserved, mixed with it. They are, however, in part dissolved, and hence cause the uniform red color of the sputum. Separate spots containing much blood are often seen in it. Besides the red blood-corpuscles, the microscope shows numerous partly swollen or fatty degenerated pus-corpuscles. We also see long threads of mucus; sometimes large, round, pigmented cells (alveolar epithelium ?); and finally, in rare cases, ciliated epithelium and crystals of hæmatoidin. Micrococci always, and capsule-cocci frequently (*vide supra*), are to be found in stained preparations, but these have at present no diagnostic value, since nothing certain is known as yet as to their significance.

We have still to mention the bronchial casts as important constituents of pneumonic sputum. Since they are usually rolled up together, we may not find them except by spreading out the sputum in water. They consist of the most beautiful casts of the small bronchi, with many dichotomous divisions, and are a product of the croupous inflammation extending into the bronchi. The casts of the smallest bronchi are sometimes found in the form of "spirals," like those in asthmatic bronchitis (see page 169).

The amount of the pneumonic sputum is, as a rule, not very considerable, but it differs a good deal in different cases. The chemical examination of the sputum has so far given no remarkable results. The amount of common salt contained in it is quite considerable.

In many cases the pneumonic expectoration is absent. Sometimes it is very tough and slimy, but without any admixture of blood; in other cases the sputum is simply catarrhal, when present at all, and then, of course, it comes not from the parts infiltrated with pneumonia, but from the catarrh of the larger bronchi. We often find simple catarrhal sputum, too, beside the characteristic pneumonic sputum.

The pneumonic sputum sometimes is seen in the first or second day of pneumonia, but perhaps not till later. With the beginning of resolution it gradually loses its characteristic appearance. The expectoration then becomes less tenacious and simply muco-purulent, and finally disappears entirely.

Physical Examination.—Inspection shows no especial anomaly in the general contour of the thorax. A marked bulging of the affected side occurs only when there is also abundant effusion into the pleural cavity. The action of the thorax in respiration is very important. With a limited pneumonia we often notice a very marked delay and limitation of motion of the affected side on inspiration. This is due in part to the pain in the side, which comes on with every deep inspiration, and also, in extensive pneumonia, of course, to the physical conditions resulting from the anatomical changes. The unaffected portions of the lung act all the more forcibly.

The acceleration of respiration is very striking, its frequency increasing to thirty or forty, or even more, a minute. We have repeatedly counted sixty respirations in adults, even in cases that finally resulted favorably. The breathing is shallow, but yet in all severe cases very labored. We see the inspiratory contraction of the sterno-cleido-mastoids and scaleni in the neck, and often in the face a marked dilatation of the nostrils on inspiration. The patient usually reclines in bed with the upper half of the body raised. The cheeks and lips are cyanotic. The pale parts about the corners of the mouth are often sharply contrasted with the circumscribed bluish-red coloring of the cheeks.

The results of percussion are directly dependent upon the changed physical condition in the lung, due to the anatomical processes. In the beginning of pneumonia, so long as the total amount of air in the lung remains unaltered, the percussion-note remains clear, but when the elasticity and tension of the tissue in the diseased portion of the lung diminish, the resonance often becomes quite tympanitic. With increased exudation into the alveoli and smallest bronchi the amount of air in the lung constantly grows less, and therefore the percussion resonance becomes very dull, but it usually retains its tympanitic timbre. Since the pneumonic lung is only rarely absolutely deprived of air—for a certain amount is always left in the larger bronchi—the percussion resonance seldom becomes so completely dull—"flat"—as it does, for example, with a large pleuritic effusion. As soon as the absorption of the exudation begins, the volume of air in the lung increases, and the percussion-note becomes clearer, and remains for some time still markedly tympanitic, until the lung has regained its normal tension and clasticity. We have also to note that the intensity of the dullness in croupous pneumonia is sometimes subject to quite marked variations, since the secretion retained in the bronchi is at one time abundant and at another, after expectoration, scanty.

The extent of the dullness or of the tympanitic resonance is naturally dependent upon the extent of the anatomical process. Small and central infiltrations may entirely escape detection by percussion.

Auscultation is of greater importance than percussion in the detection of a beginning or limited pneumonic infiltration. The auscultatory signs depend in part upon the presence of the pneumonic exudation and in part upon the change of the lung into a firm tissue containing air only in the larger bronchi. In the beginning of the disease we hear over the affected portions large or small râles, and very often, too, the characteristic crepitant râle on inspiration discovered by Laennec. This arises because the walls of the alveoli and smallest bronchi, which are cemented together, are torn apart at each inspiration. The crepitation, however, is neither pathognomonic of pneumonia, nor is it heard in every case of pneumonia. With increasing infiltration, bronchial breathing replaces the vesicular. The bronchial breathing in pneumonia is, as a rule, very loud and sharp, and sounds close to the ear. Beside this, we may detect more or less numerous sonorous rhonchi. We may hear a pure, loud bronchial breathing when there is marked infiltration, without any adventitious sounds. With the beginning of the "resolution of pneumonia"—that is, as soon as the exudation becomes more fluid—abundant, loud, moist râles occur, which are usually rather large, and they more or less obscure the bronchial breathing. At this time we frequently hear the characteristic crepitant râle again—*crepitatio redux.* The râles gradually disappear, the respiratory murmur loses its bronchial character, becomes harsh and indefinite, and finally is normally vesicular once more.

We often hear a few rhonchi over the unaffected portions of the lungs, but the respiratory murmur is completely normal over them.

The auscultatory signs just described undergo an important change if the larger bronchi leading to the affected portion of the lung are completely plugged by the secretion, as they are quite liable to be. The respiratory murmur may then almost entirely disappear, and we hear, perhaps, only here and there a few obscure râles. Since such a plugging may be very transitory, we understand why in one day, over the same portion of the lung, we hear first loud bronchial breathing and râles, and then quite obscure and diminished breathing.

Wherever there is bronchial breathing, we hear marked bronchophony. The vocal fremitus persists or is somewhat increased over a pneumonic lung so long as the large bronchi are open, but when they become plugged, the vocal fremitus is weakened or wholly absent.

We have yet to add a few remarks about the parts of the lung in which we may expect first to perceive the physical signs of pneumonia, especially the auscultatory signs.

In the first place, we should never neglect to examine carefully the lateral portions of the thorax and the axillary region when we suspect a developing pneumonia. We often find the first râles here in pneumonia of the lower lobes. The first signs of infiltration may be found in the posterior middle portion of the thorax, and extend downward from this point. Pneumonia of the upper lobes begins just as frequently behind in the apices as in front in the infra-clavicular fossæ. Isolated pneumonia of the right middle lobe also occurs, to be made out in front, on the right, between the fourth and sixth ribs.

Few general statements can be made about the nature or the rapidity of the extension of pneumonia, since in these respects the greatest differences are observed. The infiltration may remain confined to a small portion of the lung, and again it may spread over a whole lobe or more in a short time, even in one or two days. We call the pneumonia, whose constant extension by contiguity we can follow from day to day, wandering pneumonia (*pneumonia migrans*), or, from a purely superficial resemblance, which has given rise to many wrong ideas, "erysipelatous pneumonia." In these cases all the signs of resolution are present in the parts first attacked, while the parts affected later are found still at the height of the disease, or in the beginning of infiltration; but we may also find in the autopsies of wandering pneumonia the parts of the lung affected later in a more advanced stage (gray hepatization) than the parts first attacked, which are still in the stage of red hepatization. Wandering pneumonia is almost always severe and quite protracted.

Pneumonia in rare cases progresses by leaps. Such cases have been termed erratic pneumonia.

2. **Symptoms on the Part of the Pleura.**—As we have already mentioned, every pneumonia which reaches the periphery of the lung is associated with a fibrinous pleurisy, but in many cases this causes no objective symptoms. The stitch in the side in pneumonia, however, is due to the affection of the pleura. In other cases the dry pleurisy is marked by a distinctly audible, and often a very loud, pleuritic friction-sound, and a rub, which may sometimes be felt by laying the hand on the chest wall. We rarely hear the pleuritic friction sound in the beginning of pneumonia, but more frequently in the later stages, and perhaps not till many days after the crisis has taken place.

The cases in which pleurisy with effusion develops as a sequel to pneumonia, which may occur quite early, are more important. We usually have to do with a serous effusion, but in rare cases purulent pleurisy also comes on after pneumonia. Purulent effusions of this kind have been repeatedly found to contain the above-described lancet-shaped pneumonia coccus. In one case, which ended fatally, we saw a hæmorrhagic pleurisy, leading to an abundant effusion of blood into the pleural cavity.

The diagnosis of pleurisy with effusion, complicating pneumonia, is seldom difficult. The percussion resonance is duller than we find it in pure pneumonia (*vide supra*). The respiratory murmur and the vocal fremitus are constantly diminished and finally entirely absent. The symptoms of pressure on the neighboring organs and cavities, the heart, the liver, and the semilunar space (see page 260), are especially important because they are most unequivocal. An exploratory puncture with a Pravaz's [hypodermic] syringe, that has been carefully cleansed and disinfected, gives a very certain and safe method of recognizing pleurisy in doubtful cases.

A moderate degree of pleurisy may somewhat delay the course of the disease, but it has no special significance. Large effusions, however, may decidedly increase the difficulty in respiration. Otherwise the pneumonia may recover, leaving the pleuritic effusion quite undisturbed. In pneumonia of an upper lobe, too, the pleurisy may develop below and lead to an effusion there, while the lower lobe itself remains quite free from pneumonia.

3. **Circulatory Apparatus.**—The pulse is accelerated from the beginning of the disease. In cases of moderate severity its frequency reaches 100 or 120, and, in very severe cases, a still higher rate up to 140 or 160 is seen, and is always a dangerous symptom. This high rate of the pulse does not have as bad a significance in children as it does in adults. The consideration of the quality of the pulse is important. Smallness, weakness, and irregularity of the pulse are of bad omen as symptoms of the onset of cardiac weakness. The attacks of collapse, which sometimes come on quite suddenly in severe cases of pneumonia as in other acute diseases, are especially dangerous. They consist of sudden attacks of weak-

ness of the heart with a very small and frequent pulse. The temperature sinks to subnormal, 95° to 93° (35°–34° C.). The peripheral parts, the nose and extremities, become cool, pale, and somewhat cyanotic. The general weakness is extremely marked. The collapse may be recovered from, especially with timely assistance, but patients may die in it.

The pericarditis that sometimes accompanies a fibrinous or sero-fibrinous exudation is one of the most important anatomical changes in the heart. This can always be explained by a direct conduction of the inflammatory process from the neighboring pleura, and is therefore somewhat more frequent in left-sided pneumonia than in right. It is a complication to be borne in mind. Its diagnosis is seldom difficult if we make a careful physical examination of the heart, but with very severe and extensive symptoms in the lungs a complicating pericarditis may be overlooked.

A slight fresh endocarditis is sometimes found on autopsy, but it has no clinical significance. Diseases of the cardiac muscle, especially fatty and parenchymatous degeneration, may be discovered *post mortem*, but they are by no means frequent. In very weak people, drunkards, etc., who die of pneumonia, we sometimes, indeed, find the heart remarkably flabby, with the right ventricle dilated, but in many cases of pneumonia we find the muscle of the heart at the autopsy perfectly normal. A constant relation between the finer histological changes in the cardiac muscle and the condition of the heart's action during life is, at any rate, not proved.

4. **Digestive Apparatus.**—In severe cases of pneumonia the tongue is dry, coated, and quite like the tongue in typhoid. The appetite is also almost wholly lost from the beginning. Vomiting is not infrequent, especially in the beginning of pneumonia, and it also occurs later. It is observed with especial frequency in the pneumonia of children. Severe symptoms on the part of the intestinal canal are rare. The bowels are ordinarily somewhat constipated, but quite severe diarrhœa is also observed.

The complication of pneumonia with jaundice has a certain significance, but its causes are not always very clear. It is sometimes due to an accompanying catarrh of the duodenum. In other cases the veins of the liver, dilated from stasis, may exert a pressure on the bile-ducts. Slight jaundice has no special significance, and is frequent, even in mild cases; a marked jaundice, however, is seen only in severe cases, especially in drunkards' pneumonia. We call such cases, associated with jaundice, "bilious pneumonia." They have often other severe gastro-intestinal symptoms, like vomiting, diarrhœa, and meteorism, and severe nervous symptoms, like stupor and delirium.

The liver sometimes shows the signs of passive congestion. The spleen is moderately enlarged, especially in severe cases—acute splenic tumor—as in other acute infectious diseases.

5. **Kidneys and Urine.**—The infectious character of pneumonia is also shown by the occurrence of a genuine acute nephritis, which is not especially common, but which still has been repeatedly observed. It begins most frequently on the third to the sixth day of the disease. It is recognized by the presence of albumen, casts, and blood in the urine. The nephritis usually gets perfectly well, but we have once seen it pass into the chronic form. The slight albuminuria which is found in severe pneumonia is, in our opinion, also due to a mild disease of the kidneys, and not to the fever as such (see the section on diseases of the kidney, pages 825 and 837).

Great weight was formerly laid upon the diminution of the chlorides in the urine in pneumonia. In fact, the precipitate of chloride of silver, when we put a drop of solution of nitrate of silver into the urine, may be very slight or entirely absent. The chief cause of this diminution of the chlorides is the small amount of nourishment taken by the patient, but we must also bear in mind the large amount of chloride of sodium contained in the pneumonic exudation.

Great significance was also formerly laid upon the abundant sediment of uric acid (*sedimentum lateritium*—brick-dust sediment) which is often noticed on the day of the crisis. This is in part due to a real increase in uric acid, but in greater part to the fact that the conditions for the deposition of the sediment are especially favorable on that day. The urine is scanty in amount from the abundant perspiration, and is concentrated and relatively very acid. The uric acid contained in it can therefore readily be precipitated in the form of a sediment.

Pneumonia, in common with most of the other acute febrile diseases, is attended with an increased secretion of urea during the disease.

6. **Nervous System.**—As in every severe febrile disease, nervous symptoms of a mild type are very rarely absent in any case of pneumonia. Among the nervous symptoms are the general weakness and dullness, and especially the headache, which is often very intense, and is usually increased by coughing. The onset of more severe cerebral symptoms, especially delirium, is of great importance. We see this chiefly in individuals who have a peculiar predisposition to delirium, and particularly in drunkards. Delirium gives the drunkard's pneumonia (*vide infra*) its characteristic stamp.

While no anatomical basis has as yet been discovered for the above-named symptoms, nor for the worst form of delirium in drunkards, there is an anatomical disease of the brain which is, to be sure, a rare complication of pneumonia, but which yet is undoubtedly related to it: this is purulent meningitis. This complication has been repeatedly observed, especially at times when an epidemic of cerebro-spinal meningitis was prevailing, and also at other times. It is usually hard to make the diagnosis of a complicating meningitis, since its appearances are hidden under the other severe symptoms. The chief points for diagnosis are intense headache, rigidity of the neck, and a stupor increasing to deep coma. In many cases these symptoms are very slightly developed. The termination of meningitis is probably always fatal.

7. **Skin.**—The frequent appearance of herpes in the course of pneumonia is characteristic, and is sometimes of diagnostic importance. It appears from the second to the fourth day of the disease, or sometimes later. Its ordinary seat is on the lips, especially at the corners of the mouth, also on the alæ of the nose, and more rarely on the cheeks or the ear (*herpes labialis*, *nasalis*, etc.). It has been seen only very rarely on other portions of the body beside the face, for example on the forearm and the buttock, and in some cases on the mucous membrane of the tongue. We once saw two eruptions of herpes separated by an interval of several days. In repeated instances, under our own observation, herpes labialis, with a fresh rise of temperature, appeared some days after the crisis had taken place. The especial cause of the development of herpes in pneumonia is unknown to us. It is connected, at all events, with the infectious nature of the disease. and it is accordingly quite analogous to the occurrence of herpes in intermittent and recurrent fever, epidemic meningitis, etc. Other affections of the skin are of rare occurrence. We have seen urticaria in some cases. The jaundice occurring in pneumonia has already been described.

8. **Course of the Fever** (see Figs. 23 and 24).—Pneumonia is, almost without exception, accompanied by a more or less high fever with a very typical course. In the beginning of the fever the temperature rises very rapidly to a high point. Even during the initial chill the bodily heat increases from normal to about 104° (40° C.) and over. There are at present no observations to show whether,

in the cases of pneumonia that begin gradually, there is also a the fever. During the course of the disease the fever shows c tinuous or remitting character, but there is with this a decided deep falls of temperature. Since these at first may easily be taken for the actual occurrence of crises, although later they are proved by the renewed rise in temperature to be a mere temporary decline in the bodily heat, they are termed pseudo-crises. Pseudo-crises are usually seen in the first days of the disease, but in some cases they appear later. They may be repeated once or oftener, so that then the fever has a decidedly intermitting course. These intermitting pneumonias, so called from the course of the fever, have nothing at all to do with malaria, which fact must be especially noted because of the frequency of erroneous statements.

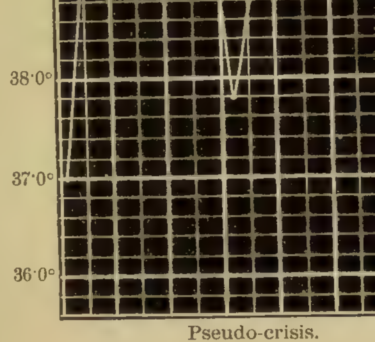
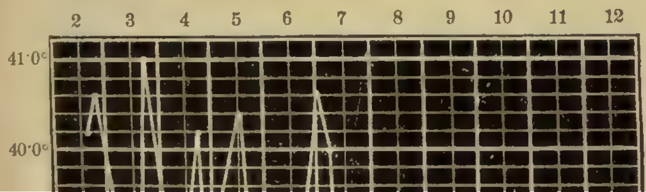


Fig. 23.—Example of the tempe
pneumonia. (Personal

The fever may be decidedly high in pneumonia, often re (40°–41° C.). The highest temperature observed by us was 1 general there is a correspondence between the height of the fe of the c the mo fatal ca with a fever, v and 10 We ex



During the night there is generally a sinking of the temperature with a more or less abundant perspiration, in which as a rule the temperature may reach a subnormal point—96° to 95° (36°–35° C.). The critical decline is often broken by new and slight elevations of temperature, so that on the morning of the next day there may be a definite increase of fever, the so-called protracted crisis. Only in a comparatively small number of cases does the fever end by lysis, in which the temperature goes down like steps. The duration of lysis is seldom more than three or four days at most. A decline of temperature by lysis is most frequent in severe and protracted cases, in so-called typhoid pneumonia (*vide infra*), or in pneumonia migrans.

Although the pathological process in the lungs by no means ends with the crisis, we usually consider the day of the crisis as the last day of the disease. The pneumonia makes no advance after that, but resolution and absorption of the exudation and the return of the patient's strength still take time. Hippocrates knew when the time of the crisis occurs, and that the odd days, especially the fifth and the seventh, have a special significance in regard to it. In an infectious disease that has a typical course there can be nothing strange in the fact that the cessation of fever, to a certain degree, is associated with a definite period of time; but Hippocrates's rule has frequent exceptions. The crisis sometimes occurs on the ninth, the twelfth, or the thirteenth day, and even later, and, on the other hand, there are quite short pneumonias of but one or two days' duration.

In the days following the crisis the temperature, which, as we have said, falls to subnormal, regains its normal height. The pulse, which usually sinks to fifty or sixty during the crisis, when it often shows a slight irregularity, reaches its normal frequency again in a few days. We are quite apt to see, in the days immediately following the crisis, a slight increase of temperature again, 100° to 102° at most (38°–39° C.), but this has no special significance.

The general revolution which the whole condition of the patient undergoes after the crisis is often astonishing. The rapid decline in the respiratory symptoms is especially striking. The return of the affected portion of the lung to normal follows in quite a short time. The expectoration is more abundant but less viscid. It loses its bloody character and is simply catarrhal. In cases which run their course regularly, the signs in the lung on auscultation and percussion become normal again in about five to eight days after the crisis. Abnormally delayed resolution will be mentioned below.

SPECIAL PECULIARITIES AND ANOMALIES IN THE COURSE OF PNEUMONIA.

1. *Pneumonia in Children.*—Beside the common lobular pneumonia there is also a pure, lobar, croupous pneumonia in children, which is by no means so rare as some authors formerly supposed. An initial chill is seen only in older children; initial vomiting, however, is very common in children. In many cases severe cerebral symptoms, like delirium, drowsiness, or convulsions, obscure the pulmonary symptoms at first. The further course, the development of physical signs, the fever, and the complications, are quite analogous to the appearances in adults. The pneumonic sputum is only exceptionally to be observed in children under eight years of age.

2. *Pneumonia in old people* is always dangerous. It may begin suddenly, as in people of middle age, but often it begins more slowly and insidiously. Its course is marked by the speedy onset of great weakness and debility. Nervous symptoms, like delirium, are not infrequent.

3. *Drunkard's Pneumonia.*—We see croupous pneumonia in drunkards with remarkable frequency. The clinical course is characterized by the develop-

ment of delirium tremens, usually in the first days of the disease. The patient's mind is disturbed, he is very restless, constantly tries to get out of bed, and he fumbles night and day with his bed-clothes or articles of clothing. The alcoholic character of the delirium is shown by the patient's whole manner, the tremor of the hands and tongue, and the usually happy character of the delirium. The delirium refers to his favorite beverages, his previous boon companions, etc. He becomes tearful or raving only when forcibly restrained. He may think himself involved in the tavern brawls. The alcoholic delirium is almost always associated with hallucinations. The hallucinations of little moving black figures are especially characteristic. They are either animals, rats or beetles, or little black men, and they give him much trouble. The subjective symptoms of pneumonia are wholly in the background. No delirious patient with pneumonia complains of cough, pain in the chest, or dyspnœa. Careful objective examination is the only thing that confirms the diagnosis. Very often patients with a happy delirium serve to entertain those about them, until suddenly very severe symptoms arise, and they become somnolent and succumb, with the symptoms of pulmonary œdema. The prognosis of every case of drunkard's pneumonia, therefore, is to be regarded as very unfavorable.

4. *Pneumonia in Pre-existing Chronic Diseases.*—Croupous pneumonia is occasionally seen in all forms of chronic disease. It is especially dangerous in persons who are already enfeebled, or afflicted with chronic cardiac or pulmonary disease, like phthisis or emphysema. The pneumonia which often attacks patients with emphysema is clinically important, since emphysema may render the objective evidence of pneumonia very difficult. The croupous exudation does not completely fill the dilated alveoli; hence decided dullness and bronchial breathing are absent.

5. *Pneumonia with Late or Slight Localization—Central Pneumonia.*—Cases are quite often seen whose beginning, course, and subjective symptoms correspond throughout to a croupous pneumonia, but in which the objective evidence of pneumonic infiltration evades the most careful examination. The disease begins with a chill, the fever is high, the patient complains of pain in the chest, which is usually slight, there is perhaps herpes, but only on the fourth, fifth, or sixth days can we make out anywhere any bronchial breathing or crepitant râles. In other cases even the crisis may set in before we are able to localize the pneumonia with certainty. In most of these cases we probably have to do less with an actual late localization than with a central infiltration which nowhere approaches the periphery, and hence is made out objectively only late or not at all. A careful examination of the sputum is of the greatest diagnostic importance, since it sometimes has a perfectly characteristic appearance in spite of the absence or the indefinite character of the physical signs. If there is no sputum, the diagnosis must of course remain very uncertain. In such a case under our own observation a slight pleuritic friction-sound was the only thing heard and that not until the day after the crisis, but this, added to the rational signs, made the diagnosis of pneumonia certain.

6. *Typhoid Pneumonia—Asthenic Pneumonia.*—By typhoid pneumonia we mean those cases in which, beside the local pulmonary symptoms, which may be either slight or well marked, there are remarkably severe general symptoms. The cases do not often begin as suddenly as ordinary pneumonia, but more gradually, like typhoid. Even at first the general symptoms, such as great dullness, loss of appetite, or headache, predominate over the thoracic symptoms. At the height of the disease there is a decided typhoidal state, stupor, delirium, a very dry tongue, great general weakness, and also enlargement of the spleen, and fre-

quently mild jaundice, albuminuria, etc. Such cases are to be regarded as pneumonia with an unusually severe general infection. They sometimes occur in epidemics. It is said that pneumonia of the upper lobes shows a somewhat more frequent tendency to severe nervous symptoms than pneumonia of the lower lobes. Recovery from this typhoid or asthenic pneumonia, which may last two weeks or more, often follows by lysis. Typhoid pneumonia is by no means a sharply defined disease. The term serves merely as a short name for the grave constitutional disturbance. Clinically it is impossible to distinguish it sharply from pneumonia migrans, bilious pneumonia, and other forms.

The croupous pneumonia which comes on in the course of genuine typhoid is ætiologically quite different, but in diagnosis it is often very hard to distinguish it. Here we have typhoid with a secondary localization in the lungs—"pneumotyphoid" (see page 13)—which is rare. In the cases first described, however, we had a pneumonia with very marked general infection—"typhoid" symptoms. The only things which render an accurate differentiation of the two diseases possible in such cases are observation of the whole course of the disease, attention to all the single symptoms, such as the intestinal symptoms, and roseola, and the ætiological relations, so far as we can learn them.

7. *Pneumonia with Delayed Resolution.*—While the resolution of pneumonia is complete, as a rule, in three days to a week after the occurrence of the crisis, there are cases in which this process demands a much longer time. Not infrequently, and particularly in severe cases of pneumonia, one sees after the crisis a surprisingly rapid disappearance of all the physical signs, while, on the other hand, recovery is sometimes remarkably slow in apparently mild cases; but this rule is of course not universal, and not without exceptions. The course of the disease is often enough precisely the opposite. Just what are the conditions upon which the rapidity or the slowness of resolution depends we do not know. Sometimes unfavorable constitutional conditions, such as anæmia, debility, phthisical tendencies, and kyphoscoliosis appear to delay resolution. Sometimes, on the other hand, no such explanation can be found. It seems to us that at certain times all the cases of pneumonia exhibit more of a tendency to delayed resolution than at others, so that it is not impossible that there are variations in the pathological process itself. Perhaps, however, secondary pulmonary diseases play a part in the phenomenon.

With regard to the symptoms of delayed resolution, there are various forms. In the first place, we see cases where the crisis takes place in the usual way, and the temperature thereafter remains permanently normal. The patients perhaps feel quite well, and are troubled little by thoracic symptoms; nevertheless, the dullness upon percussion remains unchanged, or diminishes at best very gradually, and the bronchial breathing or subcrepitant râles can still be heard. All the signs diminish very slowly, sometimes occupying several weeks in their disappearance, and then complete recovery ensues. In other cases there is no distinct crisis, but the fever continues, although lower than at first, and the physical signs remain to a greater or less extent. At the end of two or three weeks, or even still later, the fever slowly ceases, and thereupon normal resonance and vesicular breathing gradually return. Still another course, which differs somewhat from those already described, and of which we have repeatedly observed typical instances, is the following: The patients remain free from fever for about a week after the crisis appears. During this time the dullness remains, and the bronchial breathing, although not very loud, is constant. Then a moderately intermitting fever comes on again, with an increase of temperature to about 102° or 103° (39°–39·5° C.). This fever may last from two to four weeks, or even longer. Only occasionally, if ever, do we hear a râle over the affected portion of the lung. A moderate contraction of the affected side gradually appears. Then the resonance slowly becomes clearer, and the respiration becomes louder and vesicular again. The fever disappears, and complete recovery finally takes place. In many other cases of delayed resolution there is also a marked absence of râles and a mild degree of contraction. The differentiation from secondary pleurisy is here often very difficult, except by tapping the chest. Again, it is not very exceptional to observe delayed resolution and secondary pleurisy simultaneously in the same patient.

If there is really merely delay in the resolution and no distinct secondary disease (see below), complete recovery will almost always come at last.

8. *Termination of Pneumonia in Phthisis, Contraction of the Lungs, Pulmonary Gangrene, or Pulmonary Abscess.*—Three terminations of pneumonia are ordinarily mentioned as unusual and anomalous—the termination in "chronic pneumonia," in gangrene, and in abscess.

Concerning the termination in chronic pneumonia, we have already mentioned a process belonging here, the termination in contraction with ultimate recovery; but in rare cases the contraction is persistent. From the complete lack as yet of careful post-mortem examinations, we can not give fuller details as to the anatomical process in these cases (hyperplasia and retraction of the inter-alveolar connective tissue ?).

By the "termination in chronic pneumonia" we usually mean a termination in phthisis—that is, tuberculosis. With our present conception of the two diseases there can, of course, be no question of an actual transition from one to the other. Accordingly, where a clear case of phthisis develops as a sequel to genuine croupous pneumonia—which, however, is quite rare—either the pneumonia develops upon a pre-existing tuberculosis, or tuberculosis develops after the pneumonia in a person predisposed to tubercle.

The transition from pneumonia to pulmonary gangrene is sometimes seen, especially in old and weak individuals. Here, too, in our opinion, a new infection, with a foul and putrid substance, must always take place, and this excites the gangrene. The previous pneumonia furnishes only the occasion for the development of gangrene, and perhaps prepares the soil for the agents of decomposition. The development of gangrene is appreciated clinically from the change in the sputum.

The transition from pneumonia to pulmonary abscess is very rare. We can not decide whether a specific further cause is also needed for this, or whether the pneumonic process may exceptionally go on into the formation of abscess. The transition to an abscess may be recognized by the character of the sputum, which contains fragments of pulmonary tissue, such as elastic fibers, beside abundant pus. Besides that, we find, on microscopic examination of the sputum in abscess, sometimes scales of cholesterine (Fig. 25) and hæmatoidine crystals; the latter may be so abundant as to give the expectoration a brownish color. Sometimes we have seen a peculiar greenish color of the sputum. The signs of a pulmonary cavity are found if the abscess bursts.

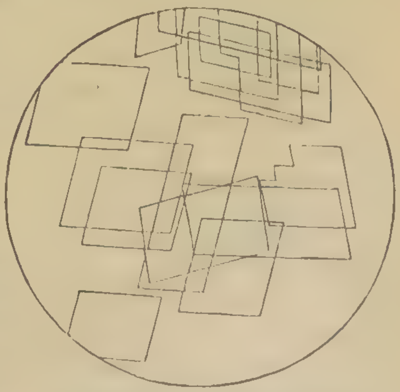


FIG. 25.—Cholesterine crystals.

Diagnosis.—No special remarks on diagnosis need to be added to the description we have given of all the important symptoms which may occur in croupous pneumonia. We should pay particular attention to the sudden onset, the characteristic sputum, the physical signs, the frequent occurrence of herpes on the face, and finally to the whole course of the disease, especially to the temperature curve. We will discuss the differential diagnosis between pneumonia and pleurisy with effusion more fully in the description of the latter affection.

Prognosis.—Croupous pneumonia belongs in general to the benignant infectious diseases. The great majority of cases, in previously strong and healthy individuals, run a favorable course, and end in complete recovery. On the other hand, pneumonia brings a number of perils with it, the knowledge of which should always make us cautious in giving a prognosis.

A grave danger lies in the extension of the process. If the advance of the pneumonia can not be stopped, if the whole of one lung is involved, and, besides that, a great portion of the other lung, the diminution of the respiratory surfaces forms a factor which may give rise to a fatal termination.

A further danger lies in the onset of certain complications. An intense pleurisy, with effusion, especially if purulent, causes greater difficulty in respiration, and thus increases the danger. Still worse is a sero-fibrinous or purulent pericarditis, which, in not very rare cases, is revealed at the autopsy as the special cause of death. We must note, however, that recovery sometimes finally takes place in spite of an empyema or of a purulent pericarditis. The complication with a purulent meningitis, which is fortunately very rare, is probably invariably fatal.

The dangers of general infection (or perhaps a more correct expression would be general intoxication) occupy a subordinate place in pneumonia, on the whole, in comparison with other infectious diseases, like typhoid, but the constitutional state is important in the so-called typhoid, asthenic pneumonia. Sometimes unusually severe and malignant forms of pneumonia, with a high mortality, occur in epidemics and endemics, but these cases are often characterized by the extent of the local process, or by the development of the dangerous complications mentioned above.

The individual circumstances of the patient play the most important part in the prognosis of pneumonia. While a constitution that was previously healthy and uninjured usually survives the disease, one that was previously weak and diseased readily succumbs. In this fact lies the danger of pneumonia in old, weak, badly nourished persons, and in persons with a pre-existing emphysema, kyphoscoliosis, heart disease, etc. In this, too, lies the great danger of every pneumonia in drunkards. Since the nervous system is much affected by chronic alcoholism, we very often see outbreaks of delirium tremens in pneumonia, which come on very readily. In like manner the other nerve-centers are weakened and incapable of resistance, especially the regulatory centers for the heart and respiration. Hence we can understand why even moderate drinkers, though previously strong and well to all appearance, succumb to pneumonia from insufficiency of the heart and lungs.

If we ask upon what symptoms our prognosis in any given case should depend, we must reply that no single factor can be given especial prominence. Chief stress must always be laid upon the state of the lungs and the respiration, but attention must also be given to the general condition, the heart's action, the height of the fever, etc. The worst dangers of pneumonia have just been mentioned.

Of the abnormal terminations of pneumonia, contraction gives the best prognosis, but recovery, or at least a marked subsidence of all the symptoms, may sometimes take place after gangrene and abscess.

Treatment.—Many of the milder cases of typical pneumonia need no special active treatment when the disease on the whole takes a favorable course. Most cases get well under, or, we can almost say, in spite of any treatment. From the method of treatment by large bleedings, which was formerly customary, and also by certain drugs which are even now occasionally used—we refer especially to veratrine—we should expect an injurious effect, and not a favorable one; and yet even with such treatment many cases of pneumonia recover.

[In robust individuals, with a full, hard pulse and great oppression of breathing, venesection affords a prompt relief to be obtained in no other way.

In general, the main indications for treatment are, as in other acute self-limited diseases, to support life until the disease has run its course.]

We do not know a remedy which can in any way exert a favorable influence upon the pneumonic process itself. The treatment of pneumonia must therefore be purely hygienic and symptomatic, but in this respect it can accomplish very much.

The symptoms which are usually prominent in every pneumonia, even in the milder cases, and of which the patient is especially desirous to be relieved, are the pain in the side, the troublesome cough, and the difficulty and distress in breathing. Since the respiratory symptoms, as we have seen, are partly due to the pain, as this improves the patient's breathing often undergoes a decided improvement. For the pain, we may first mention a number of external applications to the skin on the affected side. An ice-bag sometimes gives marked relief. Many patients can not bear this, but prefer warm poultices or cold wet compresses. The application of mustard plasters or dry cups to the skin may be of advantage. Subcutaneous injections of morphine, however, are the most effective remedy, and are often indispensable. There is no reason why we should not use this remedy, with care and in moderate doses, for the relief of pain; and, as the disease is of short duration, we need not particularly fear the morphine habit. Small doses of morphine, subcutaneously or by the mouth, may also be required to alleviate the cough.

Local blood-letting is a remedy the action of which can not be explained physiologically, and yet experience has shown that it is of undoubted advantage. The relief which many patients feel after the application of ten or twelve leeches to the affected side is very striking; but we should prescribe them only when there are severe symptoms at the beginning of the disease, and in persons who were strong and healthy before the attack. Wet cups accomplish the same thing, but the effect is somewhat more powerful, and hence they should be reserved for strong and robust persons, such as laborers.

The tepid bath serves as the most effective means of improving the respiration, of aiding expectoration, and of stimulating and refreshing the whole system. We hold it useless, if not injurious, to give a patient baths if the disease is progressing favorably, for almost every bath has some disagreeable feature. These disadvantages, however, are always less, in severe cases, than the benefit and improvement which baths give the patient, and which most patients recognize with gratitude. The main point is that the patient should make no physical exertion while in the bath, that he should be lifted into it, held and supported while in it, and lifted into bed again after it. Since the baths are given primarily not on account of the fever, but to improve the respiration, and because of their favorable influence on the nervous system, their temperature need not be especially low. We give them from 85° to 90° (24°–26° R.); somewhat warmer with sensitive and weak people, and cooler, down to 77° or even 72·5° (20°–18° R.), with strong people, or with very high fever or severe nervous symptoms. We need not employ more than two or three baths a day, and at night we employ them only when there are threatening symptoms. The favorable action of the baths is especially seen in the great relief and refreshment that the patient feels. The respiration is quieter and slower, but deeper. The patient often falls into a quiet sleep after the bath.

Antipyretics are seldom useful in pneumonia. Even if the temperature rises very high, up to 106° (41° C.), cool baths are sufficient to avert the dangers of an increase of bodily heat. If any antipyretic must be used, antipyrine is to be preferred.

Expectorants are much used, especially since in practice we seldom can avoid giving some internal remedy. Among the most useful are infusion of ipecacuanha, benzoin, liquor ammonii anisatus, and tartar emetic, once held to be a necessary specific against pneumonia (!). Finally, digitalis may be indicated under some circumstances with a weak and frequent pulse, either in powder or in an infusion of about 1 to 100.

We must give the greatest attention in all severe cases to keeping up the patient's strength. We must therefore take care to furnish an easily digestible but nutritious diet. Meat in small pieces, cut up fine or scraped, may be freely allowed if the patient has an appetite for it, but during the first days of the disease we are usually confined to giving soups, milk, and eggs. As soon as the signs of more marked general weakness appear, and the pulse becomes smaller, we must order stimulants, wine, strong *café noir*, or, still better, ether or camphor. The latter is best given subcutaneously in severe cases, dissolved in olive-oil, in a ratio of one to four. If the patient swallows well, we may give it in wine, two grains (grm. 0·1) every one or two hours.

We have yet to make some remarks upon the very extensive use of large amounts of alcohol in pneumonia. Without doubt a free use of alcohol is necessary in drunkards, especially when delirium tremens is beginning or is already pronounced. Since the withdrawal of any poison that is taken habitually, like nicotine or morphine, may excite the severest symptoms, the sudden withdrawal of alcohol from drunkards may have the worst results, while, if we give an abundant supply of the stimulant to which the nervous system is accustomed, we sometimes succeed in avoiding the onset of severe nervous symptoms, like collapse and failure of the heart and respiration. It is quite a different matter with patients who before their illness have not been accustomed to take alcohol at all, or who took it only in small amounts. It may be true that in these cases small amounts of wine may have a stimulating and exciting action, although we never could satisfy ourselves of the often praised influence of alcohol upon the action of the heart. We hold it, however, unjustifiable to force large amounts of alcohol indiscriminately upon every patient with pneumonia, perhaps in spite of great resistance on his part. Why should we expect sick persons to bear doses of alcohol which have only bad results on healthy men unaccustomed to them?

[Few American physicians of any experience will accept the reasoning of the author on the employment of alcoholic stimulants in those not accustomed to their use.

The toxic effects of alcohol are as undesirable in pneumonia as in any other disease, but there are few affections in which so great tolerance is shown for this agent. The chief indications for its exhibition are derived from the pulse and the first cardiac sound at the apex. A flagging heart calls for alcohol, the effect of which on the symptoms and on the circulation is to be carefully watched; the quantity is to be diminished, increased, maintained, or the agent is to be omitted entirely, according to the conditions present in the individual case. I am no advocate of indiscriminate alcoholic stimulation; but I believe that lives have been frequently saved in the past, and will be saved in the future, by the judicious and sometimes extremely free use of this class of remedies in acute pneumonia. Inhalation of oxygen has won a prominent place in the treatment of severe pneumonia of late years, and cases are reported in which life seems unquestionably to have been saved by it. In a considerable number of cases the editor has seen only one which could come under this class. He has seen, however, marked temporary improvement in pulse and respiration, with diminution of cyanosis and induction of relatively quiet sleep. But the oxygen must be given in large quantities, sometimes continuously. If its inhalation fatigues the patient, the gas may be allowed to escape before the mouth or under the nose of the patient. The objection to the efficient use of oxygen in some cases is the expense of perhaps some hundreds of gallons a day at five cents a gallon.]

The treatment of complications follows the ordinary rules which have been given under the individual affections. We must also mention that, in delirium tremens, tepid baths with cold douches sometimes have a very good effect. Besides this, we may try subcutaneous injections of strychnine, seven to fifteen minims of a one-per-cent. solution, once or twice a day. We can not wholly dispense with narcotics, such as morphine and chloral, but we must warn against the imprudent use of large doses of chloral, above thirty-five grains (grm. 2·5).

CHAPTER VI.

TUBERCULOSIS OF THE LUNGS.

(*Pulmonary Phthisis. Pulmonary Consumption.*)

General Pathology and Ætiology of Tuberculosis.

Ever since Bayle, in 1810, demonstrated the extensive distribution of peculiar nodules in the various organs, and their relation to pulmonary consumption, few questions have so taxed clinical observers and pathologists as those relating to the cause and nature of tuberculosis. Harmony could not be reached, however, so long as the criterion for the decision of the questions was sought in the presence of definite anatomical changes, which were regarded as specific of tuberculosis. Laennec considered the peculiar change in the tubercular products, which later was named caseation by Virchow, to be characteristic, and called everything tubercular where it was found. He distinguished the isolated tubercle from diffuse, tubercular, cheesy infiltration. Thus Laennec recognized that many processes were allied whose affinity was often disputed afterward, and has only recently been established, such as the affinity between "scrofulous" enlargement of the glands and tuberculosis. Another opinion became quite prevalent, after Virchow discovered that precisely the same anatomical process as tubercular caseation was also found in inflammatory products, which were certainly not tubercular, and in cancerous ulcerations. Hence Virchow made a sharp distinction between tubercle and those new growths and inflammatory processes which had become cheesy. The anatomical criterion of tuberculosis was, in his view, the presence of the miliary tubercle, a gray nodule, the size of a millet-seed at the largest, made up of cells like lymph-corpuscles. The study of the finer structure of the miliary tubercle was now pushed most eagerly by Wagner, Schüppel, Langhans, and others, but they were unable to reach perfect harmony regarding its origin and significance.

As long ago as 1865, however, a discovery was made which pointed unequivocally to the only way which could lead to a correct knowledge of tuberculosis. It was the fact, discovered by Villemin, that tuberculosis can be produced artificially by inoculating healthy animals with small amounts of tubercular and cheesy substances. Although doubted and misinterpreted at first in various quarters, the fact that tuberculosis can be transmitted, and consequently the fact of its infectious character, must now be regarded as proved beyond a doubt. In the general change which our opinions upon the nature of infectious diseases have undergone, especially in the last few years, the existence of a specific, organized cause of tuberculosis, too, had to be assumed. Klebs, and later Cohnheim, had already without reserve defined tuberculosis as a specific, infectious disease and, sooner than we dared to hope, R. Koch discovered the special carriers of the disease in the shape of the tubercle bacilli, in the year 1881. The definition of tuberculosis no longer rests upon any external anatomical character. Every disease is tubercular which is excited by the pathogenic action of a specific kind of bacteria, the tubercle bacilli discovered by Koch.

The pathogenic bacteria of tuberculosis belong to the group of bacilli. The tubercle bacilli are rod-like, of small diameter, slightly rounded at their extremities, and either straight or somewhat bent. Their length is perhaps a fourth or a half that of the diameter of a red blood-corpuscle. In the interior of these rods it is not seldom possible to distinguish very minute colorless spots which are probably to be regarded as endogenous spores. The tubercle bacilli have no independent motion whatever. Their reaction to certain coloring matters is very characteristic, and of the highest importance with regard to their recognition (see below). We know with absolute certainty that the tubercle bacilli are always present in all the different forms of pulmonary tuberculosis, both in the lung itself and in the expectoration (*vide infra*), and also in tubercular diseases of other organs, like the brain, the intestines, the spleen, the liver, and the kidneys, and also in "scrofulous lymph-glands," in "fungous" diseases of the bones and joints, and in the so-called lupus, which is nothing but a local tuberculosis of the skin. Precisely the same bacilli are also found in the "spontaneous" tuberculosis of animals, such as monkeys, puppies, and guinea-pigs, and in every tuberculosis that is artificially produced in animals by inoculation. Finally, by the discovery of tubercle bacilli in the "pearly distemper" of cattle, the identity of this disease with tuberculosis—an identity which had already been established by experiments in inoculation—was confirmed anew.

Koch, by his successful "pure cultures" and inoculations with the cultivated bacilli, has established the fact that these bodies, known as tubercle bacilli, are to be regarded as organized, and as the special cause of tuberculosis. Bacilli coming from any fresh product of tubercular disease may be cultivated at a constant temperature of 98° to 100° (37°–38° C.) upon blood-serum which has been stiffened by heating, and upon several other artificially prepared soils. In this cultivation they show certain characteristic properties in their growth, which can not be fully described here, and they multiply to an unlimited extent. In this way we can keep up perfectly "pure cultures" of tubercle bacilli. Inoculation experiments tried with them on all sorts of creatures always give a positive result. The animals fall ill, lose flesh, and finally die, and at the autopsy we find undoubted tubercular disease of the internal organs, which may be more or less extensive. The most instructive inoculations are those into the anterior chamber of the eye in puppies or guinea-pigs, which were first tried by Cohnheim and Salomonsen. After an incubation of two or three weeks we see here very plainly an eruption of the first nodules of tubercle in the iris, and the tuberculosis spreads to the other organs of the body later.

ÆTIOLOGY OF TUBERCULOSIS IN MAN.

The distribution of tubercle bacilli must be remarkably extensive, for tubercular diseases occur in almost every country on earth. The predisposition of mankind to the disease is also very great, and thus we understand the frightful fact, which statistics show, that nearly one seventh of all deaths are from tuberculosis! It has neither been proved, nor is it probable, that tubercle bacilli multiply outside of the human body, like the bacilli of splenic fever, since they can develop only in a constant and uniformly warm temperature between 85° and 104° (30°–40° C.). We must therefore regard them as true parasites, which can live—that is, which can propagate and multiply—only in the bodies of animals, but they seem to preserve their virulence and their ability to multiply outside of the body for a long time. Phthisical sputum may be used for inoculation with success, even if it has been dried for several weeks. The tubercle bacilli also resist most chemical reagents, like nitric acid, very decidedly.

If the body becomes infected, then, with tubercle bacilli, it is always probable that they have come from some other individual—man or beast—with tubercular disease. We need not mention how numerous the opportunities for infection may be, considering the present general distribution of tuberculosis. The chief stress in this respect is to be laid upon the expectoration of phthisical patients, which contains the bacilli. This dries on the floor, on the linen, and on other objects, and then the small particles which contain the germs of infection are carried off by the air. The view most generally held is that the material which contains the bacilli or spores is taken into the body, in the majority of cases, along with the inspired air. This is probable, because, in most cases, tuberculosis has its starting-point in the air-passages, the lungs, or larynx. Inoculation experiments show that the first extension of the process depends upon the point of inoculation. If it be in the anterior chamber of the eye, the first nodules appear in the iris, as we have said; if it be in the abdominal cavity, we have first a tuberculosis of the peritonæum; if the infectious matter be inhaled, we have first a tuberculosis of the lungs. For several years Tappeiner and others have experimented with inhalations of powdered tuberculous sputa at the Munich Pathological Institute. Pulmonary tuberculosis could always be produced by these inhalations in the animals experimented upon. Hence it seems very probable that, in tuberculosis in man, the infectious matter is taken directly into the air-passages by the breath. In this way it sometimes, though rarely, attacks the upper air-passages, as in primary tuberculosis of the nose, the pharynx, and the larynx, but more frequently it affects the deeper portions of the respiratory apparatus, as in primary tuberculosis of the lungs and bronchi.

We must also consider other methods of infection, first among which is the possibility of infection of the intestinal canal, from swallowing the infectious material. The transmission of tuberculosis from domestic animals to man plays a part in this connection which perhaps is not unimportant. Since the pearly distemper in cattle is identical with tuberculosis in man, the use of the flesh of such animals as food furnishes a possible means of infection. It is a still more important circumstance, however, that, when pearly nodules are present on the udder, the milk of the diseased animal may be polluted by tubercle bacilli, and that the use of such milk as food, when it is uncooked, certainly involves the danger of the transmission of tuberculosis. Primary tuberculosis of the intestines, however, does not seem to be very frequent, probably because the tubercle bacilli which have been swallowed are usually destroyed in the stomach.

In some cases the tubercular infection may probably arise from little fissures and excoriations of the skin. In this way we get either a local tuberculosis of the skin, like lupus, or the bacilli are carried by the lymphatics to the neighboring glands of the neck or axilla, establish themselves there, and set up a tubercular disease in them. In conclusion, we have still to mention that the apparently primary appearance of tuberculosis in the genito-urinary apparatus leads us to think of the possibility of an infection of the genital or urinary organs.

Considering the wide distribution of the tubercle bacilli, and the many chances for infection, it seems wonderful that in spite of it so many men escape the disease. One factor, which has been already mentioned by Koch, must be borne in mind, however, and that is the extremely slow growth of the tubercle bacilli. This is the reason why the bacilli do not always remain in the body, but in many cases are eliminated again before they have gained a definite foothold.

Individual predisposition, however, is another factor which perhaps is still more important—a factor which we can not well explain, but which we can not get on without, at the present time, in the pathology of many infectious diseases. In our conception of most of the other infectious diseases, as well as of tuberculosis, we must assume provisionally an unequal predisposition to disease in different individuals. Only a part fall ill of all who are exposed to the action of the poison. These are persons in whom it is particularly easy for the poison to establish and propagate itself.

We have long considered people with a generally feeble physical constitution as especially liable to tubercular disease. We speak of a "tubercular habit" (*vide infra*). In this connection we must, of course, remember that much that we once regarded as merely the signs of a special predisposition to disease is really the expression of a disease that already exists. If, for example, we once affirmed that "scrofulous" children had a special predisposition to tuberculosis, we know now that the so-called scrofulous diseases of the mucous membranes, the lymph-glands, and the bones, are the results of an existing tuberculosis; or, at least, that this is so in a large number of cases.

We now believe that many evil influences which were once thought to be causes of tuberculosis act only in increasing the predisposition to the disease. Insufficient food, bad air, severe illness, childbirth, want, and care—these alone, of course, can never produce tuberculosis; but we can easily imagine that the body which has become weakened affords less resistance to the injurious influence of the tubercular poison than does the strong and healthy body. Thus it seems to us, from our own observations, to be in the highest degree probable that chronic alcoholism increases the liability of the individual to tuberculosis.

People used often to speak of the transition of other affections of the lungs into pulmonary consumption—that is, into tuberculosis. It was imagined that an old bronchial catarrh, croupous inflammation of the lungs, or catarrhal pneumonia in measles, or whooping-cough, could readily become "tubercular." It goes without saying that such an idea is no longer tenable, after the demonstration of the specific, infectious nature of tuberculosis. If we see a pulmonary tuberculosis develop as a sequel of any other affection of the lungs, we can express the relation between the two diseases only in this way, that the first disease furnished a favorable soil for infection with tubercular virus, and that, consequently, the tubercle bacilli could more easily take root upon a previously diseased mucous membrane than under normal conditions; moreover, there is no doubt that many of the affections, whose "transition into tuberculosis" we once assumed, are themselves tubercular. This is the case, as we shall see, in many cases of pleurisy. No one will now admit the truth of the theory, which Niemeyer once vigorously defended, that a primary pulmonary hæmorrhage could cause the development of pulmonary phthisis. Certainly, in the cases which apparently support such an opinion, the pulmonary hæmorrhage is not the cause, but a symptom, of pulmonary tuberculosis.

No single factor, however, of those which favor the predisposition to tuberculosis, plays so manifest and so visible a part as does heredity. The fact of the heredity of phthisis meets us with such uncommon frequency that it must have forced itself upon the notice of the older physicians. In the great majority of all cases of phthisis we can make out, by close questioning, that in the family, either among the older members or among the brothers and sisters, one or more cases of tubercular disease have already occurred. The closer we investigate, and the more we search for some one of the different forms in which tuberculosis can show itself, like pleurisy, or affections of the bones and joints, the more frequently we can make out this hereditary predisposition.

While there can be no doubt of the facts themselves, their explanation is by no means a simple one. At all events, the question of the heredity of tuberculosis now needs to be studied with renewed care. The hereditary character of tuberculosis may very well harmonize with its infectious character.* We might assume in this case a perfect analogy with syphilis—namely, a transition of the infectious material from the parent to the child before birth. There is only one striking difference between syphilis and tuberculosis—that the children of syphilitic parents very often come into the world with definite signs of infection, while congenital tuberculosis in this sense is an extremely rare occurrence. We must accordingly compare tuberculosis to that form of hereditary syphilis (*lues hereditaria tarda*) in which the first symptoms of the affection come on at a late period.

Since certain considerations, however, constantly oppose such a theory, we are of late disposed to assume that, as a rule, tuberculosis in itself is not inherited, but only the predisposition to tubercular disease. This opinion agrees with the facts that members of a family in which tuberculosis prevails very often show the so-called tubercular habit even without any real tubercular disease; and that they often have "weak lungs"—that is, that they easily get out of breath, and manifest a distinct tendency to catarrh of the respiratory organs. Another fact, which to a certain extent may be regarded as an argument against the assumption of a direct hereditary transmission of the disease, is that, in cases of apparently hereditary tuberculosis, as a rule those organs first show evidences of disease which are most exposed to an infection from the outer world--namely, the lungs and larynx.

Meanwhile there is a third possibility to be considered with regard to the "inheritance of tuberculosis." Many cases of apparently inherited tuberculosis are, in all probability, to be explained by the fact that children or relatives who are constantly in the company of a patient with phthisis are decidedly more exposed to the danger of infection than other persons. This is also the reason why tuberculosis is very often conveyed from husband to wife, or *vice versa*, of which we might furnish a series of examples from our own experience.

The age of the patient has an important influence upon the predisposition to tubercular disease. Pulmonary tuberculosis occurs with special frequency in youth, between fifteen and thirty. It is not rare in children. After forty it is much rarer in its pronounced forms, but it is seen even in the most advanced age. Slight tuberculous changes are very frequently found at autopsy in the lungs of old persons. These changes have, as a rule, no clinical significance.

It has not yet been shown that sex has a special influence upon the predisposition to the disease.

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* Jani has made the very interesting observation, that a few tubercle bacilli may be found in phthisis in the vas deferens and the prostate, or in the folds of the lining membrane of the tubes, without apparent disease of these parts. Whether this, however, explains the hereditary transmission of tuberculosis, is as yet doubtful.

PATHOLOGICAL ANATOMY OF TUBERCULOSIS, ESPECIALLY OF PULMONARY TUBERCULOSIS.

If now we inquire wherein consists the injury which the tubercle bacilli inflict upon the body, the first point to emphasize is that the action of the tubercle bacilli is at first invariably purely local. Tuberculosis does not belong to the "general infectious diseases," in which the infection of the whole organism, the "general infection" of the body, predominates over the local disturbances. The essence of tuberculosis, at least in the great majority of cases, is the local disease. The tubercle bacilli give rise to definite anatomical changes in the organs where they settle, and the consequent disturbance of function in the organ has an effect on the rest of the body. In many cases the whole body may be so little affected that we may be justified in saying that there is a purely "local tuberculosis."

The danger of tubercular diseases, however, consists in the fact that the local affection often attacks the most important organs, as the lungs and the brain, and sets up such extensive anatomical changes in them that because of these changes alone it becomes impossible for life to continue longer. Besides this, the infection in many cases does not always confine itself to one organ, but the infectious material extends over the body by ways and means which we shall learn in part later on, and attacks one organ after another, or even many at once. Finally, it is to be said that the peculiar character of the changes caused by tuberculosis explains why these are often the cause of manifold secondary processes, such as toxic effects and secondary infections. Thus arise fever and other important symptoms, which will be minutely considered later.

The entire local action of the tubercle bacilli—that is, the pathological anatomy of tuberculosis — is almost wholly independent of the organ attacked. Tuberculosis belongs to the group of so-called "infectious tumors"—that is, the local action of the tubercle bacilli consists chiefly in the production of a proliferation and accumulation of cells at their place of settlement, which is termed a tubercular new growth.

According to the later investigations of Arnold, Baumgarten, and others, the process begins with hyperplasia of the normal tissue, both of the interstitial and of the epithelial cells. In this way are developed the so-called epithelioid cells and the giant cells. The next step in the process is the migration of numerous leucocytes from the surrounding blood-vessels. The leucocytes, or round cells, collect about the above-mentioned new-formed cells, and may finally completely cover them. A network, or reticulum, is found between the individual new-formed cells and the wandering cells. This probably represents the remains of the original interstitial tissue, crowded apart by the increased number of cells. There is no new formation of vessels. The tubercle contains no blood-vessels. The tubercle bacilli lie especially inside the giant cells, but also in their vicinity.

If these changes have progressed far enough, they become visible to the naked eye as a small, circumscribed grayish point, which is called a miliary tubercle. From these minute nodules the disease itself obtained its name of tuberculosis. By approximation and coalescence of neighboring tubercles—for these keep developing because of the spread of the local infection—the tuberculous formation continually extends itself, and thus are gradually formed the large tubercular nodules, and finally the diffuse tuberculous new growth or the diffuse tuberculous infiltration.

The tuberculous new growth, as such, can scarcely ever be distinguished histologically from other infectious tumors, such as those seen in syphilis and leprosy. Tuberculosis, however, has a characteristic difference in its final stages of cheesy degeneration and eventual disintegration of the new-formed tissue, processes which are apparently connected with the absence of blood-vessels and the consequent deficiency of nutrition of the new formation. Both the tuberculous infiltration and also the portions of the original tissues inclosed by it perish, lose their nuclei, and finally become disintegrated. The manner in which they are destroyed —namely, "fatty degeneration"—belongs in the group of the so-called coagulation necroses. This process is recognizable to the naked eye, because the tuberculous infiltration when it becomes thus degenerated takes on a pronounced yellowish color. Wherever the necrotic portions of tissue are superficially situated they are cast off, giving rise to tuberculous ulcers.

Alongside the tuberculous new formation there are found in the organs affected with tuberculosis various inflammatory processes, either simple or suppurative or hæmorrhagic. We may therefore surmise that the tubercle bacilli, besides their characteristic effects, act simultaneously in another *rôle* as excitants of inflammation; but it is very probable, and especially so in pulmonary tuberculosis, that many of the inflammatory changes which arise are not peculiar to the tuberculosis as such, but are to be regarded as secondary processes (*vide infra*).

As regards the special anatomical processes and appearances in pulmonary tuberculosis, the tubercular change usually begins in the walls of the smallest bronchi, or not rarely in the alveoli themselves. The disease does not begin, however, in many different parts of the lung at once, but probably in one or two circumscribed spots only, and in a great majority of cases in one apex. We do not know why the apices are so often the starting-point of phthisis, but perhaps it is because of their relatively slighter expiratory power, which renders them a favorable lodging-place for the tubercle bacilli.

The tubercular infiltration begins in the bronchial wall and extends gradually to the periphery. A tubercular peribronchitis arises as a result of the original tubercular bronchitis. The infectious material, as soon as there is any superficial ulceration, is readily carried from the original focus of the disease to other bronchi, by means of the current of air, and thus the disease gradually extends. Tubercular peribronchitis is usually easily recognized with the naked eye. We notice the little lumen of the bronchus in the middle of the "cheesy" nodule, which at first is gray and later yellowish. Many of the adjacent nodules run together in part, and even entirely. The lumen of the bronchus is either wholly plugged by the infiltration, or the destruction of the necrotic cells begins in the midst of the peribronchitis. In the latter case the lumen is enlarged to a little irregular hole—the first beginning of the formation of a cavity.

The alveolar tissue of the lung can not long remain unaffected, with such a disease of the smaller bronchi. Lobular atelectasis, the necessary result of every permanent bronchial obstruction, must arise, but this soon passes into a lobular pneumonia, which from its specific nature later becomes caseous. We can not go into the histological details here. The alveoli are filled with pus-corpuscles and large epithelioid cells, which are considered by many authors to be the offspring of the alveolar epithelium. The alveolar walls are also infiltrated. This finally results in the destruction of the cheesy and necrotic tissue, and consequently in the formation of cavities. At other times the neighboring nodules run together, and the tubercular infiltration thus extends, giving rise to a diffuse caseous pneumonia. These processes may all be readily recognized by the naked eye. The earlier stages of atelectasis and infiltration correspond to the jelly-like, gray coloring seen in the so-called gelatinous infiltration of Laennec, and the transition to caseation is recognized by the eye from the yellowish color.

Although all the processes thus far described are destructive in their nature, changes are also found in the lungs, in tuberculosis, which seem to have a tendency toward circumscribing the disease and toward healing. Prominent among these are the chronic interstitial processes. We meet with the formation of new connective tissue, partly about the tubercular infiltration, but especially where there is already destruction of tissue, and this leads to contraction and the formation of a firm cicatrix. The encapsulated cheesy masses may then be in part reabsorbed; in part they undergo calcification. The possibility of such a halt in the tuberculous process depends, however, upon certain conditions. The tuberculous new growth and its destruction must not advance too rapidly, and the new-formed tissue must not itself be destroyed before it becòmes cicatrized. We see the cicatricial formation, therefore, more especially in chronic cases; we find it in places which have been affected the longest, and where the tubercular process, perhaps, has finally come to a standstill of its own accord. Macroscopically, this cicatricial connective tissue is composed of a thick, firm substance, usually pigmented—the so-called pigment induration. If the cicatricial formation follows a previous extensive destruction of the pulmonary tissue, the affected portion of the lung may thus be diminished to less than half its original bulk. Cavities and firm cicatricial tissue form the anatomical basis of such an extensive "pulmonary contraction." The cavities are either formed in the usual way from the destruction of lung tissue, or they may be simple bronchial dilatations due to the traction of the contracted tissue—bronchiectasic cavities.

The contractile changes in pulmonary tuberculosis teach us that the tubercular process is in itself capable of healing. The incurability of most cases of phthisis is due to the fact that the infectious material from every existing tubercular nodule is carried into other bronchi, and there sets up a new tuberculosis. Thus the disease is constantly extended. The original tuberculosis, which was localized in one apex only, gradually spreads to the lower portion of the lung. The infectious material is carried by coughing into the trachea, and from this point may be carried by inspiration into the other lung. This becomes diseased, and finally there is such an extensive destruction of the lungs as to make the further continuance of life impossible.

Besides the spots of specific tubercular disease, we very often find in phthisical lungs simple inflammatory processes, bronchitis, lobular catarrhal pneumonia, and sometimes even croupous pneumonia. This, of course, is rarely extensive. These changes may be partly due to the influence of the tubercle bacilli themselves in exciting inflammation, but it is, of course, easily conceivable that many other excitants of inflammation may easily settle in the secretions of the bronchi and the cavities, and lead to complicating diseases of the bronchial mucous membrane and the alveoli. Thus in pulmonary tuberculosis a local gangrene may occasionally arise. An especial importance attaches to the secondary inflammatory processes in the lungs, because they furnish a suitable spot for the subsequent invasion of the tubercle bacilli. Thus, in phthisical patients, foci of simple inflammation very frequently assume a tuberculous character.

If we consider the list of anatomical processes which are found in tuberculosis of the lungs, and which may be combined in the most manifold ways, we can understand the great diversity in the anatomical picture in different cases. Simple bronchitis, tuberculosis of the bronchial wall and tubercular peribronchitis, diffuse cheesy pneumonia, and destruction of the tubercular new growths, with the formation of cavities, on the one hand, contracting interstitial pneumonia, cicatricial formation, and pigment induration on the other—these are the comparatively simple anatomical lesions from which the whole process in its most varied forms is composed. Besides this, we often find here and there one or more miliary tubercles scattered through the lungs which are probably due very largely to an extension of the infectious material by means of the blood or lymph current.

The secondary tubercular diseases of the pleura, the bronchial glands, and other organs, will receive a special description.

CLINICAL HISTORY OF TUBERCULOSIS IN GENERAL AND OF PULMONARY TUBERCULOSIS IN PARTICULAR.

In judging of the various appearances in the clinical picture of tuberculosis we must especially consider the following points: The place of the first infection is of the chief importance—that is, the place where a local affection, set up by the tubercular poison, first arises. As has been said, the lungs are the organs first attacked in a large number of cases, and such cases are termed primary pulmonary tuberculosis. In other cases, the tubercular poison first fixes itself in the larynx, as in primary laryngeal tuberculosis, or it reaches the intestine, as in primary intestinal tuberculosis, or the genito-urinary organs, as in primary tuberculosis of the genito-urinary apparatus, etc. In other cases still, we apparently have to do with a primary tuberculosis of the serous membranes (*vide infra*) or of the lymph-glands, particularly the bronchial lymph-glands (*vide infra*); and finally, we very often see primary tubercular diseases of the bones and joints, to which the most part of the formerly so-called chronic scrofulous and fungous inflammations of the bones and joints belong. It is obvious that all these diseases, although identical ætiologically, must have a very different clinical appearance.

Another reason for the great variation in the course of tuberculosis is found in the fact that the extension of the local tubercular process may vary very greatly as regards time. Tuberculosis in one case may produce the most extensive destruction in both lungs in a few months or even weeks, and in another case it may remain almost quiescent for years, or advance only very slowly. We do not know fully on what these differences depend, but much is certainly due to the hygienic influences under which the patient lives. In the last instance, however, we are often led to think of individual differences of constitution, which now check and now favor the rapid extension of the disease.

A third and final reason for the differences in the course of tubercular infection is the manner of the further extension of the tubercular poison in the body. As we shall see in the description of tuberculosis in single organs, there are different ways in which tuberculosis may pass from one organ to another. Many contingencies are involved here, and we can easily comprehend how greatly the whole clinical course of the disease must be modified by the rapidity and the degree in which individual organs are affected.

After these preliminary remarks, which we have thought necessary to a right understanding of tubercular disease in general, we will pass on to the description of the clinical course of pulmonary tuberculosis.

The onset of pulmonary tuberculosis is quite slow and gradual in the majority of cases. The patient can give only an approximate idea of the time when he began to be ill. The symptoms which he notices are referred directly to the respiratory organs. The cough and its attendant expectoration are the chief things which affect him. Besides that, there is often pain in the chest, either the pleuritic stitch, or a pain in the sternal region, or pain between the shoulder-blades. The patient is also apt to complain of shortness of breath, especially on severe physical exertion.

Besides these symptoms, which point pretty directly to disease of the lungs, there are often quite striking general symptoms. The patient's emaciation is especially noticeable, which may be partly, though not wholly, explained by his loss of appetite. Besides the emaciation there is a steadily increasing pallor of the skin. The patient also shows a general dullness, weakness, and disinclination to work. There is not infrequently a slight rise of temperature in the first stages of the disease, which causes chilliness and subjective feelings of heat. Severe night-sweats may also be noticed early.

All such general symptoms should determine the physician not to regard the mild thoracic symptoms, which are also present, as insignificant, but to think of the possibility of incipient tuberculosis. It is very important to remember that the pulmonary symptoms may be entirely subordinate to the general symptoms mentioned, and that the patient himself is apt to pay little or no attention to them. Incipient phthisis is therefore frequently diagnosticated as simple "chlorosis" or "gastric catarrh" for a long time, and is treated as such. An early and careful physical examination of the lungs and of the expectoration is the only protection against such an error.

Both the pulmonary and the general symptoms assume significance, if we have to do with a patient in whom we suspect a "tubercular predisposition." We very often meet people in whose family, either in the parents or the brothers and sisters, several cases of phthisis have occurred. They are persons who are always pale and weak, and who have previously shown a special liability to disease. particularly to disease of the respiratory organs. They have perhaps had diseases which our present theories bring into direct relation with tubercular infection. We refer to those quite frequent cases of pulmonary tuberculosis in persons who have previously suffered from "scrofulous diseases," like chronic swelling of the lymph-glands, chronic affections of the eye or ear, or fungous diseases of the bones and joints. This fact does not signify that scrofula passes into tuberculosis. It is much more probable that many scrofulous diseases are really tubercular, as has been proved by the result of inoculations in animals, and recently by the discovery of tubercle bacilli in "scrofulous" lymph-glands and in the fungus nodules in the bones and joints.

As we have said, pulmonary tuberculosis often develops in persons who have suffered before from diseases of the respiratory mucous membrane, in whom, as the expression is, the lungs have always been the *locus minoris resistentiæ.* The predisposition to tuberculosis may really perhaps coincide at times with the predisposition to other pulmonary affections; thus we see tubercular patients who have previously had several attacks of croupous pneumonia. In other cases, however, the predisposition to tuberculosis is probably caused by some disease of the respiratory mucous membrane, although sometimes these previous diseases of the respiratory organs are themselves of a tubercular nature. This is especially true of pleurisy, but we shall have to take up its relation to pulmonary tuberculosis somewhat more in detail in our description of pleurisy.

Although the first symptoms of pulmonary tuberculosis often develop in people who were not quite well before, this is true in only a part of the cases. We often see precisely the same symptoms, both the pulmonary and the general, occurring in persons who previously seemed quite well and strong. No constitution is perfectly protected against the disease. We have even seen the herculean athletes of a circus die of phthisis.

In distinction from the slow and gradual method of the development of tuberculosis which has just been described, the first symptoms in other cases may be more sudden. A definite exposure is often given as a cause, after which the first symptoms of the disease have speedily developed. It goes without saying that we must consider these harmful influences—a chilling of the body, a cold draught, over-exertion, or marked mental excitement—at most, as exciting causes.

Some cases in our own observation seem to us worthy of note, in which young people have fallen ill quite suddenly, with rather severe, general febrile symptoms. At first no cause for the fever could be made out, so that the diagnosis was in doubt, or the attack was even falsely regarded as typhoid or some other disease. Some time later thoracic symptoms developed, and it became possible to detect the physical signs of phthisis. Most of these cases took quite a rapidly progressive course.

In conclusion, those cases are to be mentioned in which the first signs of tuberculosis appear, not in the lungs but in the larynx. The full description of these cases has already been given in the chapter on laryngeal tuberculosis (see page 136).

The further course of pulmonary tuberculosis may vary so much that it is impossible to give a complete enumeration of all the possibilities.

In some cases it advances rapidly. We can make out the extension of the disease objectively, almost from week to week. At first the apex of one lung alone is attacked, soon after the lower lobe of the same lung, then the other lung, either at the apex first or in the lower part. Besides the pulmonary symptoms, there is quite a high fever, rapidly increasing emaciation, and general loss of strength. Death ensues in a few months. We term such cases florid phthisis, or "galloping consumption."

In other cases, however, the disease has a remarkably chronic course. Its onset is very gradual, or else, after rather an acute onset, there is a comparative cessation of all symptoms. The thoracic symptoms do not disappear, but they are only trifling, and do not disturb the patient. Physical examination of the lungs does not show any extension of the process for months. The fever which accompanies it is slight, if there be any. The patient remains quite well nourished, but in some cases there is a good deal of weakness. He feels better and worse by turns, his condition being greatly influenced by the care and nursing he receives.

Unilateral contracting phthisis especially has this comparatively favorable course (*vide supra*). The affection remains confined to one lung for a long time. The occurrence of contraction shows the slight tendency of the tubercular process to advance, and with satisfactory care the patient may remain quite well for years.

In cases, too, which have had severe symptoms for a long time, a temporary standstill of the affection may take place, or an actual improvement in all the symptoms. At other times, in cases which have made no advance for a long time, the symptoms suddenly grow worse.

There are all possible varieties between the extremes of florid phthisis and the very chronic cases which last for years. If we recall the further modifications which the course of the disease may assume if complications arise, we can appreciate the manifold character of the clinical picture of phthisis.

Most cases terminate fatally. Death ensues, either with the signs of general exhaustion, or as a result of the final failure of respiration; or it is due to the occurrence of complications, like tubercular meningitis, miliary tuberculosis, pulmonary hæmorrhage, or pneumothorax. A recovery from the tubercular process is certainly possible. The comparatively great rarity of recovery in pulmonary tuberculosis, however, is due chiefly to the possibility of the continual spread of the tubercular poison in the lungs themselves, and also in other organs. From clinical and pathological experience, however, we can not deny the possibility of definite recovery in pulmonary tuberculosis. We do not, of course, mean a *restitutio ad integrum* of the lung-tissue, but a recovery with a cessation of the tubercular process, and with the formation of a cicatrix, or contraction. Such recoveries, as we have said, are rare, and they occur only where the changes in the lungs are of limited extent. The possibility of recovery depends mainly upon the general physical constitution and upon the external circumstances of the patient.

SPECIAL SYMPTOMS AND COMPLICATIONS.

1. **Symptoms on the Part of the Lungs.**—*Pain in the Chest.*—Extensive destruction in the lungs may exist without any feeling of pain. Many cases of phthisis are painless throughout their course. In other cases, however, the patient's chief complaint is of severe pains in the side or in the front of the chest. These are probably always due to co-existing affections of the pleura, like pleurisy, or pleuritic adhesions. In patients who suffer from severe cough, pains sometimes arise in the abdominal muscles and at the insertion of the diaphragm, due to the excessive muscular contraction. Stabbing pains between the shoulder-blades are held by some to be not wholly unimportant as a diagnostic symptom of incipient phthisis.

Cough.—In the majority of instances cough is one of the most distressing symptoms in phthisis, but its severity varies very much in different individuals, and at different times in the same patient. We sometimes see cases in which, in spite of advancing phthisis, cough is remarkably slight, or entirely absent. In these we usually have to do with patients who are very slightly sensitive. In cases with severe cough, it is often worst at night, but paroxysms of coughing of long duration are also apt to come on in the morning or evening hours, which are painful and very distressing, and unpleasant for the patient. The cough is usually associated with a more or less abundant expectoration, but sometimes there is chiefly a dry cough. The cough becomes very severe if the tubercular affection attacks the larynx and trachea (see laryngeal tuberculosis).

Expectoration.—The amount of expectoration differs very much in different cases. It is most abundant where there is extensive formation of cavities in the lungs. In such cases it is often evacuated in the morning by persistent coughing. The consistency of the great part of the sputum is muco-purulent; and it does not differ from that of simple bronchitis; in fact, a large part of the phthisical expectoration comes from the catarrhal inflammation of the bronchial mucous membrane. Another part comes from the purulent secretion of the walls of the cavities. The sputum has a characteristic tendency to roll itself together in single large lumps, into the ball-like or so-called nummular sputa. This is noticed especially where there are cavities.

The admixture of blood with the sputum is of great diagnostic and practical importance. Since no other disease so often gives rise to the presence of blood in the expectoration, coughing of blood (hæmoptysis) is almost synonymous with consumption among the laity. Little streaks of blood in the expectoration are quite frequent. They have no great significance, but, of course, they may sometimes be the precursors of severe hæmorrhages. Profuse hæmoptysis takes place when the wall of a little pulmonary vessel—almost always a branch of the pulmonary artery—is infiltrated, destroyed, and finally eroded, by the tubercular new growth. The reason why hæmoptysis is not more frequent is because the contents of the vessels usually undergo thrombosis. Severe hæmorrhages very often have their origin in the perforation of little aneurisms of the branches of the pulmonary artery, which penetrate into the interior of the cavities. In the cases of fatal hæmoptysis we frequently succeed in finding the little aneurism and its point of rupture.

Pulmonary hæmorrhages occur in all stages of phthisis. The amount of blood coughed up sometimes amounts to only one or two tablespoonfuls, sometimes to one or two pints. The blood is bright-red in color, usually quite frothy, only slightly coagulated, and partly mixed with the other constituents of the sputum. If the patient recovers from the first severe hæmoptysis, the expectoration usually contains some blood for several days. Recurrences of severe hæmorrhages are frequent. The hæmoptysis sometimes comes on quite suddenly, often at night, without any occasion, but it may return from some definite cause, such as physical exertion, severe paroxysms of coughing, straining at stool, or mental excitement. Many cases of phthisis are characterized by a special tendency to hæmorrhage, while in many others hæmoptysis never occurs. Severe hæmoptyses are, of course, always an undesirable and dangerous complication, since they weaken the patient very much, and also depress his spirits. Many patients maintain their peculiar, careless indifference, which is almost characteristic of the disease, despite the spitting of blood. The hæmoptysis may sometimes be the direct cause of death, but, as a rule, the patients survive it. We can not make the general statement that the further course of phthisis is materially hastened by hæmoptysis. In not rare instances, however, it is observed that after a hæmoptysis the pulmonary disease extends more rapidly, the fever becomes higher and more persistent, and the general condition of the patient grows worse.

A purulent sputum intimately mixed with blood is quite frequent and characteristic in many cases of phthisis with extensive formation of cavities. This is formed in the cavities from the mixture of the purulent secretion with little capillary hæmorrhages. In this way the sputum, which is often nummular, assumes a greasy character and a reddish-brown or chocolate color.

If fœtid or gangrenous processes develop in the lungs, the sputum becomes fœtid. In some cases we see temporarily in phthisis the characteristic sputum of croupous pneumonia, which comes from portions of the lung attacked with pneumonia.

Microscopic examination of the sputum may show—besides the ordinary elementary forms, such as pus-corpuscles, red blood-corpuscles, pavement epithelium, drops of myeline, and sometimes, perhaps, pulmonary epithelium—two constituents which are of decided diagnostic importance: elastic fibers and tubercle bacilli.

The demonstration of elastic fibers in the expectoration permits us to decide with certainty that there is a destructive process in the lungs, and thus it usually is direct proof of tuberculosis. Elastic fibers are also found in pulmonary gangrene, and in the very rare cases of pulmonary abscess, as well as in tuberculosis, but gangrene is easily recognized by the other peculiarities of the sputum. The search for elastic fibers in the expectoration of tubercular patients demands a certain amount of practice. We are most sure to find them if we look in the sputum, when it is spread out, for little lentiform particles, which can easily be made out with the naked eye. These consist of necrotic shreds of tissue torn off from the walls of cavities. If we press one of these "kernels" under a cover-glass we find, in the midst of the granular detritus, beautifully twisted elastic fibers, which often have quite a definite alveolar arrangement (see Fig. 26). The elastic tissue is the only one spared in the general destruction. There is a special method of looking for elastic fibers, but we have found it unnecessary. The sputum is boiled in sodic hydrate dissolved in water, and we look for elastic fibers in the precipitate which then forms. We are never justified, however, in deciding that pulmonary tuberculosis is absent because we do not find elastic fibers in the sputum. Their presence is the only thing that has real diagnostic significance.

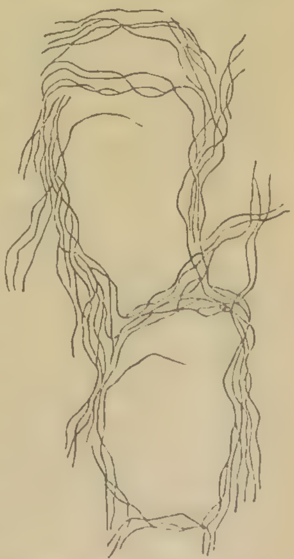

FIG. 26.—Elastic fibers.

The discovery of tubercle bacilli in the expectoration of phthisical patients is of much greater importance, and often this alone is decisive (see Fig. 27). They were first demonstrated by Koch, but Ehrlich devised the first simple method for their discovery, which can be easily employed by any physician.

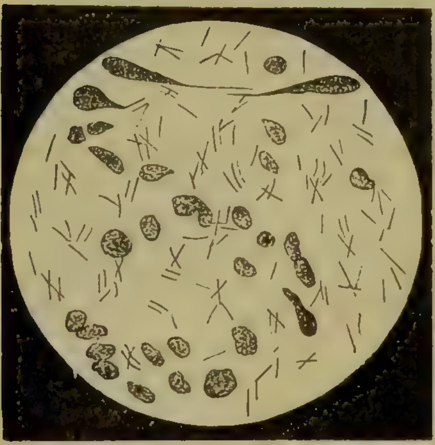

FIG. 27.—Tubercle bacilli in the sputum.

The sputum is spread in the thinnest possible film on a cover-glass, and permitted to dry on it. The best way to do this is to rub some of the sputum between two cover-glasses, and to slide one slowly off of the other. We pass the cover-glass through the gas-flame three times slowly to fix the sputum, and then, after cooling it for a little time, we let it float in the staining fluid. The latter is made in this way: One part of aniline oil and six parts of water are mixed in a test-tube, filtered, and to the filtrate, known as aniline-water, a portion of which is placed in a watch-glass, about eight or ten drops of a concentrated alcoholic solution of methyl violet or gentian violet are added. The cover-glass on which the sputum has been placed is allowed to remain in the watch-glass as long as possible—one or two hours, or more. If it is desired to shorten the process, the staining liquid is warmed, but not to the boiling-point. The bacilli will in this case usually be stained with sufficient distinctness in as short a time as ten or fifteen minutes. When the cover-glass is taken out of the staining fluid it is immersed in a solution of one part of nitric acid in three parts of water, and in this way the preparation is almost completely bleached in a very short time. The tubercle bacilli alone have the property of retaining the stain which they have taken on, despite the action of the nitric acid. When the cover-glass is taken out of the nitric acid, it is rinsed in water, dried between two layers of blotting-paper, then stained for about a minute in a one- or two-per-cent. solution of Bismarck-brown in water, then dried, and examined in water, or, better, in Canada balsam. A dry lens of the strength of, say, objective number eight of Hartnack (330 to 440 diameters) answers perfectly for the examination. The pus-cells and any other bacteria that may be present are colored brown. The tubercle bacilli, however, are stained dark blue. Ziehl and Neelsen's staining fluid is also to be highly recommended. It is composed of one hundred parts of distilled water, five parts of carbolic-acid crystals, and one part of fuchsin. After these are mixed and filtered, ten parts of alcohol are added. A solution thus prepared lasts for quite a long while, and stains the tubercle bacilli a bright red even in a few minutes, provided warmth is employed. For bleaching the other components of the preparation, a five-per-cent. solution of sulphuric acid may be employed, and the best means of secondary staining of the pus-corpuscles is to use a watery solution of methyl blue. If a solution is made up containing fifty parts of water, thirty of alcohol, twenty of nitric acid, and as much methyl blue as the liquid will take up, the bleaching and the secondary staining may be accomplished simultaneously. This is a very practical method, which was first recommended by B. Fränkel.

The number of bacilli varies a good deal in different cases, and at different times in the same case. The more bacilli there are, the more readily can we decide that there are extensive processes of ulceration. We can found no definite prognostic conclusions upon the behavior of the bacilli, but their presence is of the greatest significance in a diagnostic point of view, especially as they can be found upon careful examination in incipient cases. At so early a period all the other symptoms by themselves would very often not warrant an absolute diagnosis.

Dyspnœa.—A marked subjective feeling of dyspnœa is a symptom which is of relatively rare occurrence in phthisis.

Many patients hardly ever complain of their breathing in spite of extensive destruction in the lungs. A patient who is much emaciated manifestly needs little oxygen, and the increased frequency of respiration, which is almost constant, can satisfy his needs. If there is a greater demand upon the respiration, a subjective feeling of dyspnœa may of course very readily occur, especially on a slight bodily exertion. In many cases, however, the patient complains of a difficulty in breathing even when quiet, especially if pleuritic pains or adhesions between the surfaces of the pleura prevent him from taking a deep breath, and in the final stages the dyspnœa may be extreme.

2. **Symptoms on Physical Examination.**—In many cases inspection gives us that general impression of the patient which we term the "phthisical habit." The special signs of this are as follows: A slender but often quite a tall frame, weak muscular development, a thin layer of fat, a pale and perhaps very delicate skin with a bluish translucence, sometimes a circumscribed "hectic" flush in the cheeks, a long and slender neck, a long and narrow thorax, small, thin hands, etc. There are, of course, many variations from this type in individual cases.

The inspection of the thorax is of special value. The phthisical or "paralytic" thorax is generally noticeable from its length, but it is narrow and flat. Unusual width of single intercostal spaces, and acuteness of the epigastric angle, are associated with a long thorax. The sternum is also long and narrow, and the sternal angle—Louis's angle—is often prominent. The supra-clavicular and infra-clavicular fossæ are sunken, the neck is wasted, and the shoulder-blades stand out from the thorax. On comparing the two halves of the body, we very likely see unilateral contractions, most frequently in the upper and anterior portion of the thorax, but also in the lower portions.

The paralytic form of thorax is very often seen in phthisis, but it may be entirely absent.

The respiration is usually somewhat accelerated, and sometimes quite markedly so in women with disease of the apices. The feminine type of high thoracic breathing is largely changed to low thoracic or diaphragmatic breathing. A unilateral impairment of respiration is of greater importance; in such a case one apex, or one side, if there be phthisis of the lower lobe, holds back in inspiration. The respiration is sometimes irregular, especially if there be pleuritic pains.

The results of percussion are, of course, entirely dependent upon the sort of anatomical changes in the lungs, and hence differ very greatly in different cases. Since the phthisical process begins in the apices in the majority of cases, our chief attention is turned to the condition of the upper portions of the lungs on percussion. Slight changes in percussion may wholly escape discovery. Only when the air contained in the lung-tissue in the part affected is replaced to a certain degree by the tubercular infiltration does the percussion-note become dull. Unilateral dullness at the apex is therefore one of the most frequent physical signs of phthisis. We can usually make it out most plainly in the upper anterior intercostal spaces first, and in incipient cases often in the supra-clavicular fossæ only, but it is also observed sometimes in the back in the supra-scapular fossæ. As the infiltration advances the dullness becomes more extensive. It frequently assumes a tympanitic quality, as a result of diminished tension or partial retraction of the lung-tissue.

The formation of cavities in tuberculosis has a great influence on the percussion-note. The dullness on percussion may thus become decidedly less, the degree of resonance depending, of course, upon the fullness of the cavity and the character of the surrounding tissue. We often find a decided tympanitic resonance or a combination of dullness and tympany over a cavity. The different modifications of the percussion-note in cavities are given below. The "cracked-pot resonance," or buckram sound, is met with in percussing over cavities, but we also find it in many other pathological conditions.

Auscultation also gives no special pathognomonic signs of phthisis. Varying with the character and extent of the tubercular changes, abnormal respiratory sounds and adventitious sounds are heard in place of the normal vesicular murmur. With slighter changes the vesicular breathing is merely modified; it seems remarkably diminished or interrupted, or sometimes exaggerated, with prolonged expiration. When the infiltration of the lungs increases, we find bronchial respiration in place of the vesicular breathing; but, on the other hand, the formation of a cavity is a frequent cause of bronchial respiration.

The most constant auscultatory signs of phthisis, and the most important for diagnosis, are the different kinds of râles, which are due to the accumulation of the secretion in the bronchi or in the cavities. They are heard at one apex only, or over a larger space, according to the extent of the affection.

Physical Diagnosis of Incipient Phthisis.—On account of the importance of the diagnosis of incipient phthisis, we will here mention connectedly the physical signs which are chiefly met with in it. The auscultatory signs in the beginning of the disease are generally more certain and easier to recognize than those from percussion. He who lays too much weight on the so-called "slight dullness at the apex" will often make a false diagnosis. We will mention particularly the following symptoms: 1. Constant and evident diminution of the respiratory murmur at one apex, especially if it is associated with marked deficiency of the respiratory movement on the affected side. In some cases the respiratory murmur on the diseased side is not weaker, but it has a more indefinite and harsher character; or again it may be rude, sharp, and "puerile." 2. Markedly interrupted respiration at one apex. 3. A prolonged expiratory murmur, which has a harsh character. 4. The discovery of dry rhonchi or moist râles at one apex is most important, since we know by experience that "apex catarrhs" are, as a rule, tubercular. 5. Definite dullness, apparent on repeated examinations, or tympanitic dullness at one apex. 6. Evident contraction at one apex, as revealed by inspection or percussion above the clavicles. 7. Some authors lay stress upon a systolic murmur in the subclavian artery, especially loud on expiration. This may occur in the beginning of phthisis, if the caliber of the vessel is narrowed by processes of contraction in the neighboring apex, but this symptom is neither frequent nor of great practical importance.

The chief rule in the diagnosis of incipient phthisis must be held to be this —not to give a definite opinion until repeated examinations have been made. The other portions of the lungs are to be carefully examined as well as the apices, since in not very rare cases tuberculosis may begin in the lower lobes. We must always consider the patient's general symptoms as well as the physical signs.

Symptoms of Cavities.—The diagnosis of a cavity in the lungs by means of the physical signs is often very difficult. We may mention as the chief symptoms of a cavity: 1. Loud bronchial respiration, perhaps of an amphoric character, in places where the percussion-note is only slightly or not at all dull. Such a condition means that the bronchial respiration is not due to an infiltration of lung-tissue. Bronchial respiration, however, may of course be heard over cavities which are surrounded by thickened lung-tissue, and hence give dullness on percussion. 2. The so-called metamorphosing respiration, which begins as vesicular and suddenly becomes bronchial, is heard especially over cavities, and hence has a diagnostic value. 3. The different kinds of "changes in the percussion-note" over cavities are important signs. Wintrich's change in pitch is when the tympanitic resonance, which is obtained over the cavity, becomes, on opening the mouth, more decidedly tympanitic, louder, and especially much higher. The respiratory change of pitch of Friedreich usually consists of a higher pitch on inspiration, but here there are numerous variations. Gerhardt's change of pitch (Weil) consists in a change of the tympanitic resonance when the patient changes his position, the pitch usually being higher when the patient sits up than when he is lying down.* 4. Loud, bubbling râles are one of the most frequent signs of a cavity. They are definite indications of the occurrence of râles in a larger space than is normally present in the apices of the lungs.

Contraction of the Lungs [Fibroid Phthisis].—Unilateral contraction of the lungs, more frequent on the left than on the right, is a form of tuberculosis which is made apparent both by special physical signs and also by certain clinical peculiarities. It is usually recognized at once by inspection of the thorax, one side of the thorax being remarkably retracted. The upper anterior portions of the thorax, and, in all cases of a high degree of disease, the lower lateral and posterior portions, are much less tense than the corresponding parts on the other healthy side. The fossæ and intercostal spaces on the affected side are deeper, the shoulder-blade is drawn nearer the vertebral column, and the latter is even sometimes drawn over to the contracted side (scoliosis). The resonance is diminished to a greater or less degree, over the affected side, which either lags behind or remains almost wholly at rest on respiration. The respiratory murmur is quite loud, and bronchial; and we also hear many râles, which are usually bubbling. Anatomically, we have to do with a marked process of contraction of the interstitial connective tissue in the lungs, which is almost always associated with extensive formation of cavities, partly of an ulcerative, partly of a bronchiectasic character. The pleura is involved in the process almost without exception, but almost always secondarily; it is also thickened and contracted. If the pleuritic thickening is marked, the respiratory murmur and the vocal fremitus are decidedly weakened.

The influence of the contraction on the neighboring organs is very decided, and usually it is easy to discover. The heart especially, whose external pericardium is in most cases very adherent to the pleura, is drawn well over to the side of the contraction. The apex-beat and the cardiac dullness are correspondingly displaced. With left-sided contraction the heart may be drawn over to the line of the left axilla, and with right-sided contraction it may be drawn to the median line, or even to the right of the sternum. With contraction of the left upper lobe the anterior surface of the heart comes into immediate contact with the anterior chest-wall over a larger area than normal. We therefore see the motions of the heart over an abnormal extent, and we can often feel very plainly in the second left intercostal space the pulsation and the closure of the valves in diastole of the pulmonary artery. The upward traction of the diaphragm may be recognized by the position of the liver, or, in left-sided contraction, by the increase of the "semilunar" tympanitic space on the left. We usually find the sound lung on the other side quite emphysematous, as shown by the downward displacement of the lower boundary of the lung, and also by the drawing over of the anterior median edge of the lung to the contracted side. In a part of the cases we can make out by percussion the development of consecutive dilatation and hypertrophy of the right ventricle.

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* Fuller details of the significance of the different changes in the pitch are found in Weil's "Handbuch der topographischen Percussion." Leipzig: Vogel, 1880.

These are the chief physical signs of the so-called unilateral form of chronic pulmonary contraction. We would add here a few clinical remarks. The cases often, but of course not always, run a very chronic course, lasting for years. The general condition and the nutrition of the patient may thus remain comparatively undisturbed for a long time. The patient looks somewhat pale and cyanotic, yet he is so well nourished as to present a very marked contrast to the appearance of the ordinary cases of phthisis. The appetite remains good, the fever is entirely absent, or else a slight degree of fever may be at times discovered by careful examination. The cough and expectoration, too, though often very severe, are at other times very slight, especially when the patient has good care and nourishment. We need not wonder, then, that many physicians do not consider that these cases have anything to do with phthisis—"consumption"; and yet we are convinced by many clinical and anatomical observations that, ætiologically, they are in by far the greatest part tubercular. They represent a very slow form of tuberculosis, which has time to develop interstitial processes which lead to contraction—that is, to local healing. If such cases come to autopsy, their tubercular character is usually definitely confirmed. We find undoubted tubercular lesions in the other lung and also in the remaining organs—e. g., the intestines. Furthermore, sudden changes for the worse may occur in every "pulmonary contraction," even those cases which seem favorable; the other lung may become highly tubercular, a miliary tuberculosis or a tubercular meningitis may develop, etc. On the whole, however, the slow course of this form of chronic tuberculosis is characteristic and of practical significance, and its prognosis is therefore comparatively favorable.

We can not absolutely deny that a non-tubercular unilateral contraction of the lung may occur. As a result of fœtid bronchitis and pulmonary gangrene, processes of contraction develop, which are associated with the formation of bronchiectases, and certainly have nothing to do with tuberculosis. In rare cases also croupous pneumonia is followed by unilateral contraction of the lung; and finally there is a rare and by no means satisfactorily investigated form of unilateral chronic interstitial pneumonia, with contraction, often associated with the formation of bronchiectases. The differential diagnosis of these conditions from tuberculous contraction rests in part upon the clinical history, but mainly upon the absence of tubercle bacilli in the expectoration.

In conclusion, we must mention that there are very many transitional forms between pulmonary contraction and the other varieties of pulmonary tuberculosis. We find more or less extensive processes of contraction in one apex in most cases of phthisis.

Disseminated Pulmonary Tuberculosis.—There is a form of pulmonary tuberculosis which it is very hard to make out on physical examination. In this we have to do with numerous peribronchial nodules disseminated over the whole lung. As there is still a good deal of normal tissue, containing air, between these nodules, percussion affords no dullness, and auscultation gives at most diffuse rhonchi; hence this form is often confused with chronic bronchitis or pulmonary emphysema. The diagnosis can seldom be made from the physical signs, but only from the other symptoms, such as fever, emaciation, striking pallor of the skin, and the sputum.

This form of phthisis sometimes runs a chronic course, but usually it is quite rapid. It occurs in elderly people, and also in children. Many forms of "disseminated, coarse granular" tuberculosis are transitional forms between this and genuine acute miliary tuberculosis.

3. **General Symptoms in Pulmonary Tuberculosis.**—In the description of the general course of pulmonary tuberculosis we have already mentioned the value of the general symptoms in diagnosis and prognosis.

Fever.—Only a few cases of phthisis run their course entirely without fever, but it may be absent for a time, even for weeks and months. This is especially the case in the very chronic forms, like unilateral contraction. The more carefully we take the temperature, the more frequently shall we find a slight evening rise of temperature up to 100° or 101° (38°–38·5° C.), or at least between 99° and 100° (37·5°–38° C.), even at times when the patient is doing favorably. Such subfebrile conditions may last a long time, but many cases of phthisis are associated with high fever.

The fever in tuberculosis is generally noticeable from its very monotonous character. For months the temperature curve may be like this: in the morning a normal or approximately normal temperature, and at evening a rise to 103° or 104° (39°–40° C.), rarely higher. The fever in phthisis, then, shows a decidedly intermitting or remitting character, the so-called "hectic fever."

More rarely we see very irregular temperature curves, where elevations of temperature, which last a shorter or a longer time, alternate in an irregular fashion with periods that are free from fever. It often happens that toward the end of the disease, when the general weakness increases, the curve, that previously was regular in its intermissions, becomes irregular. The intermissions then become deeper, and we often see genuine temperatures of collapse, 94° to 92° (35°–34° C.). At other times the fever may temporarily assume a more continuous character, probably from an increase of the tubercular process. In some cases with an acute onset (*vide supra*) we have also seen, at the beginning of the disease, quite a high and approximately continuous fever, which later went over gradually to the ordinary hectic.

We must probably look for the cause of the fever not in the tuberculosis itself, but in the absorption of septic substances from the decomposition of the secretion of the bronchi and cavities.

In general, it may be said that continuous fever points to an advance, complete absence of fever, on the other hand, to a halt (at least a temporary one), of the tuberculous process; hence is evident the great prognostic importance of the temperature in pulmonary tuberculosis.

Emaciation.—The great emaciation of the patient is very striking in most cases of phthisis. The muscular system and the fatty tissue are affected in equal degree. The soft parts of the thorax are often especially involved. The emaciation is due in part to the patient's loss of appetite, and to the small amount of food which he takes in consequence thereof, but the chief cause lies in the persistent fever and the increased metamorphosis of tissue. Quite a high degree of emaciation, however, may appear in the beginning of the disease with no fever. This we are wont to ascribe to the "general illness," but the special cause of it is unknown. Under favorable external conditions phthisical patients may make quite a decided gain in weight, especially at the times when they are free from fever. In very chronic cases, which run their course from the first without fever, the nutrition of the patient may remain good for a long time. Toward the end of the disease emaciation sometimes reaches its highest degree, and many phthisical patients die "wasted to a skeleton" in the true sense of the word.

Anæmia—Color of the Skin.—In most cases anæmia appears in the course of the disease, which can be recognized by the pale and sallow color of the skin and the visible mucous membranes. The anæmia only rarely reaches that degree of peculiar waxy pallor that is found in idiopathic pernicious anæmia. The existence of the anæmia is also the reason why the phthisical patient does not look cyanotic, in spite of the respiratory disturbance. In the more chronic forms, where the general nutrition suffers less, we often see a cyanotic coloring of the lips and cheeks. Sometimes the skin of phthisical patients assumes a dirty, dusky hue. We have already spoken of the circumscribed "hectic flush of the cheeks" seen with the fever.

General Weakness—Night-Sweats—Nervous Disturbances.—We need not say that the general emaciation and anæmia are accompanied by a marked decline in the patient's power of endurance. He finally becomes so helpless that he can scarcely move alone in the bed.

The tendency which very many patients have to severe night-sweats is not wholly explained. It may have some connection with the fall from the evening febrile temperature to the morning remission, and perhaps it is due to the greater accumulation of carbonic acid in the blood from the disturbance of respiration.

The disease has remarkably little influence upon the higher nervous functions, especially those of the mind. Most patients have a perfectly clear intellect to their latest breath. We all know the contented, hopeful disposition of many patients, who do not recognize their own danger until the last stages of the disease. Occasionally the anæmia and the general disturbance of the nutrition of the brain lead to mental alterations, such as confusion, distraction, or melancholic conditions.

We find, more frequently, disturbances in the peripheral nerves and muscles. Among these are neuralgic pains, and pains of an indefinite character, which have their seat in the legs, or sometimes in the arms, especially in the ulnar region, and which may be very distressing. Marked hyperæsthesia of the skin and deeper parts is also not uncommon. The cause of such disturbances is probably often to be looked for in degenerative changes in the peripheral nerves (Vierordt and others). Well-marked multiple neuritis has been repeatedly observed in tuberculous patients (see section on nervous diseases).

We very often see an increased reaction upon direct mechanical irritation in the emaciated muscles, and great liveliness of the so called idiomuscular contractions, which is shown, for example, on percussing the pectoral muscles on the anterior wall of the chest. The phenomena grouped under the name of tendon reflexes are also much increased in phthisis.

4. **Symptoms and Complications on the Part of Other Organs.**—1. *Pleura.*—In pulmonary tuberculosis the pleura is also involved as a rule. The affection is almost always the result of a direct extension of the process from the lung to the pleura. At the autopsy, we find in the pleura a few or many miliary tubercles, besides the simple inflammatory process—tubercular pleurisy.

In many cases, in which we have to do only with an adhesive pleurisy and with pleuritic contraction, we can merely suspect the disease of the pleura, but it can not be directly made out and differentiated clinically from the pulmonary affection. In other cases we can diagnosticate a dry pleurisy in phthisis from the occurrence of the pleuritic friction-rub. The symptoms of pleurisy become more marked if there is a pleuritic effusion, which is usually readily discovered by a physical examination. The patient's pain and dyspnœa are usually much increased by such a complication. Besides a simple sero-fibrinous effusion we quite frequently find purulent and even hæmorrhagic effusions in tuberculosis of the pleura.

The formation of pneumothorax is an important complication in the pleura in phthisis. This is due to the rupture of a superficial pulmonary cavity into the pleural cavity, and the entrance of air into the latter. The different forms of pneumothorax and its symptoms will be described under diseases of the pleura.

2. *Larynx, Pharynx, and Trachea.*—The symptoms of laryngeal tuberculosis and their relation to pulmonary tuberculosis have already been given under diseases of the larynx. We saw there that, although there is a primary laryngeal tuberculosis, most cases are secondary in their development to a pulmonary tuber-

culosis. The constant passage of tubercular sputa from the lungs, through the trachea and larynx, naturally leads to a direct infection of the mucous membrane of the parts mentioned.

The same holds true in regard to the much rarer tuberculosis of the pharynx. In some cases this may be of primary origin, but it is usually a result of re-inoculation with tuberculosis by means of the sputum, or of a direct extension of the tubercular process from the larynx to the pharynx. Tubercular ulcers of the pharynx are found most frequently on the soft palate, on the tonsils, on the root of the tongue, and on the boundary between the pharynx and the larynx; they are rare in other parts of the pharynx. In exceptional cases tubercular affections are seen in the mouth—on the tongue. The local discomforts which all these ulcers cause is usually very considerable. Disseminated miliary tubercles, too, have been repeatedly seen in the mucous membrane of the pharynx.

3. *Stomach and Intestinal Canal—Peritoneum.*—Tubercular ulcers in the mucous membrane of the stomach are exceedingly rare, but we very often notice some symptoms on the part of the stomach. Loss of appetite is a particularly common symptom in phthisis. Vomiting occurs often in phthisical patients, especially when the larynx is involved. It is usually brought on by paroxysms of coughing. Less frequently the cause of the vomiting is gastric catarrh, occasioned by the irritation of the phthisical sputa which have been swallowed; but in some cases the gastric symptoms depend upon the general condition, like anæmia.

Although the tubercle bacilli swallowed with the sputum hardly ever infect the stomach, probably from the acid reaction of its contents, they very often attack the intestinal canal. In the majority of the cases of phthisis we find tubercular ulcers, either singly or in considerable numbers, in the vicinity of Bauhin's valves [the ileo-cæcal valve], in the lower part of the ileum, and the upper part of the large intestine.

Intestinal tuberculosis does not always cause very marked clinical symptoms, but as a rule we find diarrhœa in patients with tubercular ulcers of the intestine. They may have three or four stools in the twenty-four hours, and even more, but the stools have nothing characteristic. We rarely see a slight admixture of pus or blood in them. Tubercle bacilli have been repeatedly discovered in the dejecta, but the search for them is rather difficult. We must call attention, however, to the fact that many patients have diarrhœa during life in whom we find at the autopsy no intestinal tuberculosis, but only a simple intestinal catarrh, or amyloid of the intestine. On the other hand, we quite frequently find at the autopsy tubercular ulcers of the intestine which during life caused no diarrhœa.

In cases of severe intestinal tuberculosis we sometimes meet with meteorism. With deep ulcers, extending to the peritoneum, we often see marked tenderness of the abdomen.

The peritoneum may be affected by the tubercular ulcers of the intestine in a twofold manner. Genuine peritonitis from perforation, with a purulent or even a sanious exudation, is quite rare, and is excited by the rupture of an ulcer and the entrance of the contents of the intestine into the abdominal cavity. An infection of the peritoneum with the tubercular poison is more frequent. This may arise from deep-seated ulcers, which do not reach actual perforation, so that we have a peritoneal tuberculosis, or a tubercular peritonitis. During life peritonitis from perforation and that from tuberculosis are not always to be distinguished. We must also mention that simple ascitic fluid is sometimes found in the abdominal cavity in phthisis, which may lead to a false diagnosis of peritoneal tuberculosis.

Another way in which we may have a peritoneal tuberculosis in the course of phthisis is from the extension of the process in a tubercular pleurisy, through the diaphragm to the peritoneum.

4. *Liver and Spleen.*—We very often find a few or even many tubercles in the liver in phthisis, but they have no clinical significance. The liver is almost always infected with the tubercular poison from tubercular ulcers in the intestines, from which the poison passes to the branches of the portal vein and then to the liver. Fatty liver and amyloid or lardaceous liver are more important clinical changes. We can sometimes recognize the former by making out on physical examination the increase in the size of the organ, and by feeling its characteristic blunt lower edge.

Amyloid liver is almost always associated with the development of amyloid in other organs. In marked cases the liver is evidently enlarged, and its firm, sharp lower edge, and often its dense anterior surface, may usually be plainly felt.

Miliary tubercles or single large tubercular nodules in the spleen have a pathological interest only. Great splenic enlargement is found in amyloid degeneration.

5. *Kidneys, Urinary Passages, and Sexual Organs.*—The presence of miliary tubercles in the kidneys is the first change in them to be mentioned, but it has no clinical significance. Extensive tuberculosis of the genito-urinary apparatus, however, may produce marked symptoms, like pyuria, which will be described later. In regard to the symptoms of amyloid kidney, which may develop in the course of phthisis in connection with amyloid disease in other organs, we will refer to the section on renal diseases.

Genuine cases of nephritis, both acute and chronic, are also found quite frequently in phthisis, usually combined with amyloid disease. These can not escape notice if the urine is carefully examined. Cases of genuine amyloid kidney of moderate severity are seen, however, in which the urine remains normal, or at any rate free from albumen.

6. *Circulatory Organs.*—Not only is the frequency of the pulse increased in many patients in proportion to the existing fever, but even where there is no fever we often find it accelerated. The greater or less increase of the pulse, which readily comes on from comparatively trifling external causes, is especially noteworthy. It may be seen after slight physical exertion, or upon mental excitement, as during the physician's visit.

Anatomical changes in the heart are rare, except that it is often remarkably small and flaccid. Moderate fatty degeneration of the heart, slight endocarditis of the valves, or occasional tubercles in the heart, cause no symptoms. The occurrence of tubercular pericarditis, however, is important. This almost always arises from the extension of the tubercular process from the adjacent pleura, but in exceptional cases pericarditis has been seen as a result of rupture of a pulmonary cavity into the pericardium.

7. *Lymph-glands.*—The lymph-glands are a favorite seat for tubercular changes. We have stated above that the so-called scrofulous, cheesy lymph-glands, which are seen chiefly in the neck and the axillæ, are affected with tubercle in the majority of cases. The tubercular infection probably develops here from slight injuries and excoriations of the skin, by which the bacilli enter the body and reach the neighboring glands by means of the lymph-current. In other cases the infection perhaps comes from the mucous membrane of the pharynx. In tuberculosis of internal organs, too, we very often find the corresponding lymph-glands enlarged and more or less cheesy. The bronchial lymph-glands are swollen as a result of pulmonary tuberculosis, the mesenteric and retroperitoneal glands as a result of intestinal tuberculosis. The tuberculosis of the bronchial lymph-glands is of especial importance in children. Indeed, the tubercular

virus which has gained access to the lungs may apparently reach the bronchial glands by means of the lymph-channels even without affecting the lungs themselves, and occasion a tuberculous disease of the glands. Glands thus diseased break down and discharge into the lungs, and in this way generate a secondary pulmonary tuberculosis. This is one reason why the pulmonary tuberculosis of children so often begins not in the apex but in the middle and lower portion of the lung.

Pressure from the enlarged glands may affect the air-passages, the branches of the pulmonary artery, the veins, the recurrent nerve, and even the aorta. Perforation of the cheesy bronchial glands into the œsophagus, the blood-vessels, etc., has also been observed. Tuberculosis of the bronchial glands in children does not present any definite type of disease, however, and, although we may sometimes suspect it when there is pulmonary tuberculosis, we can only rarely diagnosticate it with certainty.

8. *Nervous System.*—We have already mentioned various nervous symptoms in the description of the general symptoms. We must also add that tubercular meningitis is seen in the course of phthisis (see page 702), and also that large solitary tubercles may occasionally develop in the central nervous system (see page 749).

9. *Skin.*—We have spoken of the great tendency which many patients have to severe sweats, especially at night. The frequent appearance of pityriasis versicolor, especially on the skin over the thorax, is also worthy of note. We often see moderate œdema of the legs and ankles, which is due to weakness of the heart. More marked œdema of one leg sometimes arises from thrombosis of the femoral vein. We must also mention here, in conclusion, the specific tubercular disease of the skin—lupus. This occurs alone, as a rule, without a co-existing pulmonary tuberculosis; but, on the other hand, the old term "scrofulous lupus" had reference to the fact that we often find other tubercular affections in lupus besides the disease of the skin. Thus it does not seem strange that lupus and phthisis have been repeatedly observed to co-exist. Cutaneous tuberculosis may develop not only in the ordinary form of lupus, but also in nodules of considerable size, or in rather extensive ulcers. Perhaps some of the cases of so-called corpse-tubercle belong to true tuberculosis. We have seen a similar tuberculous cutaneous disease in a woman who for a long time had washed the soiled handkerchiefs of a consumptive patient.

Diagnosis.—The diagnosis of pulmonary tuberculosis has become absolutely certain, since the discovery of the tubercle bacilli, by the demonstration of their presence in the sputum (*vide supra*). In all incipient cases, in which the other symptoms of the disease have not yet made themselves manifest, but where the suspicion of incipient phthisis has been aroused by a persistent cough, by marked pallor and emaciation, by slight hoarseness, by an evening rise of temperature, by the appearance of night-sweats, by the presence of a hereditary predisposition, and similar symptoms, the finding of tubercle bacilli in the sputum is often the sole deciding factor. We must not forget, however, that in most cases the diagnosis may be made from the other symptoms alone, and also that we can actually judge of the severity of the individual case, and of the exact extension and form of the tubercular process, only by considering the other symptoms, and especially by considering the data of the physical examination. The latter, therefore, has lost none of its importance by the discovery of the tubercle bacilli.

Confusion between phthisis and other diseases is twofold. Where the constitutional symptoms are predominant, and there are no marked pulmonary symptoms, an existing tuberculosis may be overlooked. In the beginning, especially, many cases of phthisis are considered to be merely anæmia, chronic gastric catarrh, or simple bronchitis. If an intermitting fever appears in an early stage of phthisis, before any marked pulmonary symptoms have developed, the disease may be mistaken for malaria or the like. On the other hand, it is by no means rare to consider patients phthisical who are suffering from some entirely different affection. He who lays too great stress on the uncertain results of percussion will often make a false diagnosis. Severe latent diseases of the stomach, or certain general diseases, such as anæmia, diabetes, or chronic nephritis, may falsely be taken for phthisis. Other pulmonary affections, too, may be confounded with tuberculosis, especially chronic bronchitis, emphysema, bronchiectasis, fœtid and gangrenous processes, and carcinoma of the lungs. A careful, unprejudiced, and complete examination of the patient is the only possible protection against such errors.

Prognosis.—In the present condition of our therapeutic knowledge the prognosis of phthisis must unfortunately still be regarded as extremely unfavorable. There can be no doubt that the tubercular process in the lungs is in itself curable, but, in all cases where we get certain objective evidence of tuberculosis, a definite extinction of the disease is very improbable, because the conditions for a further extension of the disease in the lungs are extremely favorable. In individual cases, however, with the best hygienic surroundings, circumscribed tubercular affections of the lungs have been certainly found to heal. Indeed, it is possible that recovery from pulmonary tuberculosis is more frequent than was formerly supposed; for it is precisely in the earliest stages, when the disease is still curable and tubercle bacilli have not yet been discovered, that the diagnosis is difficult and often uncertain. In advanced cases of phthisis the prognosis, of course, is almost absolutely fatal.

In individual cases the prognosis with regard to the duration of the disease is very difficult. Here we must always be mindful of the great differences in different persons, and hence we must be very cautious in expressing an opinion. How many patients with phthisis give us the impression on the first examination that they can not live a fortnight longer, and later on we see the disease lasting for many months, most of the symptoms improving, and the patient recovering again! In other cases, on the contrary, we think we have to do with an incipient case, and give great encouragement—and the patient dies in a few weeks with florid phthisis. Even disregarding the constant possibility of an unforeseen fatal hæmoptysis, pneumothorax, or tubercular meningitis, a conjecture as to the duration of the disease is, therefore, very uncertain in most cases which have not yet reached the last stage, and should be made only after long observation of the patient. Very much, of course, depends upon the circumstances of the patient, and upon the possibility of good care, suitable food, and good air.

Of the single factors which influence the prognosis, our chief attention must be paid to the general nutrition, the patient's weight, the extent of the pulmonary affection, the fever, and certain complications, especially laryngeal and intestinal tuberculosis. We need not repeat the special points which relate to these.

[Flint, especially, has called attention to the fact that phthisis is sometimes self-limited; that recovery or arrest takes place without any special treatment, and in spite of relatively unfavorable surroundings. The clinical evidence is greatly strengthened by the frequency with which, after death from any cause, the remains of an old phthisical affection are found.

In 1857, 39·50 deaths from consumption were returned in the State of Massachusetts for each ten thousand of the population; in 1883, 29·90. This decrease is too large to credit to greater accuracy in diagnosis and to the transference of consumptives to other States, and is mainly attributable to the prevention of phthisis by improved hygiene. Still, it seems fair to carry some of the improvement to the account of the arrest and cure of actually developed disease through early diagnosis and more rational treatment, hygienic as well as medicinal.]

Treatment.—1. *Prophylaxis.*—The question of what prophylactic measures may effectually prevent the extension of the disease has entered upon a new stage since our definite knowledge as to the infectious nature of tuberculosis. We can no longer doubt the contagious character of phthisis, in support of which isolated examples were previously brought forward. Even if, according to all experience, the danger of contagion is not very great, still it is foolish to ignore it entirely. We must therefore call the attention of the relatives of phthisical patients to the possibility of this danger, and we should not permit the children of such patients to be uselessly exposed to it. We should take satisfactory precautions for isolation, and also for disinfection of the sputum, which can best be done by a strong solution of carbolic acid. The future will teach us whether many evils may not be averted by such measures, although now these measures are greatly neglected.

The "prophylaxis" at present employed is almost exclusively confined to hardening and strengthening the threatened individual as much as possible. We should try to strengthen the bodies of children of a weak habit, with "scrofulous" symptoms, and children from families in which cases of tuberculosis have already occurred, and thus to arm them against the enemy that threatens them. Good food, fresh air, and a diminution of the sensitiveness of the body by cold sponging and cold baths—these are the factors whose favorable influence is generally recognized.

The removal of certain foci of tubercular disease, already existing, from the body may prove of great prophylactic importance. We refer to the timely treatment or extirpation of scrofulous—that is, tubercular—swellings of the lymph-glands, healing or resection of tubercular bones and joints, etc. Although in individual cases we can of course never know whether the part removed is the sole focus of disease in the body, still we are undoubtedly justified in trying to remove at least one possible source of some later general infection. A fuller discussion of this important point must be left to the works on surgery.

2. *Treatment.*—We do not yet know any actual treatment, corresponding to the causal indication, whose point of attack is aimed directly against the tubercular poison. The inhalations employed with this idea, containing antiseptic substances, like carbolic acid, benzoate of sodium, and lately iodoform, have so far all proved unsatisfactory, chiefly because the substances inhaled have not reached the bronchi in sufficient amount. Inhalations of iodoform, made by a special apparatus invented by Küssner, have been the most successful in tuberculosis of the larynx. We have already spoken of inhalations with astringents and narcotics.

We must make especial mention of arsenic among the internal remedies to which a specific action in tuberculosis has been attributed. Our own experiments, which were suggested by quite a number which were made by Büchner, have in general given no favorable result. In some cases, however, the remedy seems to have a marked therapeutic action, so that we are always justified in trying arsenic in incipient cases, the more especially since it has lately been observed to have a favorable influence in other tubercular affections, like tuberculosis of the lymph-glands, caries, and lupus. It is better not to prescribe arsenic in solution, but in pills of a twentieth of a grain (grm. 0·003) of arsenious acid, giving two or three a day, and later four or five if possible, always after eating. Good results can be obtained only after using the remedy for at least some months. Another internal remedy which has been much talked about of late years is creasote. It may be given in pills, or combined with cod-liver oil, as in the following formula:

℞ Creasoti.................................. gr. xv (grm. 1) ;
Olei morrhuæ..................... ℥ iij ʒ ijss. (grm. 100) ;
Olei menthæ piperitæ............................. gtt. 2. M.

Three or four teaspoonfuls daily.

Or it may be given in gelatine capsules, which usually contain five sixths of a grain of creasote (0·05) combined with a third of a grain of balsam of tolu (0·02). The patient should take six to twelve, or even more, of these capsules a day. Creasote may also be administered in milk. A certain improvement of the symptoms under the use of creasote, especially with regard to cough and expectoration, can not be denied. The exaggerated expectations which many physicians cherished concerning this remedy will scarcely, however, be fulfilled.

The hygienic and symptomatic treatment of phthisis is more important than the remedies so far mentioned.

The hygienic treatment, in the broadest sense of the word, aims, on the one hand, to increase the power of resistance of the body to the disease, and, on the other, to put the body under conditions which we know can combat the further extension of the disease. We try to aid, so far as possible, the process of spontaneous recovery in phthisis. First to be mentioned here is the diet of the patient, which must be as nutritious and abundant as possible. Meat, milk, eggs, farinaceous food, and butter are to be used chiefly, special care being taken that the body gets hydrocarbons and fat in sufficient amount, as well as plenty of albumen. Many of the special "cures" for phthisis have their only value in the fact that in these places the patient takes a large amount of easily assimilated nutriment, like milk, and also koumyss—which is, properly speaking, mare's milk fermented in a peculiar fashion, although with us it is artificially made from cow's milk—and also kefir, which is similar to koumyss. French physicians have lately brought forward forced feeding as a special "method" of treating consumption. It consists in giving the largest possible amounts of nourishment (pulverized meat and the like), and sometimes the food is even poured down through a stomach-tube. In ordering milk-cures we must not forget that milk soon becomes repugnant to many patients, and then it can no longer be taken in sufficient amount. In such cases we sometimes succeed in making the milk palatable to the patient by adding common salt, cognac, or coffee. It is also a good way to have an infusion of tea or coffee made with hot milk. Many patients prefer this to pure milk. As regards alcohol, we should chiefly prescribe those forms of malt liquor which are relatively rich in nutriment—e. g., porter. Small amounts of good wine may aid in improving the appetite and the general condition. Many physicians indeed believe that the stronger alcoholic beverages, such as brandy and the like, are of special benefit in the treatment of consumption, but we doubt it. Cod-liver oil, two to four tablespoonfuls a day, is a prescription which furthers the patient's nutrition, and, if it be well borne, it may be of distinct service, especially in emaciated patients. Still, we believe that one may obtain the same success by the regular daily administration of several tablespoonfuls of pure fresh cream.

We must also pay attention to the patient's manner of living, as well as care for his nutrition. Here we must endeavor, on the one hand, to remove all the injurious influences connected with his occupation, like staying in badly ventilated counting-rooms and work-shops, inhaling dust, or excessive speaking, and, on the other hand, must give him such directions as will have a favorable action on the whole body, and especially on the respiratory organs—the enjoyment of good air, free from dust, sponging the chest with cold water, and baths; but since we often can not satisfy all these demands under the patient's ordinary household conditions, it has long been the custom to send patients with thoracic diseases to certain special health-resorts, where the conditions for a prescribed manner of living may be fulfilled more completely than at home. On this depends the so-called climatic treatment of phthisis. Many physicians assert that certain climatic factors, like temperature, moisture, and atmospheric pressure, exert a specific therapeutic influence; but this view does not seem very probable.

In regard to the choice of a temporary resort for the summer, in many cases we must content ourselves with recommending a country residence for the patient, in a region as healthy as possible, protected, dry, and well wooded, paying attention at the same time to the character of the board and lodging there. A good country boarding-place may do just as well as many expensive health-resorts. We may mention in particular, among the special health-resorts, springs, and places for inhalations in Germany, (1) the acidulous saline waters at Ems, Gleichenberg, Neuenahr, Obersalzbrunn, Reinerz, and others; (2) the chloride-of-sodium waters at Reichenhall, Salzungen, Soden, and others; (3) the springs containing the earthy salts at Inselbad, Lippspringe, and Weissenburg. The favorable influence of these baths is best seen in cases where there is anorexia or other gastric disturbance. Besides these we may also mention some of the well-known high climatic health-resorts in the Alps: Aussee, Beatenberg, Berchtesgaden, Engelberg, Gmunden, Heiden, St. Moritz, Seelisberg, and others; and in the Black Forest: Badenweiler, St. Blasien, Rippoldsau, and others.

The choice of a winter health-resort is, under some circumstances, of still greater importance, since the colder season with us brings with it many dangers for the patient. The first to be mentioned here are the high health-resorts, where the weather is usually clear and sunny. Among these Davos enjoys the greatest reputation. This place is especially suited for patients who are still quite strong and free from fever, and who do not suffer from laryngeal symptoms. Among the winter health-resorts in Germany we must mention first Görbersdorf, and also St. Blasien. The southern climate is better for delicate, "erethistic" patients, and also for those with laryngeal affections. Of course only the very distant health-resorts in Algiers, Egypt, Malta, and the much-praised Madeira, can furnish a certain guarantee of constant mild weather. The Sicilian health-resorts (Catania and Palermo), and also Ajaccio and Pau, afford favorable climatic conditions; while the health-resorts of the Riviera (see p. 153), Meran, Areo, Lugano, and Montreux, are much more uncertain in this respect, and therefore are to be used merely as stopping-places by the way during the spring and autumn months.

We can not go into a full description here of all the health-resorts mentioned. We can not omit, however, calling special attention to the fact that we should always ask ourselves, in choosing a health-resort, whether the expense and inconvenience thus imposed upon the patient can be balanced by the possible benefit. The earlier the stage of the disease and the better the general condition of the patient, the more will his physician be justified in urging him to make every sacrifice in order to regain his health. It is blameworthy, from a professional and humane stand-point, to send patients in the last stages of phthisis among strangers, to die far from their home and relatives. For severe cases, when we wish to send them from home, the special institutions are the only suitable places, where the patient may at least be under the constant care and attention of a physician. Especial asylums for lung patients are Falkenstein on the Taunus, Görbersdorf, Inselbad at Paderborn, and Reiboldsgrün.

[Our own health-resorts for consumptives are too well known to demand extensive consideration here. The prime object is to secure for the patient a pure air, with such climatic conditions that he can pass the largest amount of time out of doors, at the same time that within doors his comfort is provided for, and a sufficiency of suitable and well-cooked food is attainable. In Colorado, New

Mexico, and California, large numbers of former consumptives are leading active lives. Florida, Aiken, Thomasville, Asheville, and some other southern resorts, are good winter asylums for many cases, but patients should not return to New England before June 1st. An out-door life in the Maine or Adirondack woods during the warmer months is highly to be recommended for early and otherwise suitable cases. Saranac, N. Y., affords every comfort during the colder months, combined with the very best medical attendance, and many people do well there. A relative disadvantage under which nearly all American health-resorts labor, as compared with those of Europe, consists in the greater difficulty in providing occupation and thus securing a mental attitude most favorable to recovery. In general, the northern sea-board is much less favorable than the interior, and early cases often do well during the winter removed from the dampness of the coast, with its alternations of freezing and thawing. A change of climate is a very important step, and should receive the most careful consideration of the physician—the circumstances of the patient, the stage and character of his disease, his tastes, etc., being carefully weighed before a decision is reached.]

We must also state that, in incipient cases, a residence by the sea, or a long sea-voyage, may sometimes be of great help. We have ourselves known several young physicians who have become ship-surgeons on account of incipient phthisis, and who have returned from the voyage much stronger, and some of them apparently entirely well.

The symptomatic treatment of phthisis is directed in the first place against the pulmonary symptoms. We use much the same remedies to help the cough as in chronic bronchitis. We try inhalations with a solution of common salt, or of the alkaline carbonates, or, where there is much secretion, with solutions of tannin and the balsams, like turpentine, or balsam of Peru. Where there is severe, spasmodic cough, inhalations with narcotic solutions sometimes give some relief, such as cherry-laurel water, opium, or bromide of potassium. It is doubtful whether the inhalation of nitrogen, recommended by many physicians, has any real therapeutic value. Pneumatic treatment, by inhalations of compressed air, may sometimes give good results in cases of incipient phthisis.

Morphine stands first among the drugs employed to check the cough. We should be cautious and sparing in its use at first, but it is an indispensable remedy in severe and hopeless cases. It relieves the irritation of coughing, the pain and the oppression in the chest, and at least gives the patient for a time the desired sleep. In chronic cases, with moderately severe symptoms, we may use for a long time the milder narcotics with advantage, like extract of hyoscyamus (extracti hyoscyami 1, aquæ lauro-cerasi 20, fifteen to twenty drops every two hours), one to three grains (grm. 0·05–0·20) of lactucarium in powder, or half a grain to a grain (grm. 0·03–0·05) of extract of belladonna in powder.*

If the patient complains of difficulty in loosening the expectoration, we prescribe expectorants, the action of which often fails to meet our desires, but which can not be dispensed with in practice. The expectorants most frequently used are carbonate of ammonia, ipecacuanha, apomorphine, and sulphuret of antimony.† We very often combine expectorants with narcotics, as in Dover's powder.

If severe pain in the chest comes on, we often use local applications: mustard plasters, warm poultices and cold compresses, painting with iodine, or embrocations of chloroform. Narcotics, like morphine, are indispensable in severe dyspœna, which usually occurs only in the last stages of the disease or as a result of pneumothorax.

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* Extractum belladonnæ, U. S. P., is about twice the strength of the German preparation.—Trans.

† We should use tartar emetic instead of the sulphuret.—Trans.

The treatment of hæmoptysis is important. As a slight admixture of blood in the expectoration often precedes a severe hæmoptysis, such an event always demands caution. The patient must keep as quiet as possible, and avoid hot drinks and alcohol. When there is a severe hæmoptysis, absolute rest in bed is especially necessary. We should avoid any careful examination of the lungs, especially any severe percussion. We should lay a flat and not too heavy ice-bag over the lung on the side from which we suspect the hæmorrhage; the cold is usually well borne, but sometimes it aggravates the cough, and must then be omitted. We would also recommend swallowing bits of cracked ice. Narcotics, like morphine, are the most suitable internal remedies, since they aid the cessation of the hæmorrhage by suppressing the attacks of coughing. Among the remedies which may be employed to check the bleeding are ergotine, two or three one-grain pills (grm. 0·05) every hour; also sclerotinic acid, thirty to forty-five minims of a four-per-cent. solution subcutaneously in the twenty-four hours; and one or two grains (grm. 0·05–0·10) of acetate of lead in powder every two hours, sometimes combined with morphine. The liquor ferri chloridi (a tablespoonful of a two-per-cent. solution every one or two hours) is also recommended, but in this form it is probably entirely useless. Common salt is a remedy which sometimes seems to be of service, and which is almost always at hand. We give one or more teaspoonfuls of it in water. Acids, lemonade, and Haller's acid elixir [mixtura sulfurica acida (P. G.)] are favorite household remedies in hæmoptysis.

After the hæmorrhage ceases, the patient must be extremely careful for a long time, since it often recurs.

The hectic fever of phthisis is characterized by its great resistance to antipyretic remedies. It is usually utterly useless to try to combat it with large doses of quinine or salicylate of sodium. The action of antipyrine, too, is only temporary. Cold sponging of the whole body is to be highly recommended at the times of rise of temperature. It is usually well borne, and gives the patient visible relief and refreshment.

Cold sponging often diminishes the troublesome sweats in phthisis, but if this does not check them, we may often prescribe atropine to advantage, gr. $\frac{1}{120}$ to $\frac{1}{60}$ (grm. 0·0005–0·001) at night, but its action does not usually last very long. Lately agaricine in $\frac{1}{12}$- to $\frac{1}{6}$-grain pills (grm. 0·005–0·01) has been recommended for the night-sweats in phthisis; also picrotoxin, of which gr. $\frac{1}{8}$ to $\frac{1}{6}$ (grm. 0·008–0·01) is given in pill or solution at bed-time. Dusting the body with a powder of five parts of salicylic acid to ninety-five of French chalk is also a good thing. Sage tea is a favorite remedy for night-sweats—two or three cups of it cold at night—and so is milk and cognac.

If there is loss of appetite, small doses of quinine, compound tincture of cinchona, wine of cinchona, and other bitter remedies, like tinctura amara (P. G.), are sometimes of service. It is also frequently a good thing to prescribe a little muriatic acid, five to ten drops of the dilute acid, with the meals. It is often very hard to check diarrhœa in phthisis. Opium, combined with tannin or acetate of lead, is most effective. This subject will be discussed more fully in the chapter on intestinal tuberculosis.

We often prescribe preparations of iron, combined sometimes with quinine or arsenic (*vide supra*), in the beginning of the disease to improve the general condition and the anæmia, but, as experience shows, iron is contra-indicated in patients who are feverish or who have a tendency to hæmoptysis.

The treatment of the diseases complicating phthisis is to be found in the appropriate chapters.

CHAPTER VII.

ACUTE GENERAL MILIARY TUBERCULOSIS.

Ætiology.—Acute miliary tuberculosis is a form of tuberculosis which we are justified in describing particularly because of its anatomical relations and of its peculiar clinical history. The disease is characterized anatomically by the extremely abundant development of miliary tubercles in a comparatively short time in many organs of the body. We can not liken this process to anything but an overfilling of the body with tubercle bacilli, which in some way reach the different organs at the same time, and there give rise to the eruption of tubercles. Buhl advanced the hypothesis a long time ago, that a cheesy focus could be found somewhere in the body in every case of acute miliary tuberculosis, and that the general infection of the body resulted from the absorption of these cheesy masses by the blood. Later investigations, however, have given us a much more definite explanation of the nature and manner of this general infection. Ponfick first found, in some cases of acute miliary tuberculosis, an extensive tuberculosis of the thoracic duct, with destruction of the tubercular new growth. It is easy to see how, in this way, a large amount of tubercular material could be brought directly into the circulation, from the free communication of the lymph-duct with the subclavian vein, and thus be "disseminated" through the different organs in a short time. Still more frequently, however, the tuberculosis of the large venous trunks, discovered by Weigert, especially the pulmonary veins, seems to be the starting-point for an acute general miliary tuberculosis. Usually there are tubercular lymph-glands, or sometimes other foci of tubercular disease, which unite with the wall of a neighboring vein, gradually break through it, and project into its lumen. If caseation and ulceration result in this spot, the infectious material is of course constantly washed off by the blood-current and carried away, and thus it reaches the other organs.

Since such a tubercular focus—e. g., a tubercular bronchial gland—may remain for a long time entirely without symptoms, we can understand how miliary tuberculosis may break out in an acute form in persons who previously seemed perfectly well. In other cases the patient has already suffered from some tubercular affection, until suddenly the conditions occur somewhere in the body which lead to the development of miliary tuberculosis. Thus we sometimes see it break out in a patient who has ordinary phthisis, but acute miliary tuberculosis is one of the rarities in advanced phthisis. If we find, at the autopsy of a case of acute general miliary tuberculosis, old phthisical changes in the lungs, which is by no means very common, they consist of old, partly cicatrized foci, pigment indurations, etc. We see miliary tuberculosis rather frequently as a sequel to pleuritic effusion. We have already previously called attention to the fact that in such cases the pleurisy itself is a tubercular disease. Miliary tuberculosis is also seen in people with old tubercular affections of the bones and joints, like coxitis and vertebral caries, with tubercular swellings of the lymph-glands, as in the neck and the axillæ, or with tuberculosis of the genito-urinary organs. In such cases, of course, the tubercular affection, which is discovered during life, is not always the source of the general miliary tuberculosis, but the discovery of the existence of such an affection is of the greatest significance in diagnosis, as in this way our attention is strongly directed to the possibility of a general tubercular affection.

In some cases an outbreak of miliary tuberculosis has been seen to follow other acute diseases, like typhoid, or measles.

Pathological Anatomy.—Except for the presence of some old tubercular affection in some organ, and except for the tuberculosis of a vein or of the thoracic duct, which are as a rule apparent, and which have been described above, the anatomical lesion in acute miliary tuberculosis consists in the dissemination of miliary tubercles through a large number of the organs of the body. The lungs, the liver, and the spleen are constantly affected; almost as constantly the kidneys, the thyroid gland, the marrow of the bones, the heart, and the choroid; less constantly, but still quite frequently, the serous membranes and the meninges. The miliary nodules may be found in large numbers in all the organs mentioned. They may in part be easily recognized by the naked eye, and in the lungs they may be very plainly perceived by the touch. In many organs, however, especially in the liver and often in the spleen, they are hard to recognize with the naked eye, but they are easily discovered by the microscope. In regard to the histological structure of miliary tubercles, and the discovery of tubercle bacilli in them, we must refer to what has been said in the chapter on pulmonary tuberculosis, but we must also mention that, in some of the more chronic cases, some of the nodules may grow to be large tubercular foci, from the size of a lentil to that of a pea. Less developed cases of miliary tuberculosis are also found, in which only a limited number of organs are attacked, and these with less severity.

Clinical History.—The clinical symptoms of miliary tuberculosis depend upon two factors, the first being the general infection of the body, and the second the local tubercular affection of certain organs. Although in many organs miliary tuberculosis is entirely without symptoms, as in the liver, the kidneys, the heart, and the marrow of the bones, in two organs—the lungs, and more especially the brain—it leads to the most marked local symptoms. The miliary tuberculosis of the choroid, discovered by Cohnheim and Manz, is also without symptoms, but it can be made out with the ophthalmoscope, and is therefore of great diagnostic value.

Miliary tuberculosis affords quite different pictures, according to the predominance of one or the other of the groups of symptoms mentioned. We distinguish the four following forms:

1. *Miliary Tuberculosis, with Predominant Symptoms of General Infection: the so-called Typhoid Form.*—This form may in part greatly resemble typhoid fever. The patient, who previously seemed quite well, or in whom some local manifestation of tuberculosis was suspected, falls ill with gradually increasing general symptoms, dullness, loss of appetite, headache, and fever. Since there is no local affection to be discovered to explain the symptoms, the disease at first may well be taken for typhoid. The general condition grows worse constantly, the fever is high and continually rises, and cerebral symptoms appear. In some cases an exanthematous eruption, like roseola, may increase the resemblance to typhoid. With careful observation, however, symptoms are almost always detected later in the disease which are, to a certain degree, characteristic of miliary tuberculosis, and are due to the existence of that disease either in the lungs or in the brain. The patient's complexion assumes a peculiar pallor, and with it a definite cyanotic hue. The respiration becomes remarkably deep, and there is dyspnœa, or signs of a tubercular meningitis arise, like rigidity of the neck, loss of consciousness, disturbances in the innervation of the ocular muscles, etc., and death follows with these symptoms. These cases last from ten days to three weeks, reckoning from the beginning of the severe symptoms.

2. *Miliary Tuberculosis with Predominant Pulmonary Symptoms.*—These cases, too, may begin quite suddenly, almost like an acute croupous pneumonia, or they may develop gradually with quite a long prodromal stage. From the onset the symptoms point especially to disease of the lungs or the pleura. The patient complains of a stitch in the side, cough, and dyspnœa, and there is also usually much general weakness and fever. Later on, the pulmonary symptoms

constantly increase. The patient has extreme dyspnœa, and we can often make out an intense diffuse bronchitis on examining the lungs. The patient's face is pale, cyanotic, and anxious. Death ensues with all the signs of insufficiency of respiration. The course is usually somewhat more protracted than in the typhoid form, lasting for three or four weeks and more.

3. *Miliary Tuberculosis with Predominant Cerebral Symptoms, due to Tubercular Meningitis.*—Tuberculosis of the meninges does not belong among the regular lesions of general miliary tuberculosis. It develops in about half the cases, according to our estimation; but where it occurs it almost always gives the whole case the characteristic imprint of tubercular meningitis, by which the other symptoms are entirely concealed. The predominant symptoms are headache, fever, stupor increasing to deep coma, rigidity of the back and neck, and disturbances in the innervation of the ocular muscles. In such cases the tubercular meningitis is often the only thing diagnosticated, and the general miliary tuberculosis is not made out at all. In the cases of this sort which we have seen, a remarkable, peculiarly deep and accelerated respiration, even in the deepest coma, was the only symptom which pointed to a co-existing miliary tuberculosis of the lungs.

The symptoms of tubercular meningitis in many cases predominate in this type of the disease from the onset, but in other cases they come on during the attack and form its final period. The duration of the disease varies accordingly.

4. *Miliary Tuberculosis with a Protracted Course and Indefinite Symptoms for a Long Time—Intermitting Form.*—Besides the forms already mentioned, cases occur which usually take quite a protracted course, lasting for eight or ten weeks, and having such indefinite symptoms that a definite diagnosis is for a long time, or even throughout the disease, quite impossible. The patient complains of a number of general symptoms, such as headache and dullness, and also of thoracic symptoms, for which, however, we can find on examination no sufficient basis. There is almost always fever, usually not very high, and with a very irregular course; but we have seen a regular daily rise of temperature for a time in some cases, and attacks of fever with quite a severe chill, so that at first we thought of an irregular intermittent fever—the intermitting form. Later on the symptoms gradually increase. The apparently inexplicable loss of strength, and the patient's anæmia and emaciation, are marked, and they are important in diagnosis. Finally, either severe pulmonary symptoms or the signs of tubercular meningitis set in, to which the patient succumbs.

We must mention particularly that the four forms of miliary tuberculosis just described are only the types of the disease. In individual cases we often meet with variations and transitional forms between these types.

Single Symptoms.—1. *General Symptoms.*—In all cases of acute miliary tuberculosis the general condition of the patient is very serious. Most patients have a subjective feeling of severe illness, although they make little special complaint of it from the painless character of the disease. As the disease increases, there is often a marked feeling of anxiety and oppression besides the dyspnœa. There is, especially in the face, quite a peculiar pallor, characteristic of the disease, and associated with a marked cyanosis of the lips and cheeks.

2. *Fever.*—Acute miliary tuberculosis almost always runs its course with a more or less high fever, a course without fever having been observed in only a few instances. It often happens, in more protracted cases, that the temperature may be nearly normal for a time, or only slightly elevated. There is nothing characteristic or typical in the course of the fever. In the cases with typhoid symptoms the fever is usually quite high, between 103° and 105° (39·5°–40·5° C.), and rises continually, so that the temperature curve may be exactly like that of ty-

phoid. In other cases the fever is irregular and is broken by many remissions, remitting or intermitting quite regularly for some time. Death ensues with a moderately high temperature or in collapse. In cases with tubercular meningitis there is also a marked rise of temperature at the close, up to 108° (42° C.) and over.

3. *Respiratory Apparatus.*—It goes without saying that physical examination of the lungs may give no definite results. Almost every positive evidence is often wanting, and the contrast between the labored breathing and dyspnœa and the insignificance of the physical signs in the lungs is an important feature in diagnosis. Auscultation, as a rule, gives the signs of an intense bronchial catarrh; we hear rhonchi or numerous small and medium moist râles all over both lungs. The respiratory murmur itself is usually higher in pitch than normal, and in many cases it is obscure, rough, or harsh. In one of our cases there was heard, over a circumscribed area of the lung, a wholly peculiar, sharp, "lapping" sound on inspiration, which we have never heard under any other circumstances. Jürgensen describes a soft friction sound, due to miliary tuberculosis of the pleura. Percussion usually gives no objective changes. At times the resonance is rather tympanitic, or slightly dull in some places.

In some cases circumscribed pneumonic infiltration has been observed in the lungs in acute miliary tuberculosis, which may give rise to a confusion between miliary tuberculosis and croupous pneumonia, from the presence of marked dullness, crepitant râles, and bronchial respiration.

We must mention, finally, that in some of the cases physical examination of the lungs shows old changes in them, a phthisical affection of the apex, a former pleurisy, and the like. Positive evidence of such old tubercular affections may be of great diagnostic value in doubtful cases.

Dyspnœa has been repeatedly mentioned among the symptoms in the lungs. The respiration is usually very much accelerated, especially during the more advanced stage of the disease, so that we see in adults forty, sixty, and even seventy respirations a minute. The respiration is also very deep, and is sometimes associated with a loud noise. As a rule there is cough, but it is usually troublesome only in the cases with severe bronchitis. It is often very slight. The expectoration is usually scanty, and it is not characteristic. Special mention must be made of the fact that tubercle bacilli are absent in it, unless old ulcerated tubercular foci are present at the same time in the lungs.

4. *Circulatory Apparatus.*—The pulse is frequent, about 100 to 120 a minute, often weak and small, and sometimes irregular, especially if tubercular meningitis co-exists. The miliary tubercles, which anatomically are almost always to be made out in the heart, especially in the endocardium, cause no symptoms. The presence of tubercle bacilli in the blood will be mentioned below.

5. *Digestive Apparatus.*—Vomiting is frequent at the onset of the disease. The bowels are usually constipated, but in many cases there is a moderate diarrhœa. The loss of appetite, the thirst, and the dry tongue are due to the general disease and the fever. The spleen is usually somewhat, but not very much, enlarged.

6. *Nervous System.*—In many cases where the pulmonary symptoms predominate the intellect remains quite clear until the last, but in other cases cerebral symptoms, like headache, dizziness, stupor, and delirium, come on quite early, and are a part of the general infection. As has already been said, the nervous symptoms in the cases combined with tubercular meningitis become quite prominent, but in individual instances it may be hard to decide whether they are due to such a complication, or are merely severe general symptoms.

7. *Eyes.*—The ophthalmoscopic examination of the retina is of special diag-

nostic importance, since the diagnosis may be made absolutely certain by finding miliary tubercles in the choroid. A negative result, however, is never decisive against the diagnosis, since the tubercles are sometimes absent, or at least are very scanty. Their discovery is almost always difficult, and demands much practice in the method of examination. In cases with tubercular meningitis we sometimes find an optic neuritis.

Diagnosis.—The diagnosis of acute general miliary tuberculosis is ordinarily and justly considered very difficult. It quite often happens that at the autopsy a miliary tuberculosis is found which was not even suspected during life. It must be confessed that frequently, in such cases, we might very well have thought of acute tuberculosis. If, therefore, the possibility of acute miliary tuberculosis is brought to our attention during the patient's life, we can occasionally make quite a certain diagnosis.

The severe general condition, usually associated with fever, is most important, and for this no local cause can be found. Then come the pulmonary symptoms, especially the peculiar dyspnœa, for which there is also no adequate corresponding physical change to be discovered. It gives decided support to our suspicion if we can make out a distinct predisposition to tuberculosis, either hereditary or constitutional, or the history of a previous tubercular affection, especially pleurisy, and also chronic affections of the bones. The peculiar cyanotic pallor of the patient is very characteristic.

On these factors rests the differential diagnosis between the "typhoid" form of miliary tuberculosis and typhoid fever. Marked roseola is a distinct argument for typhoid, although it sometimes occurs in miliary tuberculosis, and so are the characteristic intestinal symptoms of typhoid, like meteorism and the peculiar stools; but we must not forget that both the roseola and the intestinal symptoms may be absent in typhoid. The course of the fever must always be considered in the differential diagnosis. It is much more frequently irregular and atypical in tuberculosis than in typhoid. Of course, the temperature curve is not an absolutely decisive factor. The occasional unequivocal evidence of the ophthalmoscopic appearances has already been mentioned.

In many cases the onset of meningeal symptoms may aid the diagnosis. Of course, if the patient is not seen until the last stages of meningitis, especially where there is an incomplete history, the diagnosis is next to impossible.

Acute tuberculosis is often confounded with severe bronchitis, especially in old people who are considered emphysematous. The very severe general condition, the pallor, the rapid loss of strength, and the fever, are the only things here which call our attention to acute tuberculosis, and render the diagnosis possible.

Finally, the discovery of tubercle bacilli in the blood is of the greatest diagnostic significance. Their detection is a difficult matter, but Weichselbaum and others have repeatedly succeeded in finding them.

Prognosis.—The cases described in literature as "cured miliary tuberculosis" are so uncertain in their diagnosis that they can not be regarded as convincing. We must therefore consider the prognosis as absolutely fatal. The differences in the course of the disease have been mentioned above.

Treatment.—Although drugs are absolutely powerless, still the case in hand must always receive treatment, since the diagnosis often can not be made out with absolute certainty. Our prescriptions are purely symptomatic. The cases with a typhoid course are to be treated just like typhoid, with baths, stimulants, etc. Tepid baths, and also local applications to the chest, expectorants, and narcotics, are indicated when the thoracic symptoms predominate. If meningeal symptoms set in, we may try ice, and also local blood-letting, with iodide of potassium internally.

CHAPTER VIII.

GANGRENE OF THE LUNGS.

Ætiology.—The sole cause of pulmonary gangrene—that is, the death and putrid decomposition of the lung-tissue—is the entrance of the bacteria of putrefaction into the lungs. The opportunity for inhaling them is certainly very great, but the normal organism apparently possesses the property of nullifying them and making them powerless. Under certain conditions, however, they take root in the lung and cause the death of the pulmonary parenchyma, which then, as a result of the presence of these specific bacteria of putrefaction, succumbs to that peculiar form of putrid decomposition known as "moist gangrene."

The factor which most frequently gives rise to the development of pulmonary gangrene is the entrance of organic foreign substances, especially bits of food, into the lungs. The bacteria of putrefaction either enter the lungs with the foreign substance, or they settle there later and set up a putrid decomposition, first in that portion of the lungs, and then in the neighboring lung-tissue. The entrance of organic foreign substances into the lungs occurs in different ways. It often happens from swallowing the wrong way, or from an accidental inhalation. In this way pulmonary gangrene may arise in previously healthy people, but it occurs especially in patients who are very low, very stupid, and soporose, and also in the insane, in patients who can not swallow or cough well, and in patients with paralysis of deglutition, as in bulbar paralysis. Bits of food may also reach the lungs from eructations and vomiting. Thus are explained the cases of pulmonary gangrene which occur in patients with cancer of the stomach, and, still more frequently, with cancer of the œsophagus. Putrid organic material may also reach the lungs from ulcerative and ichorous processes in the mouth, the pharynx, and the larynx. In cancer of the tongue, the pharynx, and the larynx, in other ulcerative processes, and in injuries or wounds from operations in the mouth and pharynx that have become septic, pulmonary gangrene may develop quite readily. Finally, septic foci in the vicinity may extend to the lungs or perforate into a bronchus. In this way a pulmonary gangrene may arise from the perforation through the pleura into the lungs of an ulcerated cancer of the stomach or a gastric ulcer, or in rare cases from vertebral caries, or from sanious lymph-glands.

In some cases the cause of the pulmonary gangrene can not be made out, since the entrance of a foreign substance has perhaps been wholly unnoticed, as may happen in children, or during sleep. We had a grown-up young woman under observation for a long time with pulmonary gangrene, and one day she coughed up several fragments of chicken-bones, but she could give no account of how they entered the lungs.

Experience teaches us that pulmonary gangrene is more apt to develop in persons with a general weak nutrition, in old, marantic people and drunkards, than in those who are healthy. The tendency of patients with diabetes mellitus to pulmonary gangrene is remarkable.

Pulmonary gangrene often develops secondarily to some other pulmonary affection. We have already spoken of the relations between it and fœtid bronchitis. Fœtid bronchitis, on the one hand, often leads to pulmonary gangrene through an extension of the process to the alveoli; and, on the other hand, where there is a gangrenous focus in the lungs, the bronchi are often infected to a wide extent by the putrid secretion coming from it, and then there arises fœtid bronchitis. Both diseases often run into each other without any sharp boundary; but gangrene may develop secondarily in other affections of the lungs. A new infection with putrid material, however, is always requisite, and the affection of the lungs that already exists furnishes merely a favorable soil. This is the only explanation of the process when croupous pneumonia "runs into gangrene," or when gangrene develops in catarrhal pneumonia, in bronchiectasis, or in phthisis.

Although the agents of putrefaction enter the lungs through the bronchi in most of the methods of origin of pulmonary gangrene that have been mentioned, they may also be transported into the lungs by the blood-current. We call these forms embolic gangrene. We find such gangrenous nodules in the lungs in connection with extensive gangrenous bed-sores, puerperal processes, suppurative caries of the bones, etc. In these cases the putrid material enters a vein from the seat of the primary process and is brought to the lungs, and here we find, as a result of the putrid character of the embolus, not a simple infarction, but an embolic gangrene.

Pathological Anatomy.—We more frequently find pulmonary gangrene in the lower lobes than in the upper, corresponding to its mode of origin. Either both lungs are affected or only one, and the right somewhat more frequently than the left. We distinguish a diffuse and a circumscribed form, according to the extent of the gangrene. Embolic gangrene belongs to the latter form, and its nodules, by preference, lie near the pleural surface.

We can easily recognize the anatomical changes in gangrene. The lung-tissue is changed to a discolored, dirty, greenish-gray mass, which gradually and progressively becomes dissolved, forming a most foul-smelling ichor. We find, left in it, necrotic fragments of tissue and vessels. Gangrene cavities, with irregular, ragged walls, are formed from the partial expectoration of the softened gangrenous nodule. The lung-tissue in the vicinity of the peculiar nodule is to a greater or less extent inflamed, partly in the form of catarrhal pneumonia, partly in the form of circumscribed croupous pneumonia. The inflamed parts in the vicinity are gradually involved in the gangrene, so long as the process extends, but finally a suppurating line of demarkation may be formed about the gangrene, the whole gangrenous fragment is in a measure sequestrated, encapsuled, and gradually expelled, and so healing becomes possible. We have already stated that fœtid bronchitis may arise from a gangrenous nodule.

Whenever a gangrenous nodule reaches the pleura, a purulent and usually a sanious pleurisy follows from direct infection. Pneumothorax may arise from perforation of a gangrenous cavity.

Clinical History.—The symptoms of gangrene depend for the most part upon the local affection in the lung. The condition of the expectoration is characteristic, and it alone may decide the diagnosis.

In many of its conditions the expectoration greatly resembles that of fœtid bronchitis, and indeed a great part of it does not come directly from the gangrenous nodule, but is the secretion of the diseased bronchi. The penetrating stench of the sputum, a most repulsive, putrid odor, is very striking. The patient's breath and cough also have this bad smell, which infects the whole vicinity. The amount of the sputum is usually large; it may reach ten or twenty ounces (200–500 c. c.) in twenty-four hours. If the sputum is collected in a glass it forms three layers, as in the sputum of fœtid bronchitis—an upper layer, muco-purulent, greasy, consisting in part of nummular sputa, and covered with much froth; a middle serous layer, in which some firm masses from the upper layer float; and a lower layer, almost wholly of pus, but greasy and greenish-yellow, which usually contains many large and small plugs and shreds of tissue. We find in these plugs, on microscopic examination, beautifully twisted needles of the fat acids (see Fig. 21, p. 156) imbedded in countless bacteria, fat-drops, and detritus, and often collected in large bundles; but besides these we find in it the constituents of the parenchyma of the lungs, and this alone is the decisive factor in distinguishing between pulmonary gangrene and simple fœtid bronchitis. Traube's theory—that elastic fibers are not found in the expectoration in pulmonary gangrene, or that they are rare, because the elastic tissue also is destroyed by the gangrene—is untrue. We have almost always found elastic tissue in abundance in the expectoration, and also other fragments of parenchyma, lung-pigment, etc. Which of the coarse, rod-like bacteria, described by Leyden and Jaffé as *leptothrix pulmonalis*, are the special bacteria of gangrene is not decided. The chemical examination of the sputum shows the presence of those substances which may always be found in the putrefaction of organic matter—tyrosine, leucine, ammonia, sulphuretted hydrogen, butyric acid, valerianic acid, caprylic acid, etc. The fresh sputum usually has an alkaline reaction, but on standing it becomes acid.

Many cases of gangrene lead to erosion of the vessels and severe hæmoptysis. Slight admixtures of blood in the sputum are not infrequent.

The other symptoms on the part of the lungs are not especially characteristic of gangrene. Most patients complain of cough, pain in the side, and more or less severe dyspnœa. Physical examination, as a rule, permits us to make out the seat of the nodule, but not always, since the physical signs, of course, depend upon the situation and extent of the gangrene. Small nodules, situated centrally, often give no objective evidence of their presence. Every extensive infiltration, however, must cause dullness on percussion. Over the area of dullness we hear bronchial respiration, and usually quite numerous moist râles. If a gangrenous cavity is formed, the physical examination may show plain symptoms of a cavity—tympanitic resonance on percussion, amphoric respiration, large moist râles, etc.

The physical signs are sometimes due to the accompanying pleurisy; the dullness is more intense, the respiratory murmur and the vocal fremitus are diminished, and the adjacent organs are displaced by the abundant effusion; but an absolute diagnosis of an accompanying pleurisy is often to be made only by an exploratory puncture. We have already spoken of the occasional development of pneumothorax.

In many cases there is fever, of quite an irregular character and of very varying intensity. In the cases where the gangrenous nodule is sequestrated, and where the secretion can be freely emptied through the bronchi, and where, accordingly, there is no absorption of septic material into the blood, the fever also may be entirely absent.

We often see gastric and intestinal symptoms in pulmonary gangrene, the disturbance being without doubt due to swallowing some of the fœtid sputum. Many patients complain of loss of appetite, and sometimes of vomiting or diarrhœa. We also see rheumatic pains in the joints and muscles, as in fœtid bronchitis. It is also worthy of note that secondary abscess of the brain has been repeatedly observed in pulmonary gangrene (see page 741). We must bear this in mind if very severe cerebral symptoms, like somnolence or paralysis, develop in the course of a gangrene.

The general course of the disease shows very great variations. In all cases where the pulmonary gangrene is secondary to some other affection, the course and the general type of the disease depend very largely upon the primary attack, but the cases of idiopathic gangrene also present great variations. The onset is either quite gradual and slow, or quite acute, and associated with fever and thoracic symptoms. The stinking expectoration and the bad odor from the patient's mouth first direct the attention to the existence of putrid processes in the lungs. The disease is usually very chronic, lasting for months or even years. Many remissions and intermissions occur. With proper care and treatment we may see a clear improvement, and often apparently a complete cessation of the disease. The bad odor ceases, the expectoration diminishes or disappears entirely, and the patient's strength and nutrition become almost normal; but relapses may occur after long intervals. Where the affection is of slight extent we may even see a complete recovery.

Pulmonary gangrene always takes a worse course in previously weak and marantic persons, and an unfavorable termination may follow in a comparatively short time. Death ensues either from a general loss of strength, as a result of the disease, or from complications, such as pulmonary hæmorrhage, ichorous pleurisy, pneumothorax, or abscess of the brain. Rupture of an ichorous empyema outwardly, or into the peritoneum, or into other cavities, is rare.

Special mention must be made of the fact that the symptoms of pulmonary gangrene are not always so very pronounced. In people who are weak and run down we often see pulmonary gangrene at the autopsy, although during life there have been no marked symptoms, not even offensive sputum nor the fœtor from the mouth

Diagnosis.—The diagnosis can be made with certainty only when the characteristic sputum is present. We can decide whether the sputum comes from a fœtid bronchitis or from the fœtid contents of a bronchiectasis, or from actual gangrene, only by finding under the microscope the remains of lung-tissue in the expectoration. Physical examination in gangrene, at least in part of the cases, also gives the signs of infiltration or of cavity-formation in the lungs.

Prognosis.—The prognosis depends first upon the nature of the underlying disease, and then upon the extent of the affection, the strength of the patient, and the possibility of sufficient care and proper treatment. If the process in the lung becomes sequestrated, marked improvement may follow, even in the severest conditions; but we must always remember that a relapse is possible. We have already mentioned the dangers which may cause a fatal termination in pulmonary gangrene.

Treatment.—Prophylaxis plays an important part in those cases where there is danger of the entrance of bits of food into the air-passages from defective deglutition. We must think of the possibility of this with all patients who show great stupor, and also with patients who have paralysis of deglutition, in order to watch them while taking food, and eventually to try artificial feeding with the œsophageal tube.

The treatment of already existing pulmonary gangrene has, as its chief aim, to check the putrid processes of decomposition in the lungs. Unfortunately, the remedies at our command are not in all cases sufficient for this. The different disinfecting inhalations are the most effective. They are used in the same way as in fœtid bronchitis (*vide supra*). Turpentine deserves the most confidence, and it may also be given internally with good results. According to Lépine, however, terpine acts still better than does turpentine. We may also call attention to inhalations with carbolic acid, Curschmann's carbolic mask, inhalations with salicylic and boracic acids (4 parts of salicylic and 20 of boracic acid to 1,200 of distilled water), bromine (bromine and bromide of potassium, 2 parts of each to 1,000 of water), and similar substances.

Besides oil of turpentine, other internal remedies are recommended: half a grain to a grain (grm. 0·03–0·06) of acetate of lead every two hours, creasote, carbolic acid, etc. Their action is uncertain. Of late, myrtol has been greatly extolled. It is given in capsules containing grains ijss. (0·15), of which two or three are to be taken every two hours.

The general treatment of the patient is very important—he should have good food, and live in as good air as possible. We must treat the pain in the chest and the cough symptomatically, local applications and morphine being most useful. The fever seldom gives occasion for direct interference. In general, cold sponging, or else tepid baths, suffice, so that quinine is only rarely to be used. We may try to relieve the gastric and intestinal symptoms by giving antiseptics internally, especially by small doses of muriatic acid, salicylic acid, or creasote, as well as by the ordinary remedies, such as bitters and opium.

If a secondary ichorous pleurisy develops, with or without pneumothorax, removal of the fluid by operation is necessary, if the patient has sufficient strength to bear it.

CHAPTER IX.

DISEASES FROM THE INHALATION OF DUST.

(Pneumonoconiosis.)

ALTHOUGH there are a number of important contrivances in the respiratory apparatus to impede the entrance of foreign substances contained in the air into the lungs, still, if a person remains in a dusty atmosphere, so many particles of dust may be inhaled that they are not without effect on the lung-tissue. The diseases from the inhalation of dust are usually purely professional diseases, which occur especially in workmen whose occupation brings with it the continual inhalation of some kind of dust. Since we do not have to deal with a specific influence, as in the inhalation of infectious substances, but usually only with a mechanical effect, all diseased conditions excited by the different sorts of dust may be treated in common.

We must first mention, however, a condition of the lungs which can scarcely be regarded as pathological, although it has its origin in the constant inhalation of dust, especially of coal-dust—the ordinary black pigmentation of the lungs. There can now no longer be any doubt, although there was once a long dispute about it, that the black pigment in the lung comes, in large part at least, from the inhalation of carbon. The particles of carbon pass into the lungs themselves, and thence into the bronchial glands by means of the lymphatics. Only a part of the coal-dust inhaled is removed with the expectoration, and it may easily be found in it microscopically, and often by the naked eye, as we see it in the well-known black expectoration which we often have in the morning, if we have spent the previous evening in a room filled with smoke. In Germany, Traube was the first to discover the particles of carbon in the expectoration of a charcoal-burner. In the man's lungs, after death, the vegetable formation of these particles could be recognized, and Traube gave the correct explanation of them. In workmen who inhale large amounts of charcoal-dust, anthracite coal-dust, soot, or graphite, the "normal" pigmentation of the lung passes into a pathological condition, *anthracosis pulmonum.*

Zenker first discussed comprehensively the fact of the entrance of the different sorts of dust into the lungs and the consequent results. Besides the anthracosis already mentioned, the pulmonary disease from inhaling the dust of flint and other stones is of especial importance, the so-called stone-cutter's lung—*chalicosis pulmonum*—and also that from inhaling metallic dust, especially oxide of iron—*siderosis pulmonum.* The "stone-lungs" have been observed in workmen in the stamping-rooms of glass-factories, in mill-stone cutters, stone-polishers, stone-hammerers, plasterers, workers in porcelain, masons, slate-quarrymen, potters, etc. "Metal-dust lungs" occur in file-cutters, iron-workers, mirror-polishers, and especially in grinders, who inhale a mixture of stone- and iron-dust. The first case of a "red iron lung" was observed by Zenker, in a girl who had inhaled a thick dust of iron for ten or twelve hours a day. Her work was to color blotting-paper with a powder of red oxide of iron. Among the other forms of dust which may give rise to pulmonary disease we may mention tobacco-dust, cotton-dust, saw-dust, and flour-dust.

The anatomical changes in the "dust-lungs" consist of a chronic bronchitis, and especially a chronic interstitial inflammation, leading to the formation of connective tissue, and caused by the mechanical irritation of the foreign body. The lungs are studded with nodules, which feel hard to the touch, and which grate on section with a knife. All of these nodules consist of firm connective tissue, in which the particles of stone or iron are encapsuled. By the union of single nodules we may get more extensive induration and cicatricial formation. Chemical examination of such lungs gives, as might be supposed, a large amount of silicic acid, iron, etc.

In most of the cases which come to autopsy we find further changes in the lungs, which are not the immediate result of the inhalation of dust, but consist of sequelæ and complications. Chronic diffuse bronchitis in the worker in dust, like any other chronic bronchitis, may give rise to pulmonary emphysema, and later to cardiac hypertrophy, etc. We very often find in the lungs co-existing and pronounced tubercular changes. It need not be stated more fully that these changes are not the direct result of the inhalation of dust, but that the changes in the lungs set up by such an inhalation furnish merely a favorable soil for infection with tuberculosis. In most cases, the "dust-lungs" acquire a marked clinical significance chiefly from the sequelæ mentioned—namely, emphysema and tuberculosis. The circumscribed nodules of interstitial pneumonia have no very marked symptoms following them. In all cases where there is a fatal termination, with pulmonary symptoms, we should regard the immediate action of the dust as much less the cause of death than are the secondary diseases.

The actual points to be considered in judging of the clinical symptoms of the diseases from inhaling dust are contained in what has been said. The symptoms are those of common chronic bronchitis, or pulmonary emphysema, or phthisis, and attention to the injurious influences associated with the patient's calling is the only possible way of making a diagnosis, but in individual cases it may always be a matter of doubt how far other accidental causes of disease may come into play.

The prognosis depends, in the first place, upon whether the patient can be removed from the action of these injurious influences or not, but we must also mention the fact, which has been often observed, that many people get somewhat used to the dust. After they have once recovered from the initial bronchitis, such people can live in an atmosphere of dust for a long time without any noticeable injury.

The prophylaxis of diseases from inhaling dust forms an extended chapter in the hygiene of occupations, which we can not dwell upon here. The workman must be taught the danger to which he is exposed, and the danger itself must be diminished as much as possible by sufficient ventilation of the work-rooms, by cleanliness, and, under some circumstances, by a change in the technicalities of the business.

We need not give any special data regarding the treatment of diseases from inhaling dust. They are founded on the same principles as are given for the treatment of chronic bronchitis, emphysema, and chronic pulmonary tuberculosis.

CHAPTER X.

EMBOLIC PROCESSES IN THE LUNGS.

(*Hæmorrhagic Infarction of the Lungs.*)

Ætiology.—The sources from which the material for an embolic plugging of branches of the pulmonary artery comes lie either in the right side of the heart or in the veins of the body. Pathological anatomy teaches us how often thrombi are formed in the veins, especially in the veins of the lower extremities and in the pelvic veins, and in the right side of the heart, in the recesses between the trabeculæ, in the auricles, on the valves and the chordæ tendineæ, and at the apex of the ventricle. The particles, torn loose from thrombi so situated, are carried on by the blood-current, reach the lungs, plug a larger or a smaller branch of the pulmonary artery, according to the size of the particles, and thus cause further changes in the lung-tissue. Since the branches of the pulmonary artery are "terminal arteries," and since thus the vascular territory belonging to each branch can not be supplied, or can be supplied only to a small amount, with blood by collateral circulation from other vessels, the closure of a branch of the artery shuts the territory supplied by it out of the circulation. The pressure, in the part of the vessel lying peripherally to the point of plugging, becomes almost nil, and, as a result, there is a backward current into the region shut off, flowing from the capillaries in the vicinity, and even from the veins belonging to it. Thus we get a typical "engorgement." The walls of the capillaries and veins, in which the normal blood-current has ceased, lose their natural consistency as a result of this. The vascular walls become abnormally pervious. The fluid of the blood, the white blood-corpuscles, and also very many red blood-corpuscles, penetrate through the walls of the vessels into the surrounding tissue, and change it into the so-called hæmorrhagic infarction.

Every embolic closure of a branch of the pulmonary artery does not necessarily result in the formation of an infarction. Upon the sudden plugging of a main trunk, or of several large branches of the pulmonary artery, death may ensue at once, so that naturally all further changes in the lung-tissue cease. We also find quite frequently, especially in central portions of the lung, embolism of single branches of the pulmonary artery, without the formation of an infarction. In such cases there must necessarily be a little circulation in the vascular territory which has been shut off, either by anastomoses between the territory of the pulmonary artery and that of the bronchial or mediastinal artery, or by the neighboring capillaries, whose arteries of supply remain open.

The changes thus far described are the result of a purely mechanical closure of a pulmonary artery. We have noticed this especially where simple fibrinous plugs have given the occasion for the embolic process. Pulmonary infarctions are most frequent in chronic heart disease, in all forms of primary and secondary dilatation of the heart, but especially in disease at the mitral orifice, and in mitral stenosis. Thrombus formation is frequent in the dilated right side of the heart, and furnishes material for pulmonary emboli; but these emboli are seen in all other possible conditions of disease, in which thrombosis in the right side of the heart or in the veins may occur.

The changes in the lungs assume quite a different appearance if the embolic material is not simple fibrine, but if it contains at the same time some specific infectious matter. If emboli reach the lungs from an acute malignant endocarditis in the right side of the heart, or, as is most frequently the case, from a purulent (septic) phlebitis anywhere in the body, giving rise to a puriform, liquefying thrombus, the specific factors in inflammation—that is, bacteria—get into the lungs. Thus arise embolic abscesses and embolic gangrenous nodules in the lungs. We have already spoken of the latter, and the former are among the most constant lesions in every typical pyæmia.

The fundamental facts as to the occurrence and significance of embolic processes in general, and those located in the lungs in particular, were discovered by Virchow. For the fuller understanding of the results of embolic closure of the vessels we must thank chiefly the labors of Cohnheim.

Pathological Anatomy.—Hæmorrhage infarctions may involve one or more lobules, or almost a whole lobe of the lung, according to the situation of the embolus. Most infarctions are situated at the periphery of the lung, and have approximately a conical shape, corresponding to the extent of the region of the vessel. The base of the cone lies against the surface of the pleura. It generally projects a little above that surface, and its dark color can usually be plainly recognized through it. The pleura itself is the seat of a fibrinous inflammation at the point to which the infarction reaches, and sometimes for a large space around it. The conical shape of the infarction is plainly recognized on section. The lung-tissue is changed to a firm, fragile, uniformly black-red tissue, devoid of air. The embolus can usually be readily found in the branch of the pulmonary artery leading to the infarction. Under the microscope we see a diffuse infiltration of tissue, with red blood-corpuscles in the infarcted portion. The alveoli and finer bronchi are also filled with coagulated blood. Under favorable circumstances, in cases of longer standing, the blood may be reabsorbed in part. The lung again contains air, but it remains much pigmented in that place, and more or less indurated from the development of interstitial connective tissue.

Hæmorrhagic infarctions are usually situated in the lower lobes, and more frequently on the right side than on the left.

The smaller embolic abscesses are sometimes very numerous, and are disseminated over the whole lung. In larger abscesses the conical shape may often be plainly recognized. Where an abscess extends to the pleura, a purulent pleurisy arises from direct infection. Combinations and transitional forms between the ordinary hæmorrhagic infarction and embolic abscesses are occasionally found in the lungs.

Symptoms.—We often find at the autopsy embolism of single branches of the pulmonary artery, with or without infarction, which has caused no symptoms at all during life.

Embolism of the main trunk, or of a large branch of the pulmonary artery, may cause sudden death, as has been repeatedly observed in patients with heart disease, or with venous thrombosis. If death be not immediate, sudden severe dyspnœa and oppression arise. The diagnosis may at least be suspected in such a case if we know of a possible source for an embolus. In some instances, where an embolus is situated in a large branch of the pulmonary artery, but has not completely filled it, we can hear a systolic vascular murmur over the affected spot, as has been observed by Litten. The diagnosis, however, becomes certain if the further signs of infarction appear later.

The most characteristic symptom of infarction in the lungs is the bloody expectoration. If we see quite suddenly bloody sputum in a patient with mitral stenosis, we are usually right in assuming a hæmorrhagic infarction of the lung. Either the sputum consists almost entirely of dark blood, or the blood is mixed with more or less mucus; but there is never much air in it. The bloody expectoration often lasts for several days.

We try to learn more of the size and situation of the lesion by a physical examination of the lungs. Of course this often gives a negative, or at least a doubtful, result. It goes without saying that small infarctions, and also those which are central, can not be made out by physical examination. Large peripheral infarctions may give rise in many cases to dullness on percussion, crepitant râles, and harsh or bronchial respiration, but it is often hard to decide in an individual case whether the physical signs which we meet with are not due to other pathological changes in the lungs, like bronchitis or hydrothorax. We sometimes hear a pleuritic friction-sound in some part of the chest a few days after we suspect that an infarction has occurred, by which the diagnosis gains additional certainty. We have already mentioned the subjective symptoms in embolism of a large pulmonary vessel—sudden dyspnœa and oppression. Small nodules often cause no special symptoms, but in other cases the patient feels a severe pleuritic pain, due to irritation of the pleura.

Fever may be wholly absent, though we sometimes see a moderate rise of temperature at the onset.

The embolic abscesses in the lungs hardly ever give rise directly to clinical symptoms. They form a part of the general picture of pyæmia and similar general infectious processes. Marked symptoms on the part of the respiratory apparatus are seen only when the abscesses are present in very large number. If an empyema develops from a focus which extends to the pleura, it sometimes gives rise to definite physical signs.

It follows from all that has been said before, that in the diagnosis of embolic processes the chief stress must always be laid on the presence of an ætiological factor. We must regard the bloody sputum as the main direct symptom in hæmorrhagic infarction. Embolic abscesses in the lungs may often be suspected in pyæmic diseases, but they can hardly ever be made out absolutely.

The prognosis is entirely dependent upon the underlying disease. In heart disease the occurrence of a hæmorrhagic infarction is usually on the whole an unfavorable sign, since it points to weakness of the right ventricle, and hence to the formation of a thrombus in it; yet it often happens that the symptoms of a pulmonary infarction may pass away entirely.

We need not give special directions for treatment. It is in part purely symptomatic, and in part coincides with the treatment of the underlying affection. As regards prophylaxis, we must bear in mind the absolute necessity of as perfect rest as possible in those patients in whom the presence of venous thrombi, as in the femoral veins, suggests the possibility of pulmonary embolism.

CHAPTER XI.

BROWN INDURATION OF THE LUNGS.

(*Lungs of Heart Disease.*)

In heart disease, especially in mitral stenosis, we often find a peculiar change in the lungs, whose origin must be sought in the long-persisting engorgement of the pulmonary circulation. The lungs are hard and dense, and show on a fresh section an abnormal brownish-yellow color. In the larger pulmonary vessels, the arteries and veins, there is a thickening and cloudiness of the intima as a result of the stasis. We see here and there on the surface of the section, and beneath the pleura, little dark spots of pigment and fresh hæmorrhages. We term this condition brown induration of the lungs.

Microscopic examination shows that the capillaries are evidently dilated and twisted as a result of the persistent stasis. They even extend a good way into the alveoli, whose lumen is thus actually diminished.* The interstitial connective tissue seems somewhat thickened, and we find in it many pigment granules, the remains of the extravasated and decomposed red blood-corpuscles. According to Rindfleisch, the muscular constituents of the parenchyma of the lung, the smooth muscular fibers at the entrance and in the walls of the alveoli, show a distinct hypertrophy. In the intima of the larger vessels we often find fatty degeneration of the endothelium, and sometimes even fatty degeneration of the muscular coat.

As regards the clinical significance of brown induration of the lungs, it is possible that the extensive diminution of the lumina of the alveoli throughout the whole lung may contribute somewhat to the increase of the dyspnœa in heart disease, but in practice this factor can not be separated from the other causes which produce dyspnœa.

We have no certain factors by which to diagnosticate brown induration of the lungs during life. The anatomical lesions, too, show a certain variation, not always to be explained, in that, under apparently the same conditions, the brown induration is often very marked, and often only extremely slight. In cases where we find this induration in the cadaver we sometimes hear, during the patient's life, a very sharp, puerile respiratory murmur, which seems to be characteristic of many cases of the "heart-disease lung." We might lay still more stress upon the presence of large, characteristic cells in the expectoration, which are thickly filled with large and small yellow or brown pigment-granules. These large pigmented cells are in all probability white blood-corpuscles, which have taken up the pigment from the red blood-corpuscles destroyed within the alveoli. At the autopsy we also find these cells lying within the alveoli. Besides these pigment-cells we also frequently see the still intact red blood-corpuscles in the expectoration of patients with heart disease.

The prognosis and treatment coincide with those of the underlying cardiac disease.

CHAPTER XII.

TUMORS OF THE LUNGS. CANCER OF THE LUNGS. ECHINOCOCCUS OF THE LUNGS. PULMONARY SYPHILIS.

1. New Growths in the Lungs. Cancer of the Lungs.—Most of the new growths which are met with in the lungs are of a secondary nature. Secondary cancers are sometimes found in the lungs, with carcinoma of other organs, whose origin may always be explained by supposing a growth of the primary tumor into a vein, and the consequent carriage of the germs of the growth to the lungs. These secondary nodules in the lungs usually cause no special clinical symptoms, unless they are very numerous and extensive, when they give rise to dyspnœa, and physical signs. Several years ago there came to the clinic in Leipsic a case of secondary, and very extensive, miliary carcinosis of the lungs, which ran a brief and fatal course, simulating acute miliary tuberculosis with predominant pulmonary symptoms.

We must mention enchondroma among the other secondary new growths, but it may also occur primarily in the lungs in very rare cases. Secondary sarcoma of the lungs is also very rare. We have seen a very extensive form of it after primary sarcoma of the bronchial glands, and also in a case of lympho-sarcoma of the cervical lymph-glands, where the sarcoma had grown into the jugular vein; and, finally, we have seen it repeatedly in congenital primary sarcoma of the kidneys (*vide* chapter on new growths in the kidneys).

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* This view had been universally accepted, but it has been somewhat shaken by Basch. He believes that the effect of passive congestion upon the pulmonary capillaries is to stretch them, thus tending to make the lumen of the alveoli not smaller, but larger (*vide* chapter on heart disease).

Among the primary new growths in the lung, cancer is the only one which has much clinical significance. In its clinical relations we can also rank with it certain malignant, metastatic forms of alveolar sarcoma. The typical cancer of the lungs is always a cylindrical-celled carcinoma, which undoubtedly arises from the bronchial epithelium. It is especially common in elderly people, over forty, and seems to be found somewhat more frequently in the right lung than in the left, and in the upper lobes than in the lower. By its diffusion the lung-tissue in the parts affected by cancer is changed to a yellowish-gray and quite soft and crumbling mass, devoid of air. We can usually scrape away from the section the characteristic cancer-juice, in which the microscope shows the typical cancerous elements. The pleura is very often involved. The new growth has either extended directly into it, or single, and more circumscribed, secondary nodules have formed in it. The lymph-glands are almost invariably affected, especially the bronchial glands, and also the axillary and cervical glands. Secondary carcinoma of other organs is rare, but it is found in some cases in the other lung, the liver, the brain, and elsewhere.

It is almost always difficult to interpret correctly the clinical symptoms of cancer of the lungs at the beginning of the disease. They are referred to some other, more frequent chronic pulmonary disease, such as chronic bronchitis, phthisis, or pleurisy, but in the further course of the disease we succeed, at least in a number of cases, in making a correct diagnosis. In other cases, especially in old people, the disease may remain latent.

The general symptoms in the lungs have little that is characteristic. The patient complains of gradually increasing difficulty in respiration, and of pressure and distress in the chest, which may finally increase to the most intense dyspnœa. Most patients suffer very much from the labored, frequent, and spasmodic cough. The expectoration in some cases has no peculiarity, but it often assumes, at least for a time, a characteristic consistency which is extremely important for diagnosis. It contains blood, and also has a peculiar "currant-jelly-like" appearance. Under the microscope we can sometimes make out the characteristic elements of the tumor in it. Severe hæmoptyses are also seen in cancer of the lungs.

Physical examination of the lungs gives a positive result in many cases, such as dullness, bronchial respiration, diminished respiration, râles, and sometimes pleuritic friction-sounds, none of which has anything characteristic in itself, but they are of distinct significance in making out the seat and the extent of the new growth. Special mention must be made of the often-noticed diffuse projection and swelling of the diseased side.

The occurrence of certain sequelæ is of great diagnostic significance. The chief one is the discovery of swelling in the lymph-glands in the neck or axilla, and also a number of symptoms of compression, which either are produced directly by the new growth, or are due to the secondary enlargement of the lymph-glands. Pressure on the superior vena cava, or a large branch of it, causes œdema in the face, neck, over the wall of the chest, or in one arm. The subcutaneous veins in the regions named appear dilated and tortuous. Pressure on the œsophagus causes difficulty in deglutition; pressure on the brachial plexus, intense neuralgic pains and paresis of one arm; pressure on the recurrent nerve, paralysis of the vocal cords and hoarseness; pressure on the trachea or a primary bronchus, the symptoms of tracheal or bronchial stenosis.

Besides the symptoms already mentioned we must consider the general symptoms. As in carcinoma in general, so in pulmonary carcinoma, the well-known cancerous cachexia gradually develops. The patient grows dull, loses his appetite more and more, disturbances of digestion and sometimes moderate elevations of temperature develop, until he finally succumbs to general marasmus.

The whole duration of the disease is from six months to two years. The prognosis is fatal. The treatment can be only purely symptomatic, and we employ the same remedies as in other pulmonary affections.

We must still briefly consider a new growth in the lungs which is extremely interesting from a theoretical point of view. In workmen in the cobalt mines of Schneeberg, in the Saxon Voigtland, the development of malignant lymphosarcomata in the lungs, with the occasional formation of metastases in the glands, the liver, the spleen, etc., is of frequent occurrence. The disease runs its course under the type of a chronic pulmonary affection, and almost always ends fatally. The endemic occurrence seems to point to an infectious origin for the tumor.

2. ECHINOCOCCUS OF THE LUNGS.—Primary echinococcus in the lungs is very rare. In most cases the echinococci are brought to the lungs secondarily from other organs, either by way of the blood-current, or, as is far oftener the case, by perforation of an echinococcus of the liver through the diaphragm.

The symptoms of echinococcus of the lungs are manifold. The parasite sometimes remains entirely concealed. In other cases a more or less severe, and often febrile, affection of the lungs, is developed, with pain in the chest, cough, and sometimes bloody expectoration, and dyspnœa. Physical examination of the lungs gives in some cases dullness, absence of respiratory murmur, and diminished vocal fremitus, while after the expectoration of the echinococcus (*vide infra*) symptoms of a cavity may ensue. A correct interpretation of all these symptoms is possible only when, as has often been observed, the echinococcus cysts are coughed up, or at least when parts of them, like the membranes or the hooklets, are found in the expectoration.

The termination of the disease may be favorable if the echinococci are expectorated, or if we succeed in removing them by operative means. We can hardly hope to be able to kill the parasite by inhalations of turpentine or benzine. Sometimes the echinococcus cyst becomes gangrenous, or suppurates. Rupture into the pleura, into the peritoneum, into the pericardium, and externally, has also been observed. This last termination is the most favorable, since otherwise, if the affection progresses, a fatal result may be caused by the sequelæ, or rarely by the occurrence of suffocation. The details of the natural history of the echinococcus are given in the chapter on echinococcus of the liver.

3. PULMONARY SYPHILIS.—This would also be the place to speak of syphilitic new growths in the lungs, but, in our opinion, in spite of the quite abundant literature of this subject in recent times, no definite statement as to pulmonary syphilis can be made. Those physicians who are disposed to consider every pulmonary disease in a previously syphilitic subject to be of a syphilitic nature, certainly regard many things as pulmonary syphilis which have nothing at all to do with syphilis. At least, we have found that all those cases which at first suggested a diagnosis of pulmonary syphilis, finally, upon more accurate examination and after longer observation, have turned out to be something else, usually tuberculosis. The only form of pulmonary syphilis that has been established pathologically is syphilis of the larger and medium-sized bronchi, which is recognized at the autopsy by extensive radiated cicatrices in the bronchial mucous membrane, and which sometimes lead to stenosis. Single gummatous nodules in the lungs are of the greatest rarity. We sometimes find in the pleura peculiar radiated cicatrices, which perhaps are of syphilitic origin. Practically we are always justified, if severe and otherwise inexplicable pulmonary symptoms occur in a previ-

ously syphilitic subject, in trying specific treatment; but very seldom do we get any good result from it. The pulmonary syphilis of the new-born, which occurs in the form of single nodules or as a diffuse syphilitic infiltration, the so-called *pneumonia alba*, has only a pathological interest.

SECTION V.

DISEASES OF THE PLEURA.

CHAPTER I.

PLEURISY.

(Pleuritis.)

Ætiology.—We make the general distinction of a primary and a secondary pleurisy.

By primary pleurisy we mean those cases where previously healthy people are attacked with an inflammation of the pleura. We have no doubt that such cases occur, but they are much rarer than is generally believed, for many cases of secondary pleurisy give one the impression of a primary disease, either because the primary affection has caused no symptoms up to that time, or because it can not be detected. Taking cold is one of the chief exciting causes of primary pleurisy, and mechanical injury may also sometimes act as a cause.

Secondary pleurisy arises, in a great majority of cases, from a direct invasion of the pleura by an inflammatory process originating in some neighboring organ. We have already had to point out repeatedly, in the description of pulmonary diseases, how the different pathological changes in the lung likewise involve the pleura if they reach that organ. Thus arises the pleurisy in croupous pneumonia, in lobular catarrhal pneumonia, in pulmonary gangrene, in hæmorrhagic infarction, and in embolic abscess. Since many of these affections often develop in the course of the most diverse diseases, we can easily understand that pleurisy is a not infrequent complication of all possible severe diseases.

By far the most important secondary form is tubercular pleurisy. The teaching of daily clinical and pathological experience is that the ordinary chronic pulmonary tuberculosis is almost constantly complicated with pleurisy. The latter is often entirely subordinate to the phthisis, but in many cases certain subjective symptoms, like pain, and also physical signs, are certainly to be referred to the pleurisy. Of much greater practical importance, however, are those cases of tubercular pleurisy which arise as an apparently primary pleurisy. Among these are the larger part of all ordinary "pleuritic effusions." The type of the disease is wholly dominated by the pleurisy. This may improve decidedly, as often happens, or even in many cases be completely cured (*vide infra*), but, if we keep the patient long enough under observation, marked signs of tuberculosis usually arise later on (see the general course of the disease), whence it follows that the initial pleurisy must be regarded, in an ætiological sense, as tubercular. The special origin of tuberculosis in these cases is not always quite clear. There is certainly a small tubercular focus often present in the lungs, which has caused no symptoms in itself, but which became the point of origin for a pleurisy. Not infrequently the pleurisy seems to start from a tuberculous bronchial gland breaking into the pleural cavity and infecting the latter with tuberculous virus.

Other organs besides the lungs may be the point of origin for a pleurisy. Thus pleurisy arises from affections of the ribs or vertebræ, such as caries, from perforation of an œsophageal cancer, etc. Inflammations of other serous membranes are especially apt to invade the pleura. Thus it arises as a result of a pericarditis or peritonitis. Since the pleural and peritoneal cavities are in direct communication with each other by the lymphatics of the diaphragm, we can conceive that both a purulent and a tubercular peritonitis may result in a secondary pleurisy.

Secondary pleurisy sometimes arises in the course of certain diseases in another way. In acute rheumatism, in rare cases, a pleurisy develops, which must be considered due to the specific causes of the disease. In chronic nephritis and in genuine gout we sometimes see pleurisy of which the precise origin is as yet not certainly known. Perhaps in these conditions the abnormal accumulation of the products of tissue-metamorphosis in the blood and the tissues is the cause of the development of inflammation.

Pathological Anatomy.—The inflamed pleura is markedly injected, it has lost its normal luster, and instead has a dull surface. This dullness is due to the coagulated fibrinous exudation upon the pleura, the exudation, in mild cases, forming only a thin layer. In more advanced cases, however, the surface of the pleura is covered with thick, rough, and shaggy masses of fibrine. As long as the fluid in the pleura is little or not at all increased, we speak of a simple fibrinous or dry pleurisy (*pleuritis fibrinosa vel sicca*).

In other cases, however, besides the layer of fibrine there is an abundant exudation of fluid from the capillaries of the pleura, forming a pleuritic effusion. This is ordinarily of a simple serous character—serous and sero-fibrinous effusions. The fluid collects between the surfaces of the pleura, or, if there is at the same time an abundant coating of fibrine, between the gaps and in the meshes of the fibrinous exudation. In such cases there are often many flakes of fibrine floating in the fluid.

In every serous effusion we also find a number of pus-corpuscles, which give to it a slight cloudiness, but if the number of pus-corpuscles increases very much, we have a fibrino-purulent or a purely purulent exudation. This is always due to the presence of a specific organized poison which excites the suppuration. The pleurisies which come from embolic abscesses, from gangrenous foci in the lungs, and from carious ribs, and those which arise from the rupture of tubercular cavities into the pleura, are almost always of a suppurative character. We call the purulent pleuritic effusion empyema. If putrefactive agencies enter the pleural cavity at the same time with the pus poison, as in the pleurisies which develop in pulmonary gangrene, the purulent exudation assumes an ichorous, putrid character—ichorous effusion.

Under certain circumstances the effusion assumes a hæmorrhagic character—hæmorrhagic effusion—especially if hæmorrhages arise from the old or newly formed capillaries dilated by the inflammation. They arise partly by diapedesis and partly from rupture of the walls of the vessels. The exact cause of the hæmorrhages is usually unknown. We know by experience that hæmorrhagic effusions are most frequent in tubercular pleurisy, a fact which is of diagnostic importance. Hæmorrhagic pleurisy is also a frequent complication of septic—especially puerperal—diseases, as a result of embolic affections of the lungs. We can often refer the onset of a hæmorrhagic pleurisy, too, to a general hæmorrhagic diathesis, as in scurvy.

The amount of fluid collected in one pleural cavity is, in the majority of cases, somewhere between a pint and a quart (500–1,000 c. c.), but it may reach three or four quarts. Every large effusion must influence the position of the yielding walls of the pleural cavity, the chest-wall, the lungs, the mediastinum, and the diaphragm, through the consequent increase of pressure in the affected pleural cavity; and the resultant symptoms of pressure in the neighboring organs are of the greatest clinical significance. Attention is first called to the lungs themslves. Since the normal lung is expanded in the thorax beyond its elastic equilibrium, it will retract as soon as a part of the pleural cavity is occupied with fluid. Until it has reached its position of elastic equilibrium there can be no question of a positive pressure on the lung. The lung floats on the effusion, in a certain way, if there be no adhesions, but, as the amount of the fluid further increases, compression of the lung follows. With a very large effusion the lung is pushed wholly up and back against the vertebral column, and is changed to an almost bloodless, airless, flat mass. It is, however, possible that the atelectasis of the lung is not caused exclusively by compression from without, but that, after the normal respiratory movements have ceased, a part of the air in the lung may be absorbed by the vessels, or even by the effusion.

We also see the results of the pressure exerted by the pleuritic effusion in the mediastinum and diaphragm, as well as in the lungs. Displacements of the heart arise from the lateral pressure on the mediastinum, which must take place if the pressure in the diseased pleural cavity is equal to that of the atmosphere, for a greater and positive pressure is unnecessary, since a negative pressure prevails on the healthy side.

The downward pressure of the diaphragm, which usually affects both halves of it, although in unequal degree, makes itself manifest on the right by the low position of the liver, and on the left by the downward displacement of the stomach and large intestine. It must be particularly noticed, however, that adhesions may prevent all pressure-displacements which we have mentioned, both of the lungs and of the neighboring organs.

As regards the further changes and terminations of the pleuritic processes, they depend upon the amount and character of the effusion. Favorable cases may result in complete recovery and absorption of the effusion. The fluid contents are taken up directly by the lymphatics of the pleura, and the solid constituents, the fibrine and the white blood-corpuscles, are decomposed, dissolved, and absorbed.

In most of the severe cases, however, an extensive new formation of connective tissue and vessels takes place. The fluid effusion is mostly absorbed, but the pleura itself is thickened and changed—the so-called pleuritic thickening. Very commonly extensive loose or firm adhesions form between the two layers of the pleura—adhesive pleurisy. Spaces may be left between the adhesions, in which the remains of the fluid effusion may be encapsuled—"sacculated pleuritic effusion." In protracted cases, and especially in relapses of inflammation, as a result of phthisis, the pleural membranes may finally reach a thickness of one or two centimetres.

Even in cases with marked thickening final recovery is possible. This always is attended with great cicatricial contraction of the pleura, in which the whole chest-wall is involved. The normal expansion of the lungs and thorax is not restored for months, if at all.

It is largely owing to the nature of the primary disease that extensive pleuritic effusions so seldom fully recover. Hence we often notice that, after temporary improvements, new returns of the pleurisy occur, or serious and usually tubercular diseases of the lungs and other organs.

In old pleural thickenings we sometimes see a deposit of lime-salts, the so-called "pleuritic ossification."

In purulent effusion, too, a final absorption is also possible; but this demands much time, and thick, cheesy masses of pus are often left in the pleural sac. In most cases of empyema, if there is not timely operative interference, the pus seeks an outlet for itself. It may break through the visceral pleura into a bronchus, and be emptied externally, thus giving rise to a pyo-pneumothorax; but in many cases the pleura seems to be destroyed only superficially, and the pus is pressed into the alveoli as into a sponge, especially by the movements of coughing, and thence reaches the bronchi, without letting the air enter the pleural cavity (Traube). In other cases the empyema breaks externally through the chest-wall —*empyema necessitatis.* The point of rupture is usually found in the vicinity of the sternum, where the chest wall is thinnest. In very rare cases the empyema breaks into the deeper parts of the body, or into the abdominal cavity.

Course of the Disease.—We will speak in what follows especially of the course and symptoms of ordinary, apparently primary (*vide supra*), fibrinous or sero-fibrinous pleurisy, the so-called simple pleuritic effusion. What is said of it obtains in large measure in the other forms of pleurisy also. The physical signs, of course, are almost wholly independent of the character of the effusion. As far as the different forms of pleurisy differ clinically, we will mention their peculiarities below.

Only rarely is the onset of pleurisy quite acute and sudden, beginning with a rigor. In such cases we must guard against confusing it with croupous pneumonia. Pleurisy usually begins slowly and gradually. The symptoms, which the patient himself feels, are in many cases to be referred directly to the disease of the pleura. One of the most constant is the pleuritic pain, the stitch in the side. A more or less severe pain comes on in the side at every deep breath, and hence upon any physical exertion; also upon movements of the body, in stooping, coughing, or gaping. Shortness of breath soon appears, and constantly increases. There is often a frequent, dry cough. Besides that, severe general symptoms almost always develop; the patient feels dull, looks pale, and has no appetite. Patients who can endure a good deal often keep at work for a long time, until, after feeling miserable for three or four weeks, they are forced to stay at home and summon a physician. It is very important to know that in not a few cases the general symptoms are much more prominent at the beginning of pleurisy than the local ones. The patient comes to the physician complaining only of weakness, loss of appetite, or headache, and the physical examination is the first thing that shows the presence of perhaps a large pleuritic effusion.

In most of the severe cases the further course is slow like the beginning, but sometimes the severest symptoms, most intense dyspnœa, marked cyanosis, etc., may come on in a short time, owing to a sudden increase of the effusion. On the other hand, in mild cases the symptoms may disappear again in a few weeks, but the objective signs in such cases are generally to be made out for a longer time. The disease ordinarily lasts for at least five or six weeks, and often much longer. Gradual recovery follows, or the onset of some new disease, usually tubercular (*vide infra*).

Single Symptoms.—The pleuritic pain, the stitch in the side, is one of the most frequent subjective symptoms. We have previously mentioned that in primary diseases of the lungs, too, as in croupous pneumonia, the stitch in the side is due to the accompanying pleurisy. It is remarkable that the intensity of the pain in no way corresponds to the apparent intensity of the disease. There is often the severest pain in the side when the physical examination shows almost nothing. On the other hand, we often hear a decided pleuritic rub without the patient's complaining of any special pain. Pressure on the chest-wall on the affected side is often very painful. With severe pain we may consider the possibility of an invasion of the intercostal nerves by the inflammation. We have never observed cases, like those described by some authors, of "crossed pleuritic pain"—that is, cases where the pain is localized on the side not affected.

Cough and Expectoration.—The cough is probably directly excited by the disease of the pleura. We often find the pain in the side, and also the cough, brought on by a deep inspiration. Expectoration is entirely absent in uncomplicated pleurisy, or it is scanty, and consists simply of mucus. Much expectoration always means a pulmonary complication. A large amount of purulent sputum is evacuated if a purulent effusion breaks into the lungs (*vide supra*).

Dyspnœa.—The respiration is usually shallow, and consequently frequent, because of the pleuritic pain. In every large effusion which prevents respiration in one lung the dyspnœa becomes more severe, and may, with very extensive effusions, reach the highest degree of orthopnœa. The stronger the patient was before the disease, and the more rapidly the effusion develops, the more severe, as a rule, is the dyspnœa.

Fever.—Most severe pleurisies are associated with fever, but its height is not very great, so that it quite rarely reaches 104° (40° C.). The fever has no typical course. In cases with an acute beginning it is sometimes quite continuous, or slightly remitting at first. If improvement takes place, the fever goes down in about two or three weeks by lysis, so that this part of the temperature curve may be precisely like the period of defervescence in typhoid.

In the more protracted cases the fever gradually becomes more remitting, varying between 100° and 101° (38°–38·5° C.), and it assumes more and more the form of hectic fever. The longer the evening rise of temperature lasts, the more we are justified in suspecting tuberculosis. In empyema we see a higher, irregular fever, sometimes associated with severe chills.

The pulse is constantly rapid, up to 100 and over. In all severe cases the strength and tension of the pulse are much diminished. Irregularity of the pulse is not infrequent. All these changes are probably due in great part to the pressure of the effusion on the heart and large vessels. Lichtheim has discovered experimentally that the compression of the vessels in the compressed lung does not lower the arterial tension.

General Symptoms.—Pleurisy is almost always associated with a pronounced general malaise, muscular weakness, and dullness. The patient is pale, and often markedly cyanotic in cases with much disturbance of respiration. There is great emaciation if the disease is of long duration.

The appetite declines from the outset. There is often occasional vomiting, especially in the first period of the disease. The bowels are usually constipated. Many patients complain of headache.

The condition of the urinary secretion is very important. In every pleuritic effusion the amount of urine is decidedly diminished so long as the effusion increases or remains at the same height. The daily amount is sometimes only eight or ten ounces (200–400 c. c.). The urine is also concentrated, its specific gravity being about 1020–1028. Sediments of urates often form. This diminution of the excretion of water by the kidneys is largely the result of the diminished arterial pressure. An increase of the amount of urine is always a favorable symptom, often the first sign of beginning absorption of the effusion. If a large effusion is rapidly absorbed, the amount of urine may increase to eighty or a hundred ounces (2500–3000 c. c.) daily. The urine, then, of course, is abnormally clear and of low specific gravity.

PHYSICAL SIGNS.

1. **Fibrinous Pleurisy—Pleuritis Sicca.**—Simple fibrinous pleurisy sometimes gives rise to no physical signs at all. If it develops as a result of some pulmonary affection, the physical signs present are often dependent upon this alone.

In many cases, however, dry pleurisy may cause marked objective signs. On inspection, we are struck by the impaired mobility of the affected side on respiration, which is due to the pain caused by breathing. Because of this same tenderness the patient at first often lies on the sound side. Percussion gives no qualitative change of resonance as yet. With the beginning of exudation slight dullness appears, at first almost always in the lower posterior portion of the lungs. Sometimes the resonance becomes tympanitic as a result of retraction of the lung. We can almost constantly make out, especially in the back, that the lower edge of the lung moves less than usual on respiration. Auscultation gives a respiratory murmur that is either qualitatively unchanged or indefinite, but it is always diminished. The peculiar pathognomonic sign of dry pleurisy, however, is the pleuritic friction-rub, that characteristic rubbing, grating, creaking sound, which arises from the sliding of the rough pleural surfaces over each other, and is detected especially in the lateral portions of the thorax. We can hear it both on inspiration and on expiration. It is often jerky, one rub following another after a considerable interval. If we are sure we hear a pleuritic rub, it is direct evidence of the existence of a dry pleurisy, but its absence will not let us exclude pleurisy. The friction-sound must be absent if there are pleuritic adhesions. We can often feel a marked rub by laying the hand on the chest. Sometimes the patient feels it himself, but in other cases he has no sensation of it. We may confound a slight rub with fine crepitant râles. Repeated examinations before and after the patient has coughed usually confirm the diagnosis, since the râles are often changed by coughing.

2. **Pleuritic Effusion.**—Small amounts of fluid in one pleural cavity escape discovery. Physical signs first appear when the amount of effusion reaches eight or ten ounces (200–300 c. c.).

Inspection shows first the more or less marked impairment of motion on the affected side on respiration. If the amount of the effusion is large, there is an evident distention of the affected side in the lower posterior and lateral portions of the thorax. The intercostal spaces are flattened or even a little convex. The nipples and shoulder-blades are farther removed from the median line on the affected side than on the healthy side. The hypochondrium on the affected side is more prominent. In an extraordinarily large effusion on the left side we have seen and felt, in the left hypochondrium, the lower surface of the diaphragm, which was actually arched downward. By direct measurement in severe cases we can make out accurately that the affected side is expanded several centimetres.

With every large effusion there is marked dyspnœa and accelerated respiration. The slight excursions of the affected side on respiration are usually very striking, while the sound side moves so much the more. In this stage of pleurisy the patient often lies upon the affected side, in order to breathe with the healthy lung with as little restraint as possible. With large effusions complete orthopnœa may develop.

The signs due to displacement of the neighboring organs, which are noticeable on inspection, will be mentioned below in the appropriate connection.

Everywhere that a layer of fluid comes between the lung and the chest-wall there is a loss of clearness in the percussion-note. If the thickness of the layer of effusion is five or six centimetres, the resonance seems completely dull or flat. The pleuritic dullness is almost always first made out in the lower posterior portions of the thorax, more rarely in the lower lateral portions. With a slight effusion the height of the dullness is only a few centimetres, but, with much effusion, it rises higher in the back and the lateral portions of the thorax; and on the right, resonance gradually grows dull anteriorly and inferiorly, above the liver. With very large effusions the dullness may begin in front at the second or third rib, or in rare cases even the whole half of the chest, front and back, may give a totally flat percussion-note. Pleuritic dullness is always attended with a marked feeling of resistance on percussion.

With medium-sized effusions, where the dullness does not extend over the whole back, the upper boundary of the dullness usually forms an oblique line, highest at the vertebral column and thence running obliquely downward to the side of the thorax. We can no more confirm the opposite opinion, which some authors hold, than can Weil and others, yet of course no one can establish a schematic rule. The lower boundary of the effusion can not be distinguished on the right by percussion from the liver dullness. On the left in front and on the side, however, we can often distinguish the dullness of the effusion from the tympanitic resonance of the stomach, and this is of diagnostic value (see the displacement of organs, as given below).

[In moderate effusion without adhesions or pneumonic complications, the line of flatness in the back, the patient being in the vertical position, is lowest near the spinal column, and rises in a curve like the letter S as it passes outward toward the axillary region. The experiments by Dr. Garland, of Boston, with reference to this point are well known. Before attempting to mark out this line the patient should be told to take several deep inspirations, in order to inflate the triangular portion of lung which dips down near the vertebræ.]

The percussion-note above a pleuritic effusion deserves attention. The beginning of pleuritic dullness is almost always relative, gradually passing to an absolute flatness. The pulmonary resonance above the beginning of dullness is usually tympanitic, from retraction of the lung-tissue. We find the tympanitic resonance beautifully distinct in large effusions in the first and second intercostal spaces in front. It is loud and deep, and remains unchanged with the mouth open—Skoda's resonance. With very large effusions, which cause an actual compression of the lung, we sometimes find, in the second intercostal space, a dull tympanitic resonance, which becomes higher on opening the mouth. This resonance arises from the vibrations of air in a large bronchus surrounded by compressed lung—" Williams's tracheal tone." With large effusions we sometimes hear over the retracted lung, in the upper anterior intercostal spaces, a distinct buckram sound—the " cracked-pot sound."

Displacement of the neighboring organs, which is made out chiefly by percussion, forms one of the most important physical signs in pleurisy with effusion.

In right-sided effusions the liver, especially the right lobe, is displaced downward. We find the lower border of the liver dullness extending several centimetres below the ribs. In very large effusions the liver may be pushed down to the level of the umbilicus. The pushing of the mediastinum to the left in large effusions may be recognized upon percussing from the right toward the left, by dullness over the upper part of the sternum, reaching to or beyond the left border of the sternum. The displacement of the heart to the left in the majority of well-marked cases is associated with a displacement of the apex of the heart upward. This is easily explained by considering the position of the heart and the direction of the pressure, which first acts from below. We recognize the displacement of the heart chiefly by the position of the apex-beat, which is seen and felt at or outside the left mammillary line in the fifth space, or often higher, as we have said—in the fourth. Percussion gives a corresponding displacement of the left boundary of the cardiac dullness to the left.

In left-sided effusions the displacement of the heart to the right, which can usually be made out even in moderate effusions, is especially noticeable. Resonance over the lower part of the sternum is diminished, the heart's dullness extends to the right border of the sternum or several centimetres beyond it. In the most marked cases the heart is pushed to the right mammillary line. The displacement of the mediastinum is also to be made out over the upper part of the sternum, the dullness reaching to the right border of the sternum or beyond. The low position of the diaphragm is made out by a depression of the left, and in marked cases of the right, lobe of the liver. It is an especially important sign, however, that dullness occurs in the zone, about a hand-breadth wide, of normal tympanitic resonance above the left border of the ribs—the "semilunar space" of Traube. The normal tympanitic resonance here comes from the stomach or large intestine. As the diaphragm is pressed downward the pleuritic effusion presses on these organs. The semilunar space is therefore diminished, and finally, with large effusions, there is absolute dullness down to the edge of the ribs.

Changes in dullness in pleuritic effusions may occur with a change of the patient's position, but they may often be absent on account of adhesions. The respiratory displacement of the lower border of the lung is almost always absent.

Auscultation always gives a diminished respiratory murmur over the pleuritic effusion. With a beginning effusion it may sound approximately vesicular, but later it becomes indefinite, hoarse, and finally bronchial, if the larger bronchi remain open for the respiratory current of air. The bronchial respiration sounds distant and low, and has the character of the sharp German *ch*, but in rare cases it also assumes a distinct amphoric tone, so that it sounds almost like a cavernous respiration. The respiratory murmur may finally disappear entirely over very large effusions. Above the upper boundary of the effusion the respiration almost always sounds harsh. Among the adventitious sounds we must mention the pleuritic friction sound, which of course can be heard only at the upper boundary of the effusion, where the two pleural surfaces meet. Moist râles and rhonchi signify a co-existing disease in the lungs. With slight effusions we often hear, on deep breathing, pure crepitant râles on inspiration, as the walls of the alveoli and bronchioles in the atelectatic lung, which were stuck together, are torn apart.

On auscultation of the voice we sometimes hear bronchophony, and sometimes that bleating, nasal sound known as ægophony. Baccelli advanced the theory that auscultation of the whispered voice might be of service in diagnosticating the character of the effusion. With a serous effusion we can understand a whisper distinctly through the thorax, but not with a purulent effusion, since theoretically the cell-elements destroy the waves of resonance. This theory holds true in many cases, but by no means in all.

On auscultation of the heart we notice, as a result of its displacement, an abnormal extension of the region over which the heart-sounds are audible. If the inflammation spreads from the pleura to the outer surface of the pericardium, we can sometimes hear an extra-pericardial friction-rub, accompanying both the respiration and the action of the heart.

The vocal fremitus is always diminished over the pleuritic effusion, and in marked cases is entirely absent.

3. **Absorption of the Effusion—Pleuritic Contraction.**—The beginning absorption of the effusion is usually first made evident by the percussion-note in the upper part of the dullness becoming clearer and sometimes tympanitic. The respiratory murmur is also plainer. Where it was bronchial it becomes indefinite and gradually vesicular again. The vocal fremitus is again to be felt. All these improvements take place gradually but slowly. It is usually a very long time before the percussion-note resumes its normal clearness.

The changes in the form of the thorax are especially striking. Only in pleurisies with slight effusion does the somewhat expanded thorax resume its old form without further change. After every severe pleurisy with large effusion there is, during its absorption, a marked contraction of the affected half of the chest. In cases of moderate intensity the contraction affects only the lower lateral portions of the thorax, in severe cases the upper and anterior portions as well. We find the most marked contractions in children and young persons with a yielding thorax. The circumference of the affected side is much less than that of the sound side. The ribs are pressed together and the intercostal spaces become very narrow. The fossæ are deepened and the nipples and shoulder-blades are drawn nearer the vertebral column, which takes on an abnormal lateral curvature, in which its convexity is directed usually toward the affected side, but sometimes to the sound side. Dullness and diminution of the respiratory murmur and vocal fremitus continue with the contraction of the pleura, but they no longer depend upon the presence of a fluid effusion, but are due to the pleuritic thickening.

The process of marked contraction always lasts for months, or even longer. In favorable cases the contraction of the thorax may be readjusted very much later, often after years. The thickening is absorbed, and the lungs and thorax gradually expand, but in other cases there are extensive adhesions between the pleural surfaces, especially over the lower lobe, which result in a permanent disturbance of respiration. In almost all cases of pleurisy with contractions there arises a vicarious emphysema in the lung on the sound side.

Complications.—Peculiar complications of pleurisy are rare. Where such occur they are due either to the primary disease which has led to the pleurisy, or the simultaneous action of the same cause of disease, such as tuberculosis. Hence it happens that we speak of the frequent "complication" of pleurisy with chronic bronchitis or with tuberculosis of the lungs or other organs. It is important to bear in mind that, by a direct advance of the inflammation, the pleurisy may also invade the pericardium, and rarely the peritoneum, through the diaphragm; but we see this extension of the process almost solely in tubercular and purulent pleurisies. We must mention, finally, that we have seen several cases with a large serous effusion, in which an acute hæmorrhagic nephritis occurred. For the paralysis of the arm on the corresponding side observed in some cases of empyema, compare page 538.

Different Forms of Pleurisy.

1. **The Simple Fibrinous or Exudative Pleurisy,** as a result of croupous or extensive lobular pneumonia, often causes but few symptoms in comparison with the primary disease. Recovery is usually complete, but may be much delayed, as in croupous pneumonia.

The so-called primary simple fibrinous or sero-fibrinous pleurisy, which we must regard as a distinctly rare affection, contrary to the generally prevailing opinion, has a like favorable course.

2. **Tubercular Pleurisy.**—In an ætiological sense we must declare the larger part of the ordinary "pleuritic effusions," which clinically seem to be primary to be tubercular. We do not know how far at first the specific anatomical changes of tuberculosis are present, or whether there is always some previous tubercular affection in the lungs or bronchial glands, but the further course of the cases, if we can watch them for years, almost always permits us finally to recognize the tubercular nature of the disease; yet we can not say that phthisis is always the immediate sequel of the pleurisy.

In a comparatively small number of cases do the symptoms of acute tuberculosis, or more frequently of chronic phthisis, appear as an immediate result of the pleurisy, which at that time is usually still present or in the contracting stage. The objective changes of phthisis are evident either in the apex or in the lower lobe of the affected side. The fever continues, the pulmonary affection advances, the other lung is also attacked, and the disease takes a fatal course under the type of an ordinary phthisis, now more acute and now more chronic.

In other cases acute tubercular affections arise sooner or later as a result of the pleurisy—tubercular meningitis, or general miliary tuberculosis. In other cases still the disease develops under the form of tuberculosis of the serous membranes, to which we will return again in the description of tubercular pericarditis and tubercular peritonitis. We often have to do with a double pleurisy with no evident complication in the lungs. In varying succession are added the symptoms of chronic tubercular peritonitis, with pain, swelling, and effusion of fluid into the abdomen, or the symptoms of tubercular pericarditis. Death finally ensues with persistent hectic fever and increasing general emaciation and weakness. The whole affection usually runs a chronic course, lasts for months, and often shows marked remissions and temporary improvements.

In very many instances the pleuritic effusion has throughout an apparently favorable course. After some weeks the fever ceases, the effusion is absorbed, the patient gets up, and is finally discharged as nearly well. Of course, some dullness and retarded motion often remain in the affected side, but even these may gradually disappear. These cases, too, very often turn out in the end to be tubercular. After a longer or shorter period of apparent health, sometimes after the lapse of years, a "new" disease appears—that is, either a return of the pleurisy, a pleurisy on the other side, or some other acute or chronic tubercular affection. In such cases, too, we must look upon the former pleurisy, in an ætiological sense, as tubercular. It is not impossible, however, for even a tubercular pleurisy to recover, and for the recovery to be permanent, if no other organ is at the same time affected by tuberculosis, especially if the lungs remain unaffected.

Finally we must mention the cases where a pleuritic effusion develops secondarily to an already pronounced phthisis. Here too we almost always have to do with a tubercular pleurisy.

The anatomical changes in tubercular pleurisy consist in the ordinary signs of inflammation, and also the presence of the specific nodules of tubercle. The number of tubercles differs very much in different cases. The pleura is in some cases completely studded with miliary nodules, and in others we find the tubercles, at least with the naked eye, only in single spots. The effusion is usually of a sero-fibrinous character. Sometimes it is hæmorrhagic, as the majority of cases of apparently primary "hæmorrhagic pleurisy" are generally of a tubercular nature. The exudation is rarely purulent, and when this is the case it is apparently invariably caused by the simultaneous infection of the pleura with another sort of virus which gives rise to the suppuration.

3. **Purulent Pleurisy—Empyema.**—We have already described the ætiology of empyema, and we have seen that it can be excited only by infection of the pleura with a specific virus which can set up suppuration. The clinical symptoms are usually severe. The fever is higher than in the other forms of pleurisy, but it is irregularly intermittent, and is often associated with chills. There are severe general symptoms besides the fever, such as great dullness, headache, and a dry tongue. We sometimes notice a slight œdema of the chest-wall on the affected side. Otherwise the local symptoms and disturbances are, of course, the same as in the other forms of pleurisy. If the pus is not evacuated artificially, the empyema may finally break externally or into the lungs (*vide supra*). In the latter case a very large expectoration of pus suddenly occurs, and is usually followed by pneumothorax.

Diagnosis.—Our chief attention in regard to diagnosis is directed to the distinction between pleurisy and acute or chronic pneumonia, which is not very easy in all cases. We will therefore briefly contrast the distinctive features as made out on physical examination.

Inspection.—A marked distention of the affected side points to effusion; it does not occur in pneumonia.

Percussion.—The dullness in pleurisy is complete, and the feeling of resistance on percussion is very marked; in pneumonia, however, the dullness is rarely so marked, and there is often a tympanitic sound. The discovery by percussion of signs of displacement of the neighboring organs is of especial weight, as these signs are always absent in uncomplicated pneumonia, while with few exceptions they can easily be demonstrated in every case of pleurisy where the exudation is at all considerable.

Auscultation.—Diminished or suppressed respiratory murmur points to pleurisy, loud bronchial breathing and râles to pneumonia; but we must not forget that in pneumonia auscultation may give the same signs as in pleurisy, if a bronchus is plugged.

Vocal Fremitus.—Marked vocal fremitus over dullness is direct evidence of pneumonia, diminished or absent vocal fremitus of pleurisy; but the vocal fremitus may also be diminished in pneumonia if a bronchus is plugged.

Besides the physical signs, we must of course consider the other symptoms—the manner of onset, the course of the disease, the fever, the sputum, the occurrence of herpes, etc.

If we have diagnosticated a pleuritic effusion, the next question is always as to the character of the effusion, because the prognosis and treatment are to a large degree dependent upon this. Although certain well-known ætiological circumstances, and the severity of the fever and the general symptoms, often permit us to suspect the nature of the effusion, whether serous or purulent, the only certain information comes from an exploratory puncture with a hypodermic syringe. There is not the least reason, if the syringe be carefully disinfected and the fluid be cautiously withdrawn, why we should not perform this perfectly safe experiment in all important cases, thus making the diagnosis certain. Besides a macroscopic inspection, a careful microscopic examination of the fluid withdrawn is sometimes of importance. Besides the ordinary constituents—red and white blood-corpuscles, endothelial cells, and cholesterine crystals—we may sometimes find something of special diagnostic significance, like bacteria in septic pleurisy, carcinoma-cells in cancerous pleurisy, etc.

We can not always judge from the beginning whether a pleurisy is tubercular or not; but we should never forget, as has already been affirmed, that in every case of pleurisy, even if it is apparently primary, there is a strong suspicion of tuberculosis. We must observe in particular the general habit and the nutrition of the patient, and inquire into the hereditary predisposition and any previous illnesses. In the further course of the disease persistent hectic fever, slowly increasing emaciation and pallor, fresh relapses, and the onset of pulmonary symptoms, point to the tubercular character of the pleurisy. Every double pleurisy, and every pleurisy associated with pericardial or peritoneal symptoms, leads us most decidedly to suspect tuberculosis. A hæmorrhagic effusion, as we have said, points strongly to tuberculosis. Tubercle bacilli are usually not present in the fluid exuded in tubercular pleurisy, because the tubercular nodules on the serous membrane hardly ever ulcerate.

Prognosis.—The prognosis, as regards the immediate danger of the disease, depends entirely upon the severity of the symptoms, and especially upon the dyspnœa. The prognosis, as regards the further course of the disease, depends chiefly upon the nature of the pleurisy. Many secondary and also many apparently primary pleurisies, although extensive, recover completely and permanently after weeks or months. Unfortunately, we only too frequently have to give a doubtful or an unfavorable prognosis, especially if the tubercular nature of the pleurisy be probable or certain. The prognosis of empyema depends partly upon the underlying disease, but especially upon judicious and timely operative interference. The healing process in empyema may occupy many months, but it may finally be complete. The possibilities of a spontaneous rupture of empyema, internally or externally, have been mentioned above. With incomplete healing, which leaves a pleural fistula, we must fear the appearance of general amyloid disease in various organs.

In rare instances with large effusions sudden death occurs, an event which can not always be explained with certainty. Probably there are different factors in different cases, such as pulmonary embolism, cerebral embolism, sudden cerebral anæmia, weakness of the heart, or the onset of pulmonary œdema.

Treatment.—In the beginning of the disease the treatment is purely symptomatic. We try to alleviate the patient's symptoms, the pain and dyspnœa, by local applications, especially by mustard plasters, warm poultices, which are usually more grateful than cold applications, sometimes, too, by dry cups, also by embrocations with chloroform liniment, and, with severe symptoms, by morphine internally or subcutaneously. Unfortunately, we have but few remedies to check the inflammatory process in the pleura. If an ice-bag is well borne, it may be of service. The efficacy of the much-used painting with iodine is doubtful, but it may always be tried if there is a severe pleuritic pain. An iodoform ointment, one to fifteen, or iodoform collodion, perhaps deserves more confidence. If a large effusion has formed, we try to hasten its absorption by diuretics. Acetate of potassium, squills, or boro-tartrate of potassium and sodium* [tartarus boraxatus, P. G.] may be prescribed. When there is weak action of the heart, infusion of digitalis may be given, alone or combined with diuretics. The attempt has also been made to draw off a large amount of water from the body, and thus to hasten the absorption of the effusion, by prescribing drastic purgatives or such diaphoretics as pilocarpine, salicylate of sodium, and hot packs. The so-called Schroth's treatment serves the same purpose—that is, withdrawing as much fluid as possible from the ingesta—but the last-named methods of treatment generally have the result of exhausting and weakening the patient. We therefore make use of them only rarely. It is very doubtful whether the internal exhibition of iodide of potassium can further the absorption of the effusion, as many physicians believe. Besides the treatment by drugs we must also take care to give the patient sufficient food, in order to prevent the loss of strength.

In many cases the operative treatment of pleurisy (introduced by Trousseau)—the evacuation of the effusion by puncture—is of the greatest importance. Many cases of pleurisy with effusion run a favorable course without it, and we consider it at least superfluous to puncture every effusion without sufficient grounds, but puncture is often one of the most serviceable therapeutic influences at our command. The first and most important indication for puncture is present when the effusion becomes so large as to be directly dangerous to life. As soon as the patient's dyspnœa reaches a dangerous degree, and the cyanosis becomes marked and the pulse weaker, a puncture must be made as a direct vital indication. Where the exudation is of considerable size there may be a very sudden aggravation of the symptoms, so that in such cases one should not wait too long. Trousseau urged that tapping should invariably be performed when the dullness caused by the exudation involves not only the back, but also the whole or nearly all of the anterior wall of the thorax. The benefit of such a puncture is often pronounced.

[* We would use the simpler bitartrate of potassium.—TRANS.]

The second indication is a too protracted absorption of the effusion. Puncture is indicated if the effusion does not disappear after an apparent remission of the inflammatory symptoms, especially after the fever has gone. We often see the further absorption stimulated by such a puncture. It has been said that, if possible, tapping should be delayed until the fever has ceased, but to us this does not seem at all necessary. We have repeatedly performed paracentesis in febrile cases, whether because of the extent of the exudation, the dyspnœa of the patient, or the duration of the disease; and we have not infrequently observed a speedy abatement of the fever after the withdrawal of the exudation. In early cases, of course, one would not tap unless there were some urgent reason for it.

[There is considerable danger in delaying interference with a large effusion, especially if it has come on pretty rapidly, however comfortable the patient may be. The liability to sudden and fatal dyspnœa under these circumstances is now well recognized.]

As regards the performance of the puncture, we can not here go into all the numerous methods and forms of apparatus proposed. The distinctions are immaterial. The simpler the method, the easier it is to perform, and hence the better it is.

Every puncture must be preceded by an exploratory puncture in order to settle the diagnosis as to the presence and character of the exudation. A medium-sized trocar with a lateral opening, to which a rubber catheter can be fastened, serves to evacuate the fluid. Billroth's and Fräntzel's trocars are useful. The operation can be done with at least equal convenience by means of a hollow needle. Our own experience with the form of needle proposed by Fiedler leads us to recommend it highly, for the point of this needle can not irritate the tissues, and it permits of the removal of any clots of fibrine which may offer obstruction to the flow of liquid. The instruments and the chest-wall at the point of puncture must be carefully disinfected. We usually choose rather a low point for puncture, somewhere in the sixth intercostal space, in the middle or posterior axillary line. The patient sits up in bed, but is held and supported by another person, when it is possible. Before and during the puncture he takes a little strong wine. A small cut of the skin beforehand sometimes aids the insertion of the trocar. In many cases, especially with a large effusion, we can evacuate a large part of the fluid by simple puncture and siphoning, since the prevailing pressure in pleuritic effusions is, with few exceptions, positive, from ten to twenty-five millimetres of mercury. The evacuating tube of the trocar must be previously filled with carbolized water and conducted under a layer of the same into the vessel prepared to receive the effusion The evacuation of the effusion should always be slow and gradual. Many physicians advise to cease when 1,500 c. c. have been removed, but if the fluid is allowed to escape slowly and everything is going well, this quantity may often be exceeded with impunity in the case of large exudations. While, as we have said, in most instances the exudation may be satisfactorily removed by simple puncture and siphonage, it will sometimes prove necessary to employ aspiration. Hence some physicians invariably do so, and to this there is no objection, even if it is unnecessary. The forms of apparatus most used for this are those invented by Dieulafoy, and Potain. In puncture with aspiration we proceed more slowly and cautiously.

[The necessity for two punctures—one exploratory, the other distinctly operative—does not seem clear. The two can be perfectly combined, a fair-sized trocar or needle being as easy of introduction and producing really no more pain than a very fine one, and being more sure to give results on which reliance can be placed. A preliminary incision through the skin seems also useless, and has never been practiced by the editor in a considerable experience with these cases. An ordinary Davidson's syringe makes a very satisfactory pump, and can always be obtained. The needle or trocar should be withdrawn immediately as soon as cough comes on, or the patient shows the slightest discomfort due to the removal of the fluid.]

Unpleasant incidents which may cause a cessation of the process are rare. If the patient complains of dizziness and faintness we must stop. Sometimes very severe cough occurs on puncturing, at which we must also stop. Sometimes we see, after the puncture, an abundant expectoration of frothy, serous sputum—"*expectoration albumineuse*"—a kind of pulmonary œdema, caused, perhaps, by a marked perviousness of the walls of the vessels, or by weakness of the left ventricle.

When the process is over, we close the little opening with a bit of sticking-plaster or with iodoform collodion. A regular surgical dressing is scarcely ever necessary.

If the exploratory puncture has shown a purulent effusion, we can first evacuate the pus by puncture, if the vital indication exists. But a permanent cure from tapping is exceptional. The pus almost always reappears. Empyema is like an abscess, which can not be cured until a permanent, free exit for the pus has been provided; hence drainage of the pleural cavity must be provided for after the empyema has been evacuated. To this end, therefore, most physicians at present open the pleural cavity in empyema by an incision—"thoracotomy." We incise it by layers in the fifth or sixth intercostal space, outside of the mammillary line. The length of the incision is some two or three centimetres. After the pus has been removed we have an artificial pneumothorax opening outward. The incision may be dilated if necessary, and a long drainage-tube is then thrust through it into the pleural cavity, the external end being prevented from slipping into the chest by transfixing it with a safety-pin. Then an antiseptic bandage is applied, which at first must be changed frequently, as long as there is much secretion. If the pus has a sufficient exit, the fever will subside at once, in an uncomplicated empyema. A fresh rise of temperature almost always depends upon the retention of pus. The point of puncture is soon changed to a good drainage-canal by granulations. We can then take out the tube, clean it, and easily reinsert it. If all goes well, we can gradually shorten the drainage-tube more and more, and finally remove it altogether. The cavity of the empyema has filled with granulations, and there follows a definite healing, almost always, of course, with marked contraction. Many cases do not run so undisturbed a course. If the exit is insufficient, we must sometimes dilate the opening with some blunt instrument, and insert a larger tube. In simple empyema, where the pus does not smell badly, it is unnecessary to wash out the pleural cavity with disinfecting fluids, like salicylic and boracic acid solution, permanganate of potassium, or dilute chlorine-water. Carbolic acid should not be used, on account of the danger of poisoning by it. If the empyema is septic, or if there is from the first a stinking, sanious exudation, it is necessary to wash it out. We must then sometimes make a second counter-opening in the chest-wall in order to get a completely free discharge, and to wash out the pleural cavity well. The details upon this and upon many other special points in the treatment of empyema, and especially upon the resection of a rib, which is sometimes necessary, are to be found in the text-books on surgery. It may be added that in the surgical clinic at Leipsic the pleural cavity was often punctured with an ordinary large-sized trocar, through the cannula of which a drainage-tube was introduced into the chest. The cannula was then removed, and the further treatment was as above described.

In treating the chronic, contracted pleurisies with thickening, but without fluid effusion, methodical respiratory efforts, "lung-gymnastics," are of use. Besides these we should strengthen the general condition as much as possible. We should advise the patient to breathe deeply, and prescribe cold sponging of the chest daily. Inspiration of compressed air by means of a pneumatic apparatus is often accompanied by good results. Well-to-do patients, who have had a severe pleurisy, should be sent to a suitable climatic health-resort.

CHAPTER II.

PERIPLEURITIS.

UNDER the name of "peripleuritis" Wunderlich was the first to describe a rare form of disease, which consists of an inflammation of the connective tissue between the costal pleura and the ribs, and which terminates in the formation of an abscess. Similar cases have since been repeatedly observed, and all were characterized by the lack of any discoverable ætiology. There is neither a previous injury, nor a primary disease of the ribs or the pleura. Nevertheless the cause must be sought in an invasion of micrococci, which excite the suppuration. A knowledge of the particulars, however, can only be gained from future investigations. They will determine whether peripleuritis can be regarded as an independent disease or not.

The disease occurs chiefly in men. It usually begins suddenly with a chill, and runs its course with quite a high fever. In pronounced cases the local symptoms have the greatest similarity to those of an empyema, but the greater protrusion of the chest-wall is striking. The ribs are crowded apart by the abscess, and there is often spontaneous rupture externally, scarcely ever into the pleura. Percussion gives no symptoms of displacement of the neighboring organs, a distinguishing point from empyema. It is of diagnostic significance that we can often discover normal lung-tissue containing air below the abscess. The mobility of the lower border of the lung is also usually retained, contrary to what is the case in empyema. Another important sign was first brought to notice by Bartels: the wall of the abscess relaxes on inspiration and becomes tense on expiration. We may also mention that acute nephritis has often been observed among the complications.

From these points we may be able to make the diagnosis during life, at least in many cases. The prognosis is quite unfavorable, but recovery does occcur. The treatment can be only operative, and is quite analogous to that for empyema.

CHAPTER III.

PNEUMOTHORAX.

(*Pyo-pneumothorax. Sero-pneumothorax.*)

Ætiology.—Pneumothorax—that is, a collection of air or gas in the pleural cavity—arises, in an overwhelming majority of cases, from the penetration of air into the pleural cavity through an opening in the pleura. The opening may be in the external chest-wall from a penetrating wound of the chest or an empyema operation, or it may be in the pulmonary pleura. Pneumothorax is by far most frequently associated with phthisis, when a cavity lying beneath the pulmonary pleura perforates into the pleural cavity. This is more apt to happen in comparatively acute phthisis than in very chronic forms, because the extensive adhesions and contractions in the latter hinder its development. It usually appears in quite far advanced cases, but it may sometimes arise with but slight changes in the lung.

Pulmonary gangrene or abscess, as well as phthisis, may cause pneumothorax by perforation into the pleural cavity. It may also arise from the rupture of an empyema into the lung. In some cases a perforation of the œsophagus or stomach into the pleura, as in gastric ulcer, has been observed, with the formation of pneumothorax.

The development of this condition from severe injuries, as from laceration of the lung, without injury to the chest-wall, is rare. Forced respiratory movements, associated with physical exertion, seem especially capable of exciting such a process. We have ourselves seen pneumothorax develop suddenly in a previously healthy woman while hanging out her washing, and another time in a young man during very labored rowing. Both cases recovered rapidly and completely.

All the last-named causes, however, are far less important than phthisis. We have yet to mention that in phthisis, too, there is sometimes a definite exciting cause—severe coughing, vomiting, or muscular exertion—which may favor the development of the pneumothorax.

Many authors maintain that, by decomposition of a putrid pleuritic effusion, gas may be produced, and thus we may have pneumothorax; but such an event is extremely rare, if it ever happens.

Pathological Anatomy.—On opening the pleural cavity a part of the air usually rushes out, sometimes with an audible noise. We then look into a large cavity filled with air, and find, in total pneumothorax, the lung completely retracted and lying compressed against the vertebral column. If, however, the air fills only a part of the pleural cavity, as a result of extensive adhesions of the pleuræ, we speak of a circumscribed or sacculated pneumothorax. The amount of air contained in the pleural cavity may reach 2,000 cubic centimetres. The pressure which it is under is almost always positive—on an average five or ten centimetres of water.

In the cases of pneumothorax arising from perforation of the pulmonary pleura we can usually make out the point of perforation in the lungs. This is more frequently situated in the upper lobe than in the lower. Sometimes it is already grown over or is covered by a layer of fibrine, and can no longer be found. The opening is usually quite small, but it may reach the size of a ten-cent piece. Left-sided pneumothorax seems to be somewhat more frequent than right-sided.

The pleura itself is only rarely normal. Usually agents of inflammation have entered it with the air, and hence it is found in a state of inflammation. A part of the cavity is then filled with effusion. This is usually wholly purulent—pyo-pneumothorax—or sero-purulent, but it may even be serous or sero-fibrinous—sero-pneumothorax, or hydro-pneumothorax.

The neighboring organs, especially the heart and liver, are found pushed out of their normal position, as in large pleuritic effusions.

Symptoms and Course.—The onset of pneumothorax (we speak in what follows especially of pneumothorax in phthisis) is quite often made known by a sudden pain, usually associated with an increase of the dyspnœa and of the general symptoms. There is sometimes collapse. The temperature sinks below normal, the pulse rises to 140 and over. The patient looks pale and cyanotic. He usually sits upright or is in a half-sitting position in bed, either more on the affected side, in order to use the normal lung as much as possible for breathing, or more on the sound side on account of the tenderness. If the pneumothorax has come on as a result of the rupture of an empyema into the lungs, there is at the same time a very abundant expectoration of pus.

Although in many cases the symptoms mentioned lead to a suspicion of pneumothorax, yet a certain diagnosis can be made only after a physical examination.

Inspection gives a very marked distention of the affected side. The intercostal spaces are stretched out, or even protruded. In some cases, as we have ourselves noticed, there is a marked elastic "air-cushion feeling" on palpating the intercostal spaces. On respiration, the affected side is almost entirely motionless, while the excursions of the other side are the more marked. The displacement of the heart is often evident from the visible displacement of the apex-beat.

Percussion gives over the pneumothorax a remarkably loud, full, deep note, but usually not tympanitic, on account of the tension of the walls. It is especially important to note that this resonance extends beyond the normal limits of the lung on the right down to the seventh or eighth rib, and on the left to the fifth or sixth rib, and sometimes even to the edge of the thorax.

The displacement of the neighboring organs can also be made out by percussion. With right-sided pneumothorax we find the lower border of the liver dullness abnormally low, and the left border of the cardiac dullness pushed over to the anterior axillary line. In left-sided pneumothorax the cardiac dullness is usually entirely absent from its normal place, and is found instead to the right of the sternum. The left lobe of the liver is pushed downward, and we do not find tympanitic resonance in the semilunar space.

Upon auscultation we are struck by the entire absence of respiratory murmur. This is in special contrast to the clear resonance on percussion. In other cases, however, we hear a number of metallic sounds, at least in many places and at many times, which are very characteristic of pneumothorax. First among these is amphoric, metallic respiration. This arises in open pneumothorax (*vide infra*) from the direct passage of the air in and out, but in all other cases it is the ordinary respiratory murmur of the larynx, trachea, and lungs, which has acquired a metallic timbre from resonance in the pneumothorax. In an analogous way arise the metallic-sounding râles [" metallic tinkling "], and the metallic resonance of the cough and voice. Heubner has devised a particularly beautiful and practically important method for demonstrating the metallic sound in pneumothorax. If we strike lightly on a pleximeter with a little rod, usually the handle of a percussion hammer, while we auscult near it—" rod percussion "—we very often hear quite a distinct high metallic sound.

The vocal fremitus over a pneumothorax is usually diminished, but it may be felt in spite of quite a large collection of air.

A number of special physical signs are found if a purulent or serous effusion be added to the pneumothorax. In the first place, the resonance is thereby rendered dull, to a greater or less extent, in the lower parts of the chest. The boundaries of the fluid by percussion show a very evident change with the patient's change of position, because the fluid in pneumothorax can move easily in all directions. Since the form of the air-space left must therefore change, the pitch of all the metallic sounds manifest anywhere must change too, when the patient sits up or lies down—Biermer's change of note. In many cases at every motion of the fluid, excited for example by slightly shaking the patient, there arises a metallic splashing sound, the so-called succussion of Hippocrates.

Forms of Pneumothorax.—According to the condition of the perforation during life, we distinguish three kinds of pneumothorax (Weil). We speak of an "open pneumothorax," if the point of perforation remains open, so that the air on respiration constantly passes in and out of the pleural cavity. If the perforation is completely closed, we have a "closed pneumothorax." The third and most frequent form is the "valvular pneumothorax," in which air enters the pleural cavity at each inspiration, but on expiration there is a valve-like closure of the perforation, and thus the air can not escape again; but as soon as the pressure in the pleural cavity increases so that no more air can enter it on inspiration, the valvular pneumothorax becomes closed. In open pneumothorax the pressure in the pleural cavity must be the same as the atmospheric pressure. A positive pressure in the pleural cavity can exist only in a closed or a valvular pneumothorax.

The clinical diagnosis of the form of pneumothorax is not always possible, and has usually no great practical importance. The very loud, metallic, amphoric respiratory murmur, which may be heard in open pneumothorax, must be mentioned, and Wintrich's change of pitch (see page 223) can sometimes be heard in this form. It is worthy of mention that symptoms of displacement of the neighboring organs must also arise in open pneumothorax. The predominant atmospheric pressure here is positive in contrast to the negative pressure in the other pleural cavity, and to the normal negative pressure which previously acted on the upper surface of the diaphragm. A very marked protrusion of the affected side, and great displacement of the heart and liver, however, speak most strongly against an open pneumothorax. Some authors have tried to find a point of distinction for the different forms of pneumothorax in the composition of the gas in the pleural cavity, but the results of chemical analysis are still contradictory. According to Ewald, we find in open pneumothorax not over five per cent. of carbonic acid and about twelve to eighteen per cent. of oxygen; in closed pneumothorax, however, fifteen to twenty per cent. of carbonic acid and ten per cent. at most of oxygen. If in an open pyo-pneumothorax or sero-pneumothorax the point of perforation lies below the level of the fluid, there sometimes arise on every inspiration metallic sounds, since the bubbles of air drawn in rise and come up through the fluid—"the water-pipe sound," "metallic tinkling." A peculiar sipping and short snapping sound on inspiration, heard by us in one case, seems to point directly to the existence of a valvular pneumothorax. Upon change of position of the patient we can often make out Biermer's change of note.

Course of the Disease.—In many cases the occurrence of pneumothorax causes such a high degree of respiratory disturbance that death ensues in a few hours or days. In other cases the patient improves, and may feel quite well for a long time in spite of the condition. Usually, of course, the underlying disease, commonly phthisis, leads to death after a longer or shorter period. Pneumothorax may sometimes heal. The healing takes place in this way, that the air is first replaced by a fluid effusion, and then the latter is gradually absorbed, but the air may also be wholly or partly absorbed. It depends upon the origin of the lesion, then, and upon the intensity of the underlying disease, whether the recovery is permanent or not.

Diagnosis.—The diagnosis of pneumothorax is usually easy with careful examination, but the symptoms may sometimes be of so little prominence as to excuse overlooking it. It is very difficult and often quite impossible to make a differential diagnosis between very large cavities and a saccular pneumothorax, since both conditions must have in part precisely the same symptoms. We may mention as the chief points in distinction: A cavity is apt to be situated in the apex, pneumothorax in the lower part of the thorax; over a cavity the chest-wall is often sunken in, over pneumothorax it is prominent; the vocal fremitus is usually marked over a cavity, weak over pneumothorax; symptoms of displacement, and also evident succussion, point to pneumothorax.

Treatment.—The only remedy which can alleviate the often severe symptoms is morphine. In hopeless cases we may confine ourselves to prescribing this exclusively, both internally and subcutaneously; but in cases where the patient's previous strength was fairly good, we may try to obtain, by operative interference, an improvement of the symptoms, and finally a complete healing of the pneumothorax. If there is simple pneumothorax without a fluid effusion, we try to remove as much air as possible by aspiration. With a large serous effusion removal of the effusion is indicated, and with purulent effusion either a simple puncture, or, better, puncture or incision with subsequent drainage. The method then is just the same as in the treatment of empyema. We must also state that the above-mentioned improvement or healing in pneumothorax has been repeatedly observed independently of any operative interference.

CHAPTER IV.

HYDROTHORAX. HÆMATOTHORAX.

1. **Hydrothorax.**—We term the occurrence of a serous transudation into the pleural cavity, independent of an inflammation of the pleura, hydrothorax, or thoracic dropsy. The cause of hydrothorax is in rare cases a local hindrance to the outflow of venous blood or lymph from the thorax, as in compression of the veins or of the thoracic duct by tumors; but in the great majority of cases the hydrothorax is part of a general dropsy, occurring especially in pulmonary emphysema and in cardiac or renal disease. Hydrothorax is often first developed after marked œdema of the subcutaneous cellular tissue and ascites, but it may sometimes be one of the first symptoms of dropsy. It is usually bilateral, but it is often unilateral, or at least much larger on one side than on the other. The pleura itself is normal or else œdematous. We often find it traversed with a network of dilated lymphatics. The serous fluid in hydrothorax is distinguished from an inflammatory serous effusion by the smaller amount of albumen in it, by the scanty number of cell-elements, and by the absence of or the slight tendency to spontaneous coagulation.

The clinical importance of hydrothorax lies in the hindrance to respiration which it causes. As a result of this the hydrothorax may be regarded in many cases, especially in renal disease, as the chief cause of death. The objective evidence of it comes from the physical examination, which must, in general, give the same signs as in pleuritic effusion. We can note only the bronchial respiration from compression in hydrothorax, which is often very loud, and which may even give rise to a confusion with pneumonic infiltration in the lungs. This frequent and very loud respiratory murmur, contrasting with that of pleuritic effusion, is explained by the normal condition of the lungs and the absence of all adhesions. For the same reason, too, the change in the boundary of the dullness, as a result of the patient's change of position, is usually more marked in hydrothorax than in pleuritic effusion. We often hear crepitant râles over the hydrothorax, which arise in the retracted and partly atelectatic lung. The chief factor, however, in distinguishing hydrothorax from a pleuritic effusion is the character of the primary disease if any exists.

The treatment is directed especially to the underlying disease. If we succeed in regulating the heart's action, or in restoring the secretion of urine, the hydrothorax often disappears with the other dropsical symptoms. If the dyspnœa caused by it reaches a dangerous degree, we often see great relief from aspirating the fluid. The nature of the underlying condition, of course, renders the benefit in many cases only transitory.

2. **Hæmatothorax.**—Effusions of blood into the pleural cavity (hæmatothorax)

arise most frequently from traumatic lacerations of blood-vessels, rarely from the bursting of an aneurism of the aorta into the pleural cavity, from erosion of an intercostal artery in caries of the ribs, from the rupture of a cavity into the pleura in phthisis, if it simultaneously opens a blood-vessel, etc. In many such cases a typical exudative pleurisy follows the effusion of blood. The physical signs are the same as in other pleural effusions. Severe dyspnœa may demand the removal of the blood by puncture, or even by an incision.

CHAPTER V.

NEW GROWTHS OF THE PLEURA.

THE majority of new growths occurring in the pleura are of a secondary nature. We sometimes find single metastatic nodules of cancer in the pleura after primary carcinoma of other organs, especially of the mammary gland and the lungs, but most carcinomata of the pleura arise from primary carcinomata of the lungs owing to a direct invasion of the pleura by the new growth.

Of the primary new growths in the pleura, only one is of great importance—the endothelial carcinoma, first described by E. Wagner. This develops *de novo*, in a diffuse manner, from a proliferation of the endothelial cells of the lymphatics and the connective tissue. Metastases occur in the lungs, in the lymph-glands, in the liver, in the muscles, etc.

Single secondary nodules of cancer in the pleura cause no special clinical symptoms, but the cases of diffuse cancer of the pleura as a result of primary cancer of the lungs are important, inasmuch as the symptoms of disease of the pleura often quite predominate over the pulmonary disease. The dullness is intense, the respiratory murmur and the vocal fremitus diminished. In one such case we saw a proliferation of the cancer upon the ribs in front so that there was externally a very marked circumscribed swelling. The character of the sputum is the only thing that can give us definite information as to the origin of the new growth in the lungs (see the chapter on cancer of the lungs).

Primary endothelial carcinoma of the pleura runs a course similar to chronic pleurisy. As we sometimes find a co-existing fluid effusion in the pleural cavity, displacement of the neighboring organs may occur. The affection goes on for a long time without fever, or with slight and irregular elevations of temperature. Most cancers of the pleura are associated with severe pain.

The diagnosis of new growths in the pleura can usually be made only, if at all, in the more advanced stages of the disease. At first almost all the cases are regarded as simple or tubercular chronic pleurisy. The diagnosis is founded less upon the physical signs than upon the general course of the disease, the habit of the patient, and the evidence of some metastases in the glands and other organs. In some cases characteristic elements of the new growth can be found by the microscope in the cloudy fluid obtained by an exploratory puncture.

The prognosis is absolutely unfavorable, the treatment purely symptomatic. In endothelial carcinoma we might perhaps try arsenic internally.

CHAPTER VI.

MEDIASTINAL TUMORS.

In the anterior mediastinum, in quite rare cases, extensive new growths occur, which are of importance on account of their severe clinical symptoms. The point of origin for the tumor is either the mediastinal lymph-glands, or the connective tissue, or sometimes the remains of the thymus gland. In their anatomical character the tumors are almost always sarcomata, usually lympho-sarcoma, rarely alveolar sarcoma. They usually occur in youth or middle age, and are somewhat more frequent in men than in women. The special ætiological factors are unknown. In some cases an injury is stated to be the cause of their origin.

The clinical symptoms are usually of a very indefinite nature at first. The patient complains of general languor, headache, pain in the chest, and slight difficulty in breathing, and only gradually do severe subjective and objective symptoms develop in the chest.

The symptoms are in part directly due to the tumor, but in larger part they are symptoms of compression from the gradually increasing pressure of the tumor on a number of neighboring organs.

The pain in the chest, which is located chiefly in the sternal region, and is associated with a marked feeling of oppression, may be very severe. It sometimes shoots into the lateral portions of the chest and into the arms, showing pressure on the brachial plexus.

The dyspnœa may finally increase to an extreme degree. A patient with lympho-sarcoma under our observation could, in the last days of her life, breathe only while standing. The dyspnœa is due to a compression of the heart and lungs, and sometimes to actual stenosis of the trachea or a primary bronchus. Paralysis of the dilators of the glottis may also occur from a pressure paralysis of the recurrent nerves. Paralysis of one vocal cord has been repeatedly observed. In the case mentioned above a marked goître developed, as a result of vascular stasis, which further increased the dyspnœa by pressure on the trachea. A hydrothorax from local venous stasis may also aid in increasing the dyspnœa.

Pressure on the œsophagus, and disturbances of deglutition due to it, are rare. Pressure on the vagus nerve and the sympathetic sometimes causes anomalies in the rate of the pulse—either marked acceleration or slowing of the pulse.. If the sympathetic is involved there is inequality of the pupils. In some cases, by pressure on the tumor, an artificial dilatation of the pupil can be excited at will. By pressure on the vessels, especially on the superior vena cava, the subclavian vein, etc., œdema and cyanosis may arise in the corresponding parts of the body.

Objective examination of the chest gives a marked diffuse prominence of the sternal region in a part of the advanced cases; in other cases this swelling is absent. The discovery of an abnormal dullness in the anterior part of the chest is of diagnostic importance; this usually joins the cardiac dullness on the left, and on the right it extends a varying distance beyond the right border of the sternum. The heart is often pushed somewhat to the left. We heard over the pulmonary artery in our case a marked systolic murmur, caused by compression of the vessel. A dissimilarity of the pulse on the two sides is not infrequent.

The diagnosis of a mediastinal tumor is usually possible in cases with well-marked symptoms, but in other cases it is difficult and uncertain. The differential diagnosis between mediastinal tumors and aneurism of the aorta (*q. v.*) causes especially great difficulty. Tumors may also be confounded with abscesses in the anterior mediastinum.

The prognosis is in all cases absolutely unfavorable. The disease terminates fatally, sometimes after a duration of six months or a year.

The treatment can be only symptomatic. Internally we may try iodide of potassium or arsenic, and externally iodoform ointment. In the last stages of the disease we must try to alleviate the patient's great distress by narcotics.

CHAPTER VII.

ACTINOMYCOSIS OF THE THORACIC CAVITY.

Bollinger and others have described a peculiar tumor affecting the jaw-bones of cattle, and occasioned by the presence of a special form of fungus, known as the actinomyces or ray-fungus. More recently a class of diseases has been studied in human beings, occasioned by the same fungus (Ponfick, Israel, and others). These diseases may, as in cattle, affect the jaws, the floor of the mouth, and the neck; but in these cases they are mainly of surgical interest. The actinomycotic diseases of the internal organs, however, possess a great clinical importance; and, inasmuch as the lungs and pleura are the most frequently affected organs, it will be well to present briefly here the most important facts which have as yet been learned with regard to actinomycosis. The botanical position of actinomyces has not yet been definitively settled. Cohn and O. Israel regard it as a mold fungus. Boström, on the other hand, classes it with the algæ, and namely with the variety cladothrix. In its growth, the fungus forms small or moderate-sized gray or sulphur-yellow nodules which may be distinguished with the naked eye in the pus of the diseased tissue (see below), and which upon microscopic examination resolve themselves into a tangle of mycelium. It is an especial characteristic that many of these mycelia bear on their ends a club-shaped swelling. These are placed for the most part like radii on the periphery of a nodule, and so surround the entire mass like a circlet of rays (see Fig. 28). In nature actinomyces seems to appear especially upon plants, for example upon the beard of spikes of wheat. Thus is explained the frequency of infection in the plant-eating cattle, and a similar direct infection seems occasionally to be possible in man. It is worthy of note that the fungus seems to locate itself often in carious teeth. Thus apparently arises the above-mentioned disease in the buccal cavity; while on the other hand, the fungus may be carried further by inspiration into the respiratory tract, or by swallowing into the *primœ viœ*. Of course it may also be directly swallowed or inhaled.

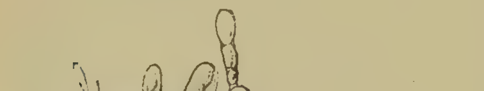

Fig. 28.—Masses of actinomyces, from Johne.

Wherever the fungus fastens itself in the body it occasions first a new growth of granulation-tissue, which has a tendency to break down into a whitish or brownish pasty mass. The brown color is occasioned by the hæmorrhage which frequently occurs. Very often actinomycosis goes on to suppuration; still, this probably depends upon the influence of pathogenic germs which have secondarily infected the part. Of especial importance is the tendency of the disease to extend from the lungs to the pleura and from the pleura to the peripleuritic connective tissue, and still further to the wall of the thorax. Thus arise not only extensive abscesses and wide-branching fistulous tracts, but also a very characteristic, extremely tough infiltration of interstitial tissue in the affected part. Not infrequently there is at last a perforation reaching the outer surface of the body.

The entire process, as a rule, is slow and insidious, but constantly progressive. The symptoms consist at first in slight thoracic discomfort, pain, cough, and expectoration. Physical examination will often detect changes in the lungs, but the correct interpretation of the signs found is of course at first difficult, if not impossible. The more the disease spreads the greater is the distress. Usually there is hectic fever, which may assume a pyæmic character if there is extensive suppuration. The patient gradually loses flesh, and in repeated instances amyloid degeneration of the liver, spleen, and kidneys has been observed. If a focus breaks into a pulmonary vein, the disease may be developed by metastasis in other internal viscera. Moreover, there may be a direct extension of the disease to the pericardium, or through the diaphragm into the peritoneal cavity.

The diagnosis of actinomycosis is at first difficult. It is established when the characteristic fungus is found in the sputum, but this has occurred in only a few cases as yet. If very extensive peripleuritic and pericostal suppuration has taken place, and the process has spontaneously broken outward or been laid open by surgical means, the demonstration above described in reference to the fungus is easy.

The treatment can only be symptomatic, unless the diseased spot can be reached by operation, and then the treatment becomes surgical. Permanent cure has so far been attained in but rare instances.

DISEASES OF THE CIRCULATORY ORGANS.

SECTION I.

Diseases of the Heart.

CHAPTER I.

ACUTE ENDOCARDITIS.

(*Endocarditis verrucosa. Endocarditis ulcerosa.*)

Ætiology.—Excitants of inflammation of different sorts, which circulate in the blood, may settle on the endocardium, especially on the valves of the heart, and there give rise to an acute endocarditis. Endocarditis, therefore, in its ætiological relations, is not to be regarded as a single disease; infectious agents of inflammation seem to be its chief, if not exclusive, cause. Lately, pathogenic micro-organisms have been injected into the blood (the streptococcus pyogenes, staphylococcus aureus, and others), and in this way an artificial endocarditis has been set up in animals. The experiments are more apt to succeed if the valves or the inner coat of the vessels have been subjected to some slight injury before the injection, thus promoting the settling of the germs upon them (Orth and Wyssokowitsch, Ribbert).

Of the infectious diseases to which man is liable, it is predominantly acute articular rheumatism in which acute endocarditis is a frequent and important complication. Endocarditis also occurs in certain diseases which are probably allied ætiologically to articular rheumatism, in certain forms of "hæmorrhagic disease," such as peliosis rheumatica, and in chorea. The appearance of endocarditis as a result of gonorrhœa or of gonorrhœal rheumatism is rare, but it has certainly occurred. In the course of the acute exanthemata, too, like scarlet fever and measles, as well as in acute and chronic nephritis, we sometimes notice the appearance of an acute endocarditis.

While endocarditis in the diseases previously named often takes a severe course, in many other infectious diseases—like typhoid, small-pox, and quite frequently chronic phthisis—slight inflammations of the endocardium are often found in the cadaver, which have an anatomical but not a clinical interest. These probably are not directly connected with the primary disease, but are a complication due to the absorption of septic material, the occurrence of this state of things being easily explained by the ulcerative processes of phthisis, the intestinal ulcers of typhoid, etc. We can probably explain in an analogous fashion the origin of the limited endocarditic aggregations which we sometimes find in persons who have died of ulcerative carcinomata, and similar diseases.

Acute endocarditis plays a very important *rôle* as a complication of severe septic and pyæmic diseases. Beyond a doubt the same pathogenic bacteria are here the cause both of the general sepsis and of the special acute endocarditis; but the latter at times stands so prominently in the central point of the picture that we may very well give its name to the whole disease, on the principle "*a potiori fit denominatio.*"

Finally, we have still to mention the important fact that acute endocarditis quite frequently develops on the soil of an already existing chronic endocarditis—the so-called acute recurring endocarditis. In women, pregnancy and the puerperal state sometimes seem to give the occasion for a recrudescence of the endocarditis; but possibly the old endocarditis merely furnishes a favorable soil for a new infection.

Pathological Anatomy.—We usually distinguish an *endocarditis verrucosa*, with the formation of large or small papillary nodules on the endocardium, and an *endocarditis ulcerosa* (*endocarditis diphtheritica*), with ulcerations as a result of the destruction and wasting away of the superficially necrosed tissue. The malignant, invariably fatal form of severe septic endocarditis is chiefly ulcerative endocarditis. Endocarditis verrucosa is the milder form, which is seen especially in acute rheumatism, but we can not draw either a sharp anatomical or a sharp clinical distinction between the two, since malignant cases of endocarditis verrucosa are also observed. It is at present still impossible to give a positive ætiological classification of the different forms of endocarditis.

The endocardial growths are usually situated on the valves, especially on their edges of closure. More rarely we find them on the chordæ tendineæ and on the endocardium of the ventricle and auricle. In the mildest cases they are scarcely as large as the head of a pin, but in severe cases they may increase to quite large warty and glandular masses. Microscopically, the base of the nodule consists of a newly formed vascular tissue, infiltrated with small cells, which on its surface changes to a granular, coagulated mass. This last is formed partly of coagulated albumen, dead cells, and fibrine deposited from the blood, and partly of micrococci. The micrococci are found without exception in all cases of ulcerative endocarditis—having been first discovered by Eberth. In the milder forms of endocarditis verrucosa micrococci have also been found by Eberth, Klebs, and others, but their invariable presence has not yet been established. The endocardial ulcers arise from the destruction of the superficially necrosed nodules. If the thin valve in any place yields to the blood-pressure, we have the so-called acute valvular aneurism. Complete perforation of a valve, and tearing off of fragments of a valve and of the chordæ tendineæ, are also seen.

The great majority of cases of acute endocarditis are confined to the valves of the left side of the heart—the mitral and aortic valves. Endocarditis on the tricuspid valve is seldom seen except as a secondary affection in old cases of heart disease. In a case of acute ulcerative endocarditis in a grown man seen by us, the process was confined exclusively to the tricuspid valve, and there were very many embolic abscesses in the lungs. This may be considered a great rarity. In contrast to the ordinary localization of endocarditis we find fœtal endocarditis most frequently in the right side of the heart.

Many other organs may be affected by the endocarditis, through embolism. In the benign endocarditis verrucosa the masses of fibrine deposited on the irregularities of the valve furnish the embolic material. They cause large or small infarctions in the kidneys and spleen, embolic softening of the brain, etc. In the malignant, ulcerative forms, however, large numbers of bacteria get into the circulation at the same time with the necrotic masses of tissue which have been torn off. Here, then, we have to do not merely with simple mechanical obstruction but with infectious emboli. The emboli in ulcerative endocarditis, therefore, either give rise to embolic abscesses in the cardiac muscles, the kidneys, the spleen, the lungs, the retina, etc., or they result in hæmorrhages, especially into the skin, but also into the kidneys, the brain, the retina, and the serous membranes. It is not yet known why in some cases abscesses are more frequent and in others hæmorrhages. The two, however, may be combined. In general, we may suppose that the development of abscesses is everywhere connected with the presence of bacteria, while hæmorrhages may also arise from toxic influences, for example, fibrine ferment; but changes in the vascular walls caused by bacteria might also give rise to hæmorrhages. Embolic abscesses belong almost exclusively to the severe form of septic endocarditis. Hæmorrhages are seen in this form, and also—without co-existing abscesses—in certain severe forms of endocarditis occurring in the course of acute rheumatism and allied diseases.

Clinical History.—Since acute endocarditis is not ætiologically a distinct disease, and since its clinical course is very different in different cases, it seems advisable to us to describe, in what follows, the most important varieties separately; but it must be expressly noted that the separate classes can by no means be sharply defined, and that there are many intermediate forms.

1. *Slight endocarditis verrucosa* is quite frequently found in the cadaver, without the slightest signs of any affection of the heart during life. The little papillary excrescences on the valves of the heart in phthisis, and carcinoma, whose ætiology has been explained above, are to be classed under this head.

2. The typical form of *benign acute endocarditis* is most frequent, clinically, in the course of acute articular rheumatism. It is much rarer in other infectious diseases (*vide supra*). In rare cases its appearance has been noticed as an apparently primary disease.

It is only rarely associated from the outset with subjective symptoms, like pain in the cardiac region, palpitation, and dyspnœa. It is usually first discovered on physical examination of the heart. The impulse of the heart in many cases is abnormally strong and diffuse, the pulse is accelerated, but strong, often somewhat jerky (*pulsus celer*), and usually regular, but sometimes a little irregular. Percussion at first shows no deviations from the limits of normal dullness. On auscultation, we hear at the apex, more rarely at the base, a loud blowing, systolic souffle. Diastolic murmurs are rare in acute endocarditis. The pulmonic second sound is often accentuated. But the physical signs in the heart are only slightly marked in many cases of acute endocarditis. This is understood if we remember that the occurrence of a heart-murmur depends wholly on the localization of the endocarditis, on the development of some valvular insufficiency, etc.

Besides the direct symptoms pointing to the cardiac affection, the onset of an acute endocarditis is often, but not always, associated with fever, or, if fever be already present, with an increase of it, and of the general disturbance. Embolic processes may occur in the brain, the spleen, the kidneys, and the extremities, but they are comparatively rare. Sometimes a pericarditis develops as a result of the endocarditis (*vide infra*).

It is hard to make any accurate statements as to the duration of this form of endocarditis. The physical signs may last for days, or for several weeks. Complete recovery is possible, but in the majority of cases this variety passes into chronic valvular disease of the heart.

3. *Malignant, non-septic form of acute endocarditis* ("rheumatoid endocarditis" of Litten). In many cases this form is perhaps only a quantitative increase of the preceding form, but in other cases it is probably distinct from it ætiologically. The severe general infection is usually quite prominent here, and the disease resembles in many particulars grave septic endocarditis. The objective signs in the heart are the same as in the preceding form, but more intense and extensive. The subjective symptoms in the heart, like palpitation and distress, may be quite pronounced, but they may also be almost wholly absent in this form. The general condition, however, is usually bad. There is sometimes high fever with

an irregular or intermitting course, but in many cases the fever is remarkably low in spite of quite severe constitutional symptoms.

The constitutional infection is very often manifested in these cases by the appearance of small or large hæmorrhages in the skin, sometimes in the mucous membranes, as in the conjunctiva and the soft palate, and rarely in the retina. Secondary articular swellings often develop; they are always of a serous character, and never purulent. Renal hæmorrhages and acute hæmorrhagic nephritis are quite frequent. Large emboli may also occur in the different organs in this as in every other form of endocarditis.

The duration of the disease extends over many weeks. In severe cases death ensues with a gradual aggravation of the general condition, and often with severe cerebral symptoms, like stupor and delirium. In milder cases, however, the patient may finally get well.

Regarding the occurrence of this form, we see it most frequently in acute articular rheumatism; also, in rare cases, in gonorrhœa, where it comes on some three or four weeks after the beginning of the urethral affection; also in nephritis, chorea, peliosis rheumatica, etc. The apparently primary cases of this sort usually belong to the recurrent form of acute endocarditis.

4. *The recurrent form of acute endocarditis* consists of an acute increase of the endocardial process, brought on by some exciting cause, in a patient already suffering from chronic endocarditis. The acute disease may show all the gradations from the mildest to the severest. The mild cases often run their course without any special symptoms. To this form we must probably often refer the more or less temporary elevations of temperature which we often see in patients with chronic valvular disease of the heart. In rarer cases the recurrent endocarditis comes on quite suddenly in the form of a severe acute attack. This sometimes seems to be clinically a primary, independent disease, especially if the previous chronic heart disease has up to that time caused no special symptoms. The patient has general malaise, headache, chills, and fever. The last may be quite high—104° (40° C.) and over—or moderate, varying between 100° and 102° (38°-39° C.), or it may be entirely absent. In many cases it is intermittent, when the return of fever is often associated with a chill. The symptoms in the heart may be quite pronounced, but in this form, too, they may be obscure and indefinite. In the further course of the disease we meet with cutaneous hæmorrhages, retinal hæmorrhages, articular swellings, large renal hæmorrhages, or typical hæmorrhagic nephritis—in short, just the same general type of disease as in the other malignant forms of acute endocarditis. The course is rarely rapid, and often lasts for weeks. Severe cases almost always end fatally.

5. The *severe septic ulcerative endocarditis* has already been described as a complication of a general septic disease. We therefore refer to the appropriate chapter (see p. 108) for all particulars. Septic endocarditis is probably entirely distinct ætiologically from the forms so far described, and is manifested by quite a rapid fatal course, with severe typhoid or pyæmic symptoms. It is characterized anatomically, apart from the cardiac affection, by the appearance of metastatic abscesses in the various organs, but in many cases, as we have said, abscesses and hæmorrhages are combined.

Diagnosis.—The diagnosis of an endocarditis, coming on secondarily in the course of articular rheumatism and other diseases, can be made only by a physical examination of the heart. We must therefore give constant attention to the condition of the heart in diseases which we know may give rise to endocarditis.

The diagnosis of the malignant form of endocarditis often causes great difficulty, especially if the patient is not seen until the later stages. It is confused with typhoid, meningitis, or acute miliary tuberculosis. Examination of the heart may furnish evident signs, but, as we have said, not invariably. Of the other symptoms the cutaneous and retinal hæmorrhages are of special diagnostic importance, since they are very much rarer in the other diseases just named. The acute hæmorrhagic nephritis, too, in connection with the other symptoms, is, at least to a certain degree, characteristic of malignant endocarditis. The course of the fever is of diagnostic value only when it is decidedly intermittent. A careful search for some ætiological factor is very important for diagnosis in all cases.

Prognosis.—In the description of the course of the disease we have already mentioned the prognosis of the different forms. The severe cases of acute endocarditis, some of which are complicated by the presence of an underlying affection, usually, and the cases of severe septic endocarditis always, end fatally. In mild cases recovery is possible, but the process of repair is often so incomplete that chronic valvular disease of the heart develops from the acute endocarditis.

Treatment.—The chief requisite in the treatment of every endocarditis is as complete rest as possible for the patient. If ice is well borne, the continuous application of an ice-bag to the cardiac region is of service. Digitalis may be indicated under some circumstances with a weak and irregular action of the heart, but on the whole this remedy is not often used in acute endocarditis. With severe local symptoms, like oppression and dyspnœa, we prescribe mustard plasters and small doses of morphine, and in some cases local blood-letting. Weakness of the heart is to be combated by stimulants—wine, camphor, and ether.

The treatment is also to be directed against the primary disease, although we can rarely succeed in influencing the endocarditis in this way. In articular rheumatism especially, which is the most frequent cause of acute endocarditis, salicylic acid is unfortunately almost wholly powerless against the endocarditis.

In the severe forms of endocarditis the treatment can be only purely symptomatic, and we try to keep up the patient's strength as much as possible. The exhibition of large doses of salicylic acid or quinine has usually no result, or only a temporary one. In many cases the use of arsenic, kept up for a long time, seems to us to be of service.

[For remarks upon the alkaline treatment of rheumatism, see page 909.]

CHAPTER II.

VALVULAR DISEASE OF THE HEART.

(Chronic Endocarditis.)

Ætiology.—A large number of cases of chronic valvular disease of the heart proceed from acute endocarditis. Hence the frequent statement in the history of heart disease that the patient has had articular rheumatism, once or many times. As a result of the endocarditis, which has its chief seat on the cardiac valves, there is considerable thickening of the valvular connective tissue. Processes of contraction also take place, and also adhesions, and finally in many cases quite marked calcification. All these processes have the necessary result, that such deformed valves can no longer fulfill their well-known physiological functions in regulating the circulation. There follows a disturbance in the circulation of the heart itself, and, as an immediate result of it, a disturbance of the general circulation, the pernicious effects of which must finally be manifested in the whole system.

In quite a large number of cases of heart disease, however, we can not obtain a history of acute endocarditis. We have to do here with an endocarditis which is chronic from the start, which also leads gradually to thickening, contraction, adhesion, and calcification of the valves. The ætiology of this chronic sclerotic endocarditis is still obscure in many of its relations. The same injurious influences which cause acute articular rheumatism, probably, act on the patient in a chronic manner from the beginning; at least, we not infrequently learn from patients with chronic heart disease, without previous acute articular rheumatism, that in former years they suffered repeatedly from slight rheumatic symptoms, to which they paid but little attention. In typical chronic arthritis deformans, too, heart disease occurs, though not very often. In other cases, however, we must consider the possibility of other injurious influences, partly infectious, and partly, perhaps, of a chemical or mechanical nature. Chronic alcoholism, and also constitutional syphilis, true gout, and immoderate muscular exertion, are the exciting causes which chiefly come to our notice. The chronic heart disease in such cases often develops at the same time with, and from the same causes as, general endarteritis, or atheroma of the vessels. To this we may ascribe the origin of many cases of heart disease in advanced age. The influence of chronic nephritis is not to be disputed in the development of chronic valvular disease. A hereditary predisposition to heart disease is not very frequent, but yet it can be made out with certainty in many cases. We have ourselves seen five members of the same family who have suffered from chronic heart disease, some from pure valvular disease and some from severe so-called idiopathic hypertrophy. Perhaps the very frequent occurrence of heart disease in many families is also connected with a special family predisposition to rheumatic affections, the occurrence of which predisposition can not, in our opinion, be denied. Finally, a small number of cases of heart disease, especially in the right side of the heart, depend upon anomalies of development of the heart—congenital heart disease.

Of 163 cases of undoubted chronic valvular disease which we have collected, 86 cases might with great probability be ascribed to articular rheumatism, while in 77 cases the patients had never suffered from rheumatic symptoms. In part of the last-named cases no definite cause could be ascertained, and in the rest perhaps some one of the factors mentioned above was to be found. A number of women referred their symptoms to previous pregnancies and parturitions. As has also been noted by others, the cases without previous articular rheumatism were more often aortic disease than mitral. Particularly in cases where syphilis was to be looked upon as a probable cause have we almost invariably found the chief changes in the aortic valves.

Valvular disease of the heart occurs at every age of life. The time of origin of most cases, corresponding in part to the occurrence of acute articular rheumatism, falls in youth and middle age, somewhere between eighteen and forty. In the female sex heart disease is somewhat more frequent than in the male.

General Pathology of Valvular Disease of the Heart.—Every valve of the heart, in order to fulfill its physiological task, must, on the one hand, open perfectly at the right time in order to furnish a free passage to the blood-current through the appropriate orifice, and must, on the other hand, close firmly and perfectly at the right time in order to make any abnormal backward flow of blood impossible. In both relations the function of the valves may be disturbed by chronic endocarditis, the disturbance being the result of their anatomical changes. If the tips of the valves are shortened on their free edges by contraction, or if the complete unfolding of the auriculo-ventricular valves is hindered by a shortening of their chordæ tendineæ, the closure of the valve can not be complete. At the moment when the closure of the valve is necessary a fissure remains open between its apices. We call this condition an insufficiency of the valve. On the other hand, the valves may lose their capability of free and sufficient separation from one another, as a result of thickening and calcification of the connective tissue, and also as a result of adhesions of the points of the valves with one another. At the moment when the blood-current should pass freely through the open orifice, the valve remains a stiff, narrow ring, through which the blood must force its way—stenosis of the orifice. The changes in the valves are often of such a sort that they cause at the same time both an insufficiency of the valve and a stenosis of the orifice. The thickening and calcification of the valves in stenosis cause, as a rule, a valvular insufficiency at the same time; but an insufficiency, set up by a contraction of the edges of the valves, may occur without a coincident stenosis of the orifice.

Every valvular disease affects the blood-current first in this way: that a stasis of the blood ensues, beginning at the diseased valve and extending backward against the current. The flow of blood through the pulmonary veins, and also through the veins of the body, is impeded, and the filling of the arterial system is thus diminished. To avoid repetition, we will describe more particularly, in the pathology of the disease of individual valves, the precise circumstances under which this disturbance of the circulation occurs. Every such abnormal distribution of the blood, and the necessary slowing of the circulation, from the increased tension in the venous system on the one hand, and the diminished tension in the aortic system on the other, would soon exert a most pernicious influence on the whole body, if a number of compensatory processes did not develop in the heart itself. We shall see how the disturbance of circulation in disease of each individual valve can be overcome by the increased work of certain definite portions of the heart, and how the heart does in fact respond to these increased demands put upon its working strength. It is one of the wisest contrivances in our organism, that the heart has control of a reserve fund of strength, which comes into action, if need be, in a way to compensate as far as possible for any disturbance of the circulation. This explains why a man with valvular disease of the heart may be almost perfectly well for a long time, while the increased work of certain portions of his heart is able to keep up an approximately normal circulation in spite of the existing valvular disease. We call a heart disease, in which there is at least no marked disturbance of circulation, a compensated heart disease.

The abnormally increased work, which single portions of the heart must perform in every case of disease in order to keep up the normal circulation, leads to hypertrophy of that portion, just as in any other muscle. This hypertrophy does not consist in an increase in thickness of the individual muscular fibers, but chiefly in an increase in number. The whole diameter of the cardiac muscle increases, and thus its capacity for work naturally becomes greater. It goes without saying that increased nutritive processes and a large supply of nourishment for the heart are necessary to bring about such a hypertrophy, by which alone a compensation of the heart disease is possible for any length of time. Hence we find the secondary hypertrophy of the heart absent, or at least only imperfectly developed, in weak people, especially in such as have suffered from some other chronic wasting disease besides the heart disease, like phthisis or carcinoma.

Although the compensatory processes in the heart can prevent for a long time any marked disturbance of the circulation, the already overburdened heart can no longer completely satisfy any additional demands upon it, even in a compensated heart disease. Hence patients with a compensated heart disease are free from subjective disturbance from their trouble only when they take complete bodily rest, while the signs of a disturbed circulation usually become quite apparent on slight physical exertion.

The hypertrophied cardiac muscle can seldom supply permanently the abnormally great drafts made upon its strength. There finally comes a condition of "fatigue," of "cardiac insufficiency." The cause lies either in the increase of the valvular disease, so that the hindrance to the blood-current caused by it can no longer be completely overcome, or in the fact that the nervous and muscular elements in the heart have their function gradually impaired by a disturbance of circulation in the heart itself. In short, in every heart disease the moment may finally come when the capacity of the heart has reached its limit, and hence the compensation of the heart disease ceases. The results of stasis now appear with increasing severity in the different organs, as we shall learn to recognize later on, and the patient finally succumbs to them, unless some intercurrent event puts an end to life.

After these general remarks, which will be understood better from what follows, we will pass on to the special description of the different forms of heart disease and their physical signs.

1. Insufficiency of the Mitral Valve.

Mitral insufficiency is one of the most frequent forms of heart disease. It develops in acute or chronic endocarditis of the mitral valve, from contraction of the free edges of the valve or from shortening of the chordæ tendineæ. In rare cases it comes on from partial adhesion of the valves with the walls of the ventricle.

The closure of the mitral valve occurs normally at each systole of the left ventricle. It prevents the return of blood from the left ventricle to the left auricle. If the mitral valve is insufficient and its closure is incomplete, at every systole of the left ventricle a part of the blood is thrown back from it into the left auricle through the open space of the ostium venosum. This abnormal backward wave encounters the blood-current coming in an opposite direction into the left auricle from the pulmonary veins. Since these two opposing currents rebound on each other, and since the backward wave of blood presses through the open space in the mitral orifice, decided vertiginous movements arise in the blood, which are the cause of a loud blowing, systolic murmur in the heart. We hear this murmur loudest at the apex of the heart, corresponding to the laws of conduction in the thorax; yet it usually is propagated so far that it may often be heard at the other cardiac orifices, although weaker. A loud systolic mitral murmur can also be heard sometimes in the back, on the left and occasionally on the right. Only in a few cases do we find the murmur louder nearer the base of the heart than at the apex, corresponding more to the anatomical position of the mitral valve. We often hear the first sound of the heart at the apex besides the murmur, but sometimes we do not. The second sound is often not to be heard at the apex, probably because it is obscured by the relatively protracted murmur.

Since the left auricle, at each systole of the ventricle, receives blood from two sides—its normal quantity from the pulmonary veins, and besides that the abnormal blood-wave from the left ventricle—it becomes much dilated. At the next diastole of the left ventricle the whole amount of blood collected in the auricle under increased pressure pours into the left ventricle through the mitral valve, which is now wide open (supposing a pure insufficiency of the valve without any stenosis). We see, then, that in pure mitral insufficiency the left ventricle must be overfilled during the diastole. The left ventricle must also expel in the following systole an abnormally large amount of blood. Although by this contraction only a part of the blood reaches the aorta in the direction of the normal blood-current while a part pours back into the auricle, the work of the left ventricle is of course excessive. This is the explanation, then, why, in pure mitral insufficiency, the left ventricle is dilated as a result of its increased filling in diastole, and is hypertrophied as a result of its increased labor. The general arterial tension thus remains approximately normal. It is not increased, since a part of the abnormal amount of blood, which pours out of the left ventricle at every systole, flows backward into the auricle. About the normal amount of blood reaches the aorta, and hence the radial pulse, in pure mitral insufficiency, remains of about normal strength and tension.

The anomalies in the movements of the blood in mitral insufficiency produce still other effects. We have already seen that the left auricle is dilated from its overfilling. It also becomes hypertrophied, so far as its weak muscular structure permits, but it is not in itself capable of compensating for the disturbance which the pulmonary circulation suffers from the mitral insufficiency, for the back current from the left ventricle, and the consequent high pressure in the left auricle, must plainly offer an abnormal hindrance to the flow of blood from the pulmonary veins. This stasis sets back through the pulmonary capillaries and arteries into the right ventricle. This may be recognized, on physical examination, by the change in the pulmonic second sound, which is louder, more valvular, and "accentuated," since the closure of the semilunar valves in the pulmonary artery now takes place under the abnormally high pressure which prevails in the arteries of the lungs. The right ventricle has the task of overcoming this abnormal stasis in the pulmonary circulation. It can overcome the abnormal resistance in the pulmonary circulation by increased work, and as a result it becomes hypertrophied. So long as the hypertrophy of the right ventricle suffices to maintain the normal pulmonary circulation, the stasis extends no farther backward, but in the later stages of heart disease we see the right ventricle becoming paralyzed, and more and more dilated as a result of stasis. The flow of venous blood from the body into the right auricle and ventricle is now rendered more difficult. The signs of venous stasis become manifest; the patient has a cyanotic hue, congestive œdema appears in the face and the extremities, symptoms of passive congestion of the liver, spleen, and kidneys appear, and, in short, there is developed the picture of an uncompensated heart disease.

If we now sum up the physical signs of mitral insufficiency, the different methods of investigation give the following results:

INSPECTION.—The cardiac region often seems rather prominent, as a result of the hypertrophy of the heart. This protrusion is most marked in young persons with a yielding thorax. The apex-beat is somewhat displaced toward the left as a result of the hypertrophy and dilatation of the left ventricle, and it is quite marked. Besides that, we often see and feel a diffuse pulsation in the whole cardiac region. In the epigastrium we sometimes see or feel an epigastric pulsation proceeding from the hypertrophied right ventricle. In cases which are no longer perfectly compensated the stasis in the veins of the body is rendered apparent by the general cyanotic appearance of the patient and the marked filling of the jugular veins in the neck. Undulatory or pulsating movements often occur in the latter (see tricuspid insufficiency, below).

PALPATION.—This confirms the abnormal strength of the apex-beat, and its displacement to the left. We often feel a systolic thrill at the apex of the heart —a "cat's purr"—by laying the hand flat on the chest. This whirl of blood, which is audible as a murmur, may be perceived as a fine tremor of the chest-wall.

The radial pulse is quite strong and usually regular. The sphygmographic tracing of it gives nothing characteristic in mitral insufficiency.

PERCUSSION.—This usually gives at first only a moderate increase of the heart's dullness to the left, and a little upward, but in the later stages there is at the same time an increase of the heart's dullness to the right, caused by hypertrophy and dilatation of the right ventricle. The whole area of cardiac dullness may finally extend two fingers' breadth beyond the right edge of the sternum, and to the left it may reach the mammillary line, or even pass beyond it.

AUSCULTATION.—At the apex of the heart we hear a loud, quite long, pure systolic blowing murmur, limited to the systole, either replacing the first sound or accompanying it. The second sound is often obscure or inaudible at the apex, but the pulmonic second sound is increased and accentuated. Auscultation of the vessels gives nothing characteristic.

2. Stenosis of the Mitral Orifice (Mitral Stenosis).

Mitral stenosis often develops in chronic endocarditis of the mitral valve, as a sequel to a previous insufficiency. The valve constantly grows stiffer and more rigid, and the signs of stenosis gradually predominate over those of insufficiency. Hence we very often find stenosis and insufficiency of the mitral valve combined, but often the signs of stenosis are so much more prominent that we can properly speak of a pure mitral stenosis.

The disturbance which the circulation suffers in mitral stenosis is much greater than in mitral insufficiency. In mitral stenosis the orifice may finally become so narrow that it scarcely admits an ordinary lead-pencil. The influx of blood into the left ventricle is accordingly much impeded. During the diastole of the left ventricle the blood must force its way through the stiff and narrow ring of the mitral valve. Thus irregular vertiginous movements develop in the blood, which, in the majority of cases, give rise to an audible diastolic murmur. In mitral stenosis the left ventricle receives a very small amount of blood. Hence, in mitral stenosis, the left ventricle is usually small, its cavity contracted, and the amount of blood thrown into the arteries with the systole is less than normal. In high degrees of mitral stenosis the radial pulse is weak and small, and, besides that, we often find it irregular (see Fig. 29). If we find a hypertrophy of the left ventricle in marked mitral stenosis, as sometimes happens, for which there is no other special explanation, it is probably always to be referred to a previous insufficiency of the mitral valve.

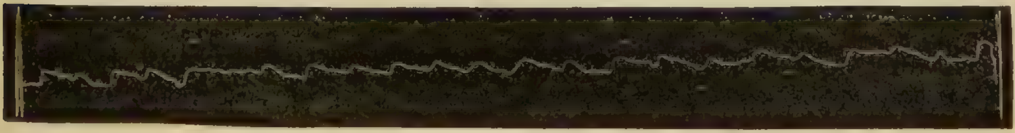

FIG. 29.—Pulse curve in marked mitral stenosis.

The hindrance to the flow into the left ventricle in mitral stenosis soon leads to a marked stasis, which extends to the right side of the heart through the left auricle, and the pulmonary veins, capillaries, and arteries. The left auricle is dilated first, and its walls are hypertrophied, but it can overcome only a very small part of the resistance at the mitral orifice. The right ventricle can, by more work, so increase the pressure in the pulmonary vessels that, in spite of the narrowed orifice, an approximately sufficient quantity of blood may pour into the left ventricle. Hence we find in mitral stenosis a very marked hypertrophy and dilatation of the right ventricle. The stasis in the pulmonary circulation, manifest objectively by the accentuation of the pulmonic second sound, has as a result a gradually developing ectasis of the pulmonary capillaries. Thickening of the intima of the pulmonary arteries and veins also usually develops. (See the chapter on brown induration of the lungs.)

The results of physical examination are as follows:

INSPECTION.—The whole cardiac region may seem slightly prominent, as a result of the hypertrophy of the heart. The heart's action is usually extended over a larger area, but in pure mitral stenosis the apex-beat is no stronger than usual, though often displaced to the left. We have frequently noticed a marked pulsation in the epigastrium, produced by the right side of the heart. The jugular veins are apt to be prominent, and show the different forms of undulatory and pulsating movement.

PALPATION.—This also gives signs corresponding to the more extended action of the heart. We sometimes feel the pulsation of the dilated right ventricle even to the right of the sternum. In some cases we feel a diastolic thrill at the apex of the heart, which alone may almost establish the diagnosis of mitral stenosis. This thrill arises from the same vertiginous currents in the blood which form the basis of the diastolic murmur (*vide infra*). The radial pulse is small in every severe mitral stenosis, and is very often irregular.

PERCUSSION.—Percussion gives especially an increase of the heart's dullness to the right, reaching to the right border of the sternum or beyond it. The dullness also often extends farther to the left than normal. This may have its origin in a co-existing hypertrophy of the left ventricle (*vide supra*), otherwise it depends on a dilatation of the right side of the heart so great that the left ventricle is pushed farther to the left and backward by it.

AUSCULTATION.—The characteristic auscultatory sign of mitral stenosis is the diastolic [pre-systolic] murmur at the apex. This is never so loud and blowing as the systolic murmur of insufficiency, but it usually sounds more rolling or rippling. It is loudest at the apex, and it is transmitted only slightly toward the base. Since, as has been said, the left ventricle in mitral stenosis is sometimes pushed to the left and backward by the very much enlarged right ventricle, in looking for the murmur we must often go far to the left, in order not to auscult the right ventricle only.

The origin of the murmur is easily explained. In the diastole of the left ventricle the blood-current must force its way through the narrow mitral orifice, whence vertiginous movements arise in the blood, and produce the murmur. Since the blood flowing through the narrow orifice has a current of relatively slight intensity, the murmur produced by it can not be very loud. Even in the highest degrees of mitral stenosis the murmur is often quite low. Not infrequently the murmur comes on in the second half of the diastole—namely, when, by the contraction of the left auricle, the blood-current is hurried through the narrow orifice. We call such a murmur, audible at the end of the diastole only, a presystolic murmur, since it usually passes immediately into the first sound.

It is by no means very rare that the murmur is absent in marked mitral stenosis. If such cases do not come under observation until the last stages of the disease, the mitral stenosis can readily escape recognition. We have ourselves often known the previously distinct diastolic or presystolic murmur in mitral stenosis gradually to disappear with the increase of the heart disease. In these cases the intensity of the blood-current through the narrow orifice becomes so slight that an audible murmur is no longer produced. The sounds that are heard arise probably from the right ventricle.

The first sound at the apex is retained in pure mitral stenosis, and often it is even remarkably loud and valvular, a condition which we usually try to explain by the theory that the difference (the "difference in tension") between the abnormally slight tension of the valve in diastole, in mitral stenosis, and its high tension in systole is relatively great. If insufficiency of the valve co-exists, we may hear a systolic murmur with the first sound or instead of it.

The very marked accentuation of the pulmonic second sound, the result of the abnormally high tension in the pulmonary artery, is almost constant. It fails only in very anæmic, weak people, or in co-existing insufficiency of the tricuspid valve (*vide infra*). The second sound at the base is very often "divided" or reduplicated. The closure of the semilunar valves in diastole does not happen at the same time in the pulmonary artery and in the aorta, on account of the unequal tension in the two vessels, so that consequently the two sounds are heard, one shortly after the other. Although this division of the second sound is particularly frequent in mitral stenosis, it is by no means a pathognomonic sign of it.

Mitral stenosis is one of the severest forms of heart disease. It almost always causes greater subjective disturbance than mitral insufficiency. Hypertrophy of the right ventricle can, indeed, maintain for a time an approximately complete compensation, but the signs of marked stasis in the pulmonary circulation, and further in the veins of the body, are apt to appear quite early. The dyspnœa becomes more marked, and dropsical symptoms gradually arise and cause a fatal termination.

3. Insufficiency of the Semilunar Valves of the Aorta.

Insufficiency of the aortic valves is most frequently due to contraction of the free edges of the valves. Tears, perforations, or adhesions of the valve to the wall of the vessel more rarely lead to insufficiency. The cause of all these changes is either an endocarditis affecting the valves, or a general atheroma of the arteries, which gradually invades the valves from the intima of the aorta.

The function of the aortic valves is to close tightly at the time of diastole of the left ventricle, in order to prevent any return of blood from the aorta into the ventricle. If these valves are insufficient—that is, if they do not close perfectly at each diastole—there is a return current of blood from the aorta into the left ventricle. The left ventricle then receives blood during its diastole from two sides: the normal flow from the left auricle, and the blood which comes back from the aorta. These two currents of blood, directed against each other, meet in the left ventricle during its diastole, give rise to a marked vertiginous movement of the blood there, and thus cause a characteristic diastolic murmur.

As a result of the excessive expansion of the left ventricle at every diastole, it finally becomes permanently dilated. Dilatation of the left ventricle therefore forms a constant anatomical lesion in every aortic insufficiency, and is shown not only in the dilatation of the whole ventricular cavity, but also in the very characteristic flattening of the trabeculæ and of the papillary muscles. The abnormally great filling of the ventricle during diastole, moreover, gives rise to increased labor; for the left ventricle must expel an abnormally large amount of blood at each systole, which is of course a sort of task of Sisyphus, since a part of the blood thrown out constantly rolls back into it. The increased labor must always lead at last to a hypertrophy of the left ventricle, which may attain the highest degree seen in any form of valvular disease.

From the facts enumerated we can easily understand the physical signs of insufficiency of the aortic valves.

INSPECTION.—Great hypertrophy of the left ventricle often causes a marked protrusion of the whole cardiac region. The very strong apex-beat, displaced downward and to the left, is especially striking. It may usually be seen in the sixth intercostal space, outside the left mammillary line, and sometimes even at the anterior axillary line. Besides that, we often see a marked diffuse tremor of the whole cardiac region.

PALPATION.—We can appreciate the heart's action to a still greater extent by palpation than by inspection. The apex-beat is very resistant, massive, and plainly heaving—that is, the finger or stethoscope applied to the apex is lifted by the beat at every systole. In rare cases a diastolic thrill, corresponding to the diastolic murmur, can be felt over the base of the heart. In two such cases observed by us the murmur had a marked musical character (*vide infra*). The appearances in the arteries are given below.

PERCUSSION.—Percussion gives an extension of the cardiac dullness to the left, beyond the left mammillary line and even to the anterior axillary line, caused by the hypertrophy and dilatation of the left ventricle. The upper boundary of the cardiac dullness is normal, or it may extend up to the third rib. The right boundary is in its normal place at the left border of the sternum, but it may also be pushed farther to the right, either because the large left ventricle of itself causes an extension of the whole heart to the right, or because the right ventricle is also hypertrophied. The latter change occurs in pure aortic insufficiency when the compensation is no longer complete, and the stasis extends backward from the left ventricle, through the pulmonary circulation, into the right side of the heart.

It may also be remarked here that, in insufficiency of the aortic valves, the ascending aorta is often considerably dilated by the marked impulse from the amount of blood pouring into it. A moderate degree of dullness is found over the dilated aorta, which may sometimes be made out at the sternal end of the second right intercostal space.

AUSCULTATION.—Insufficiency of the aortic valves is characterized by a long-drawn, loud, blowing diastolic murmur, the origin of which has been explained above. The place in which the murmur is heard loudest is not the sternal end of the second right intercostal space, the ordinary point for auscultation of the aorta, but it almost always lies farther to the left. Corresponding to the backward current of blood toward the left ventricle, which begets the murmur, we hear the latter loudest over the upper part of the sternum or even at its left border. In some cases the murmur assumes a marked "musical character"—that is, there is a definite high musical tone, which is due to a tendinous fiber arising from a wearing away of the valve, and set in vibration by the diastole, or to some similar cause. The diastolic murmur is often audible at the apex, but it is faint there. Only in rare cases, and especially when the aortic insufficiency results from acute endocarditis, is the diastolic murmur absent. During the systole there is usually a short systolic murmur heard over the aorta. This may, of course, depend upon a co-existent aortic stenosis, but it is also frequently present in cases of uncomplicated insufficiency of the valves. The explanation is, that at the time of the beginning of the contraction of the left ventricle the reflux of blood in the aorta has not wholly ceased, so that the outgoing blood-current has first to overcome this obstacle. It is easy to see why this should give rise to an audible systolic sound, and also why it should have some influence upon the development of a hypertrophy of the left ventricle (O. Rosenbach). It is very remarkable that, as Traube first pointed out, we often hear the first sound at the apex very obscure and dull, or else a short systolic murmur instead of it. This obscurity of the first sound at the apex is of theoretical interest, because it contradicts the hypothesis that the first mitral sound is a muscular sound. It would be especially inexplicable, on this theory, why the first sound is so obscure in insufficiency of the aortic valves, in spite of the hypertrophied and toiling left ventricle; but if we regard the first ventricular sound as originating in the mitral valve, then, according to Traube, its absence in aortic insufficiency may be explained by the fact that the mitral valve during the ventricular diastole is put in a certain degree of tension by the backward current of blood. The greater tension, which develops during the ventricular systole, does not suffice to produce a sound in the valves, since the origin of a valvular sound does not depend upon the absolute tension, but upon the amount of its sudden increase. The systolic murmur, often heard at the apex in aortic insufficiency, may depend upon a co-existing true mitral insufficiency, but it is probably often due to a relative insufficiency of the mitral, since the valves, which are normal in themselves, can no longer cause a perfect closure of the left mitral orifice now that the left ventricle is dilated.

Symptoms in the Peripheral Arteries.—Such remarkable symptoms are found in the peripheral arteries in aortic insufficiency that they demand a brief special description. The first striking symptom is the strong pulsation not only of the larger but also of the smaller arteries, even those the pulsation of which is not generally visible. We see and feel not only a strong pulsation in the carotids, but also in the tortuous brachial artery, in the radial, ulnar, temporal, dorsalis pedis, etc. We sometimes feel an arterial pulse in the liver through the abdominal walls.

The rapid decline of the pulse—the *pulsus celer* [Corrigan pulse]—is most characteristic of aortic insufficiency, and is to be felt especially in the radial artery, but also in the femoral, dorsalis pedis, and other vessels. An abnormally large quantity of blood is thrown into the arteries from the hypertrophied and dilated left ventricle; hence the high ascent of the pulse; but since at the next diastole of the ventricle the blood escapes in two directions, into the capillaries and back into the ventricle, an abnormally rapid and deep decline of the pulse follows the high ascent of its wave—a condition which explains the "jumping," "springing" pulse (*pulsus celer*) in aortic insufficiency. The quality of the pulse may be plainly recognized also in the sphygmographic tracing (see Fig. 30). The abnormal backward wave may even be detected in the capillaries. We often see a marked pallor of the finger-nails at every diastole of the heart in patients with aortic insufficiency—Quincke's capillary pulse.

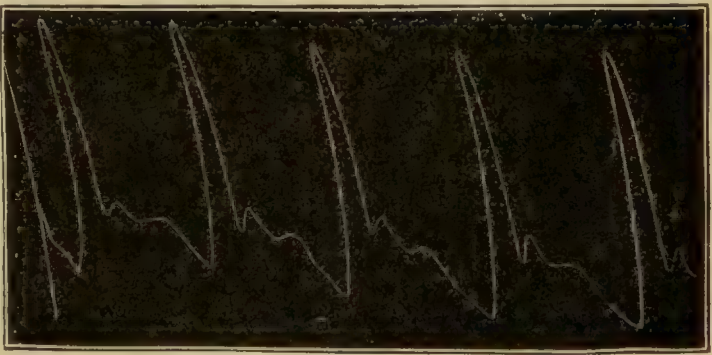

Fig. 30.—Pulse curve in aortic insufficiency.

The auscultatory phenomena over the arteries are partly connected with the changing conditions of tension of the arterial walls. We very often hear a short, rough, systolic murmur in the carotid. The second sound, which is well known to be the transmitted aortic second sound, is absent. Instead of it we sometimes hear faintly transmitted the aortic diastolic murmur. The sound of the medium-sized and smaller arteries is very characteristic. By applying the stethoscope lightly we hear over the femoral, the brachial, and often over the radial, the ulnar, the palmar arch, and the dorsalis pedis, a marked valvular sound, which is changed by pressure on the artery, especially in the larger arteries, to a loud stenotic murmur. The quicker the pulse, the more certain are we to hear these sounds in the arteries. The double sound in the femoral (Traube's double sound) is quite a frequent phenomenon, about the origin and significance of which there has been much discussion of late years. The double sounds either follow each other shortly, so that the first seems something like a preparatory blow for the second, or they are separated from each other by a longer interval, like the two sounds of the heart. Traube explained the origin of the first sound by the sudden tension of the vessel-wall, as in the simple femoral sound, and the second sound by the sudden relaxation of it. Friedreich has pointed out in regard to this that, in co-existing tricuspid insufficiency, a sound may also be produced in the femoral vein by tension of the venous valves. The double sound in the femoral may probably have different causes of origin. It is, of course, by far the most frequent in aortic insufficiency, but it has also been repeatedly observed in other forms of heart disease, as in mitral stenosis. The so-called Duroziez's double murmur in the femoral is more rare, and it is noticed almost exclusively in aortic insufficiency. This is when we hear, by pressing the stethoscope on the femoral, two murmurs plainly distinct from each other, of which the first comes from the passage of the systolic blood-wave, and the second from the passage of the abnormal backward wave coming from the periphery of the vascular system through the artificially contracted vessel.

Aortic insufficiency is in so far a comparatively favorable form of heart disease for the patient, that it may be almost perfectly compensated for years by hypertrophy of the left ventricle. Many patients with moderate aortic insufficiency feel perfectly well, and are even capable of quite hard work. They have not the slightly cyanotic hue which almost all patients with mitral disease have, but they have a normal or even a pale complexion. If, however, the signs of disturbed compensation once appear, the severest sequelæ may develop quite rapidly in aortic insufficiency. The left side of the heart can no longer satisfy the abnormally great demands upon it. Hence the stasis of the blood sets backward through the pulmonary circulation and into the veins of the body. The difficulty in breathing becomes marked, œdema appears, and the patient dies with the symptoms of general dropsy. We will speak more fully below of certain intercurrent events in aortic insufficiency, such as cerebral hæmorrhage and pericarditis.

4. Stenosis of the Aortic Orifice.

Except for the mild forms of aortic stenosis, which often come on with aortic insufficiency, aortic stenosis is a rare disease. It arises from marked thickenings and calcifications, and especially from adhesions of the aortic valves to one another. The stenosis may become so considerable that the orifice is finally reduced to a mere fissure, through which the left ventricle must force the blood at its systole. The vertiginous movements thus arising in the blood produce a loud systolic murmur. The left ventricle is compelled to do greater work in consequence of the increased resistance of the aortic orifice, and hence becomes hypertrophied. Since it demands more time to drive its contents through the narrow orifice than it does under normal conditions, we often find in aortic stenosis a marked slowing of the pulse, but the pulse, as we can conceive, is small, and the artery feels contracted and hard.

Inspection and Palpation.—On physical examination of the heart we find the apex-beat displaced outward, corresponding to the hypertrophy of the left ventricle; but nevertheless it is by no means very strong; it is even sometimes remarkably weak, which condition is explained in part by the slower contraction of the ventricle, and in part by the absence of the backward impulse (compare the Gutbrod-Skoda theory of the heart-beat).

Percussion.—Percussion gives an extension of the heart's dullness to the left. The right ventricle is also dilated and hypertrophied to a moderate degree in the later stages, if the stasis extends backward through the pulmonary circulation.

Auscultation.—On auscultation, we hear over the aorta a very loud "sawing," long-drawn, systolic murmur, which is usually transmitted to the right, corresponding to the course of the aorta, in distinction from the diastolic murmur of aortic insufficiency. It is usually to be heard loudest at the sternal end of the second right intercostal space, but it is audible to a lesser extent over almost the whole heart. It is usually quite loud over the carotids. The aortic second sound is feeble, or not to be heard at all. With co-existing insufficiency of the valve it is replaced by a diastolic murmur.

The character of the pulse has already been mentioned. The pulse is small, and is sometimes in noticeable contrast with the strength of the apex-beat; in compensated cases it is regular and often slow, to a slight or sometimes to a great degree. The sphygmographic tracing (see Fig. 31) gives a low wave, and a comparatively slow rise and fall of the curve.

An aortic stenosis of a mild degree may be quite well borne by the patient for a long time. In stenosis of a higher degree we sometimes notice peculiar symptoms, which probably must be referred to anæmia of the brain, especially attacks of dizziness and faintness. Epileptic attacks have even been observed. In other respects the same disturbances of compensation finally appear, as in all other forms of valvular disease. The course of the disease is more unfavorable than that of aortic insufficiency, but, on the other hand, it is more favorable than that of mitral stenosis.

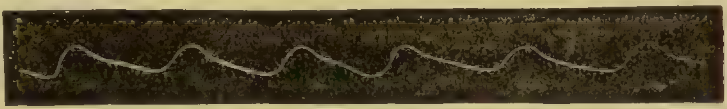

FIG. 31.—Pulse curve in stenosis of the aortic orifice.

5. Insufficiency of the Tricuspid Valve.

Insufficiency of the tricuspid valve is extremely rare as an independent disease of the heart, but a secondary insufficiency of the tricuspid is quite frequent, and is therefore of practical interest, as it complicates other already-existing valvular diseases in the left side of the heart. It arises either from a secondary endocarditis affecting the tricuspid, in quite an analogous manner with mitral insufficiency, or it is a so-called relative insufficiency. This name we give to that form of insufficiency which develops when the edges of the tricuspid valve, normal in themselves, at last fail to meet one another, from the increasing dilatation of the right ventricle.

The necessary result of tricuspid insufficiency is, that in every systole of the right ventricle a backward current passes through the open tricuspid orifice into the right auricle, and thence into the veins of the body. The tricuspid insufficiency ensuing in other forms of heart disease must therefore increase the stasis in the veins of the body, and is thus far an unfavorable complication. It has a compensatory significance only as it affords relief to the pulmonary circulation. Since a part of the blood passes back from the right ventricle into the veins, less blood than usual must reach the pulmonary arteries. The decrease in tension thus produced in these arteries makes itself apparent on auscultation, since the accentuation of the pulmonic second sound in valvular disease of the mitral orifice diminishes when tricuspid insufficiency takes place.

That tricuspid insufficiency must result in a hypertrophy of the right ventricle is explained in just the same way as the hypertrophy of the left ventricle in mitral insufficiency, from the increased influx of blood at increased tension into the right ventricle during diastole; but this effect of tricuspid insufficiency can rarely be made out in any individual case, since the right ventricle is usually already hypertrophied as a result of the disease in the left side of the heart.

The most important symptom from which we can diagnosticate tricuspid insufficiency is the venous pulse. The cause of this is the backward wave of blood produced at each systole of the right ventricle. So long as the venous valve above the bulbus jugularis is closed, we usually see only a "bulbar pulse," but very soon this valve also yields to the continued impulse of the blood, and then a strong, purely venous pulse is visible along the whole course of the jugular vein up to the vicinity of the mastoid process. The contraction of the right auricle very often causes a decidedly weaker elevation of the vein, which just precedes the marked pulsation caused by the ventricular systole (anadicrotic venous pulse). On account of the straighter course of the right innominate vein, the jugular venous pulse is often stronger on the right side than on the left. We must state, however, that the jugular venous pulse is not an absolutely certain sign of tricuspid insufficiency, since it may arise in hypertrophy of the right side of the heart without any insufficiency of the tricuspid, from the closure of the valves.

If there is pulsation in the bulb of the jugular vein and the jugular valve is still capable of closing, a low, audible, venous, valvular sound may be produced by its closure. A sound may also arise in tricuspid insufficiency, as has been already said, from the tension of the valves in the femoral vein. A visible pulsation in the larger veins of the extremities is very rare, but in tricuspid insufficiency we quite frequently feel a venous pulsation of the liver. This may be quite apparent even in many cases where the jugular venous pulse is absent, because the veins in the liver are without valves.

Auscultation over the right side of the heart gives a systolic murmur in insufficiency of the tricuspid, arising from the regurgitating blood-current. This may be heard loudest over the lower part of the sternum, or at the sternal end of the right fifth rib. The significance of this murmur in diagnosis, however, is impaired by the fact that it can not always be separated from the systolic mitral murmur that often co-exists.

6. Stenosis of the Tricuspid Orifice.

Stenosis of the tricuspid orifice is an uncommonly rare disease, and hence it is without practical significance. It has usually been observed, up to the present time, as a congenital form of heart disease, almost always combined with other anomalies of development in the heart.

The physical signs of tricuspid stenosis can easily be constructed theoretically. The first result must be a marked dilatation of the right auricle, and the occurrence of a diastolic or presystolic murmur over the right side of the heart. From the rarity and complex character of the cases, however, we have so far seldom had an opportunity to confirm these theories at the bedside.

The prognosis of this form of heart disease is very unfavorable, since a long-continued compensation by increased labor on the part of the right auricle is scarcely conceivable.

[Seventy cases of tricuspid stenosis have been collected by Bedford Fenwick, whose analysis affords good grounds for thinking that the lesion is often acquired. In fifty per cent. of the cases there was a clear history of rheumatism, and nearly all of the patients were more than twenty years of age at the time of death.

This lesion is never found alone, but invariably combined with mitral stenosis; all but eight of the cases were in women. Fenwick thinks that the influence of sex lies in the less onerous nature of the work of women than of men, the granulating edges of the valves being kept more in apposition, thus healing with adhesion and causing obstructions at the orifice.]

7. Insufficiency of the Pulmonary Valve.

Insufficiency of the pulmonary valve is also a very rare form of heart disease. It occurs as a congenital anomaly, often combined with other failures of development, or as a disease acquired after birth. The anatomical changes in the valve, which lead to insufficiency, are precisely analogous to those which cause insufficiency of the aortic valve.

The physical signs of this form of valvular disease consist chiefly of a marked dilatation and hypertrophy of the right ventricle, to be made out by percussion, and of a loud diastolic murmur over the pulmonary valve. These signs are explained in just the same way as the precisely analogous signs in the left ventricle in aortic insufficiency.

In general, pulmonary insufficiency, like aortic insufficiency, seems to be compensated quite well for a long time by hypertrophy of the right ventricle. In many cases a co-existing patency of the foramen ovale also seems to be of favorable influence, so far as it lessens the stasis in the right auricle and the veins of the body, while it renders easier the filling of the left ventricle.

8. Stenosis of the Pulmonary Orifice (Pulmonary Stenosis) and the other Congenital Diseases of the Heart.

1. **Congenital Pulmonary Stenosis.**—While the stenosis of the pulmonary orifice acquired in later life is so rare that it has only a slight practical significance, the congenital pulmonary stenosis is of far greater importance. It is, on the whole, the most frequent of the congenital forms of heart disease. Its origin is to be referred either to an endocarditis of the pulmonary valves during fœtal life, or to anomalies in the development of the heart. The stenosis is often situated not merely at the pulmonary orifice itself, but farther back in the conus arteriosus, which seems to be narrowed by the formation of myocardial cicatrices. The pulmonary artery is often also narrowed as a whole. In the majority of cases we find, in addition, other anomalies of development in the heart, especially patency of the foramen ovale, great defects in the ventricular septum, and, in about half the cases, patency of the ductus Botalli, etc.

The symptoms of congenital pulmonary stenosis sometimes appear soon after the birth of the child. The first thing that strikes us is the marked cyanosis, which is constant, or else comes on with crying, or with movements of the body. Many children, however, reach a fair age, five or ten years, but rarely more. In some cases the heart disease may be so perfectly compensated that the child may be comparatively well for a time, and severe disturbances may not appear for several years.

As a rule, children with congenital pulmonary stenosis present a very striking appearance. The cyanosis is especially noticeable in the face, the lips, the nose, and the hands and nails. The parts mentioned feel cool. The eyes are often somewhat prominent, and there is a slight œdematous swelling about them. The peculiar club-like thickening of the terminal phalanges of the fingers and toes, a result of stasis, as seen also in many cases of bronchiectasis, is very characteristic. The nails also present a characteristic claw-like curvature.

The whole development of the child is remarkably retarded. The muscles and fatty layer are slight. The gums are sometimes very spongy and disposed to bleed. In severe cases the child suffers from faintness, vertigo, etc.

On physical examination of the heart, we usually find the cardiac region rather prominent. Percussion gives an increase of the heart's dullness, especially toward the right. This extension of the dullness is due to the hypertrophy of the right ventricle, which must arise in the same way as hypertrophy of the left ventricle in aortic stenosis. On auscultation, we hear a loud systolic murmur, which is perceptible over the whole heart, but which has its greatest intensity at the sternal end of the second left intercostal space. The eddies of blood, which produce the murmur, may also often be felt by the hand as a systolic thrill. In some cases, however, we miss the murmur in pulmonary stenosis, just as in mitral stenosis. The pulmonic second sound is weak or inaudible, or it is replaced by a murmur if there is also insufficiency of the valve.

The course of congenital pulmonary stenosis is always unfavorable. As has been stated above, only a few children get beyond the age of fifteen years. Death ensues, either with general disturbances of compensation evidenced by dyspnœa and dropsy, as in every other form of heart disease, or from complications. Among the latter, we may mention especially the very frequent development of phthisis.

2. **The Remaining Congenital Lesions of the Heart.**—Inasmuch as other congenital lesions than pulmonary stenosis have but slight clinical importance, we will limit ourselves here to a brief review of the same.*

(*a*) Patency of the foramen ovale is a comparatively frequent lesion, whether alone or combined with others. Physical signs are mostly absent. In a few cases a presystolic murmur has been heard. When mitral insufficiency co-exists with a patent foramen ovale, venous pulsation may be caused.

(*b*) Defects in the septum between the ventricles. These are most frequently situated in the posterior section of the anterior septum, and they likewise are associated with other anomalies, such as abnormal distribution of the arteries, pulmonary stenosis, or defects in the septum between the auricles. Sometimes the patency of the septum gives rise to a systolic murmur, but a diagnosis of the condition during life is scarcely ever possible.

(*c*) Persistence of the ductus Botalli. Inasmuch as this contributes blood from the aorta to the pulmonary circulation the pressure in the latter is raised, hence there is to be observed accentuation of the second pulmonic sound and hypertrophy of the right ventricle. There is sometimes also a loud systolic murmur.

(*d*) We have already spoken of congenital stenosis of the tricuspid valve. Congenital narrowing of the mitral valve and of the aortic valves may also occur, but they are extremely rare.

9. Combined Valvular Diseases of the Heart.

Although in what has preceded we have dealt with the several forms of valvular disease of the heart separately, in order to present them in a general way, yet in reality combinations of them often occur in great variety. We find especially, as has already been mentioned, stenosis of an orifice co-existing with insufficiency of the accompanying valve; but diseases of two or more different valves are not infrequent, combined in the most diverse manners. The physical signs of these "combined forms of heart disease" may, of course, be inferred from the signs of anomalies of single valves, but the phenomena are often so complicated that the diagnosis of combined heart disease is generally much harder than that of the simple forms. Sometimes the single forms neutralize one another in their action. For example, the left ventricle is usually small in pure mitral stenosis, but, if aortic insufficiency be also present, it is nevertheless found dilated, at least to a certain degree. The influence of an absolute or relative tricuspid insufficiency on the action of mitral disease, especially the decrease in tension in the pulmonary vessels caused by it, and also the diminished accentuation of the pulmonic sound, have been mentioned above.

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* A more extensive presentation of the subject can be found in Gerhardt's "Handbuch der Kinderkrankheiten," Band iv, written by Rauchfuss, and in text-books on pathological anatomy—for example, Orth's.

In reference to the clinical symptoms of combined heart disease we may say, on the whole, that, in a large number of cases, the disease of *one* valve stands out as predominant in the whole picture of the disease. The other anomalies are only slightly noticeable, and often of later date. Hence, we may find at the autopsies of patients, who during life have shown the symptoms of disease of only *one* particular valve, unimportant changes, like fresh endocarditis, on the other valves, which have been without clinical significance.

General Comparison of the most Important Physical Signs in Valvular Disease of the Heart.

Form of Heart Disease.	Inspection.	Palpation.	Percussion.	Auscultation.
1. *Mitral insufficiency.*	Strong apex-beat, often somewhat displaced outward.	Systolic thrill at the apex. Quite strong radial pulse.	Hypertrophy of the left, later of the right ventricle.	Loud systolic murmur at the apex. Pulmonic second sound accentuated.
2. *Mitral stenosis.*	Area of cardiac impulse enlarged, epigastric pulsation.	Diastolic thrill at the apex. Small and often irregular pulse.	Hypertrophy of the right ventricle.	Diastolic or presystolic murmur at the apex. First sound sometimes loud. Pulmonic second sound accentuated, and sometimes double.
3. *Aortic insufficiency.*	Apex-beat very strong, displaced downward and to the left. Visible pulsation of the medium-sized and smaller arteries.	Very strong, heaving apex-beat. *Pulsus celer.*	Marked hypertrophy of the left ventricle.	Loud diastolic aortic murmur, especially over the upper part of the sternum. Sounds in the arteries (femoral and brachial sounds, etc.). Sometimes a double sound or double murmur in the femoral.
4. *Aortic stenosis.*	Apex-beat displaced to the left.	Heart's action not very strong. Pulse small, sometimes slow.	Hypertrophy of the left ventricle.	Loud systolic aortic murmur, transmitted to the right.

[Bramwell reports that of 131 cases with macroscopic valvular lesion, the tricuspid was implicated in 33·58 per cent.; in all but 12 per cent. of these the changes were recent. Hence he thinks that tricuspid endocarditis is generally recovered from, and this he attributes to the relatively small strain to which that valve is subjected. The obvious therapeutic deduction is the importance of rest in mitral endocarditis.]

General Sequelæ and Complications of Valvular Disease of the Heart.

After having discussed, in what precedes, the mechanism of the single forms of valvular disease, and the physical signs derived from it, we must now describe a number of symptoms and sequelæ which may be present to a greater or less degree in all forms of valvular disease. With them we must also mention certain peculiarities of the individual forms.

1. **Subjective Symptoms.**—Fully compensated heart disease may exist, at least for a long time, without any subjective symptoms. This is especially the case in aortic insufficiency, more rarely in mitral insufficiency. Stenosis of the mitral or of the aortic valves almost always causes subjective symptoms. These symptoms often do not exist so long as the patient keeps perfectly quiet physically and mentally, but they develop under appropriate circumstances.

The existing subjective symptoms in heart disease are by no means always referred, in the first place, to the heart itself. It sometimes happens that the patient comes to the physician complaining of various digestive disturbances, or in other cases of headache, vertigo, etc. The physical examination alone leads us to recognize the heart disease. As a rule, the patient's first and chief complaint is directed toward his difficulty in breathing. The shortness of breath, which increases on any physical exertion, comes on quite early in many cases. In the later stages it is almost always the most distressing symptom. It arises as a result of the overfilling of the pulmonary vessels with blood, and the consequent delay in the pulmonary circulation, and impairment of the transfer of gases in the lung. In the later stages the anatomical changes in the lung tend to increase the dyspnœa. Until lately it was usually assumed that the distended capillaries encroached upon the lumen of the alveoli, and thus diminished their capacity for air. Basch, however, claims that the alveoli become distended by the passive congestion in the pulmonary circulation, because the capillaries are stretched (swelling of the lungs). One element in the development of the dyspnœa is the "pulmonary rigidity." The diminished expansibility of the lungs thus occasioned offers the same hindrance to respiration as is seen in pulmonary emphysema. Further investigations are necessary in order to determine the clinical importance of these conditions.

The secondary bronchitis of heart disease is a very great factor in the dyspnœa. This bronchitis is a frequent result of the pulmonary stasis. Often the respiratory distress increases and decreases simultaneously with corresponding variations in the bronchitis. A purely mechanical cause of dyspnœa is the compression of the lower portion of the left lung by great cardiac hypertrophy. The highest grade of dyspnœa is reached when finally hydrothorax and pulmonary œdema are developed. From what has already been stated, it is easy to see why lesions of the mitral valve which directly impair the pulmonary circulation lead sooner to shortness of breath than do lesions of the aortic valves. Sometimes the dyspnœa in heart disease occurs in paroxysms (cardiac asthma). This is probably in most cases dependent upon a sudden cardiac failure, especially involving the left ventricle.

Palpitation is the first subjective symptom to be mentioned of those which are referred directly to the heart. It is not yet accurately determined under what circumstances the action of the heart is perceived by the patient himself. We sometimes see an uncommonly strong action of the heart, as in aortic insufficiency, which is not perceived at all subjectively. In other cases, where objectively the heart is not especially active, palpitation forms the patient's chief complaint. It usually first appears when the heart disease ceases to be fully compensated. It is increased or first excited by physical exertion or mental excitement. In many patients attacks of palpitation occur without any discoverable external cause, due apparently to nervous disturbance. They are sometimes associated with a striking acceleration of the pulse, the so-called tachycardia.

Pain in the cardiac region is only rarely present in heart disease. The patients more frequently complain of an indefinite feeling of pressure and oppression in the chest, but attacks of severe pain in the cardiac region do occur, especially in patients with aortic insufficiency—pain shooting into the shoulders and arms, the so-called *angina pectoris, vide infra.* Pains in the epigastrium and abdomen, which sometimes form the chief annoyance of the patient, usually depend upon passive congestion of the liver (*vide infra*), or upon the tension of the abdominal walls from ascites, œdema, etc.

We must finally mention here the rheumatoid pains in the joints and muscles, from which many patients with heart disease suffer.

The greatest subjective distress occurs in the last stages of heart disease, when general dropsy develops. The patient's helplessness usually culminates. All motions of the body are difficult, the dyspnœa and oppression in the chest constantly increase, until death finally releases the patient from his mournful condition.

2. **Sequelæ in the Heart Itself.**—We have already discussed the most important sequelæ of valvular disease in the heart itself, its hypertrophies and dilatations. It remains for us to describe the influence of the cardiac disease on the frequency and regularity of the heart's action, and also to discuss some secondary diseases of the cardiac muscle and of the pericardium.

In every well-compensated heart disease the heart's action may for a long time be of approximately normal frequency and regularity. We often find a constant and moderate acceleration of the pulse, which is easily increased from temporary causes. Permanent slowing of the pulse is rare in valvular disease of the heart. It is most frequent in aortic stenosis, where it is in part of compensatory significance. Marked changes in the frequency of the pulse depend upon severe disturbances of the nervous apparatus in the heart. Hence they are, as a rule, associated with irregular action of the heart. The frequency of the pulse may then reach 120 or 140 a minute. Diminution of the frequency to 50 or 30 are much rarer. We may mention as an unusual but interesting symptom the sudden attacks of enormous acceleration of the pulse to 200 or more—tachycardia—which seem to be especially frequent in mitral disease. In the interval there is usually a quiet action of the heart and a complete compensation of the heart disease. The increase in the pulse comes on quite suddenly, and is frequently associated with subjective feelings of palpitation and distress. It may last for several hours and then disappear again, also quite suddenly. The precise cause of these attacks is unknown. We may probably regard it as a temporary paralysis of the inhibitory apparatus of the heart.

Arhythmia of the heart is of still greater importance than anomalies of the pulse-frequency. It always points to a severe disturbance of the nervous apparatus of the heart. The general circulatory disturbance which follows every valvular disease must of course make itself felt in the heart itself, and the nerves and ganglia of the heart can not remain undisturbed by it. Hence we generally see marked variations in the frequency and rhythm of the heart's action along with the other signs of beginning disturbance of compensation; but daily clinical experience teaches us that there is not a perfect parallelism between the two symptoms. We find often enough in heart disease quite a considerable irregularity of the pulse without any of the other signs of marked disturbance of compensation, and, on the other hand, we see in many patients an almost perfect regularity of the pulse up to death. In mitral disease, especially in mitral stenosis, arhythmia of the heart is much more frequent than in aortic disease.

We can not here discuss in detail the different forms and symptoms of cardiac irregularity. An inequality in the intensity of the single beats is very often associated with the irregularity: the irregular pulse is also an unequal pulse. The weaker heart-beats sometimes cause a pulse which is no longer perceptible in the radial artery, so that we can determine the true frequency of the heart-beat, not by counting the radial pulse, but only by auscultation of the heart. The occurrence of the so-called *pulsus bigeminus* is of interest (see Fig. 32). A second weaker contraction follows the first strong systole, even before the ventricular diastole has fully ended, and then comes a longer pause. We feel alternately a strong and quite weak pulse. The latter may be imperceptible, so that it can be made out only by the sphygmograph. In such cases, with co-existing tricuspid insufficiency, we sometimes find the number of the venous pulsations twice as great as the number of the radial pulsations, because the second weaker contraction of the heart produces a visible venous pulse, but not an appreciable radial pulse. On the whole, the *pulsus bigeminus* is a bad sign, since it always points to a marked disturbance of the cardiac innervation; but it may also pass away again and give place to a regular action of the heart.

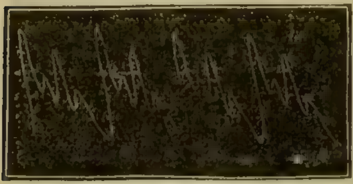

FIG. 32.—Pulsus bigeminus.

Chronic valvular disease of the heart is often combined with anatomical changes in the cardiac muscle, and sometimes in the pericardium.

Among the changes in the cardiac muscle, cloudy swelling, and especially fatty degeneration of the muscular fibers, are the most frequent. The fatty degeneration of the muscle occurs either in a diffuse form, or in little yellowish spots, which are plainly visible on the papillary muscles and trabeculæ. The opinion has often been expressed that fatty degeneration of the muscles is the cause of the disturbance of compensation; that the cardiac muscle performs its increased work until fatty degeneration ensues and reduces its strength. This theory does not entirely correspond to the facts. We have often seen the greatest disturbance of compensation in valvular disease when section of the cardiac muscle showed no fatty degeneration; and, on the other hand, we have seen great fatty degeneration of the heart, as in pernicious anæmia, when there were no signs of cardiac weakness during life. Anatomically, with our present aids to research, we can hardly ever decide with certainty whether the cardiac muscle was still capable of performing its normal functions or not. The usual state of the case is probably this, that fatty degeneration of the cardiac muscle is a result of the disturbance of compensation, and especially of the deficient supply of arterial oxygenated blood to the muscle (see the chapter on anæmia).

A frequent affection of the cardiac muscle in valvular disease is the presence of cicatricial changes and so-called myocarditic nodules in the substance of the heart. Chronic endocarditis may directly invade the subjacent parts of the cardiac muscles and set up a chronic inflammation there, but the cardiac cicatrices usually have another origin. The connective-tissue thickening beneath the endocardium is the result of a simple atrophy of the superficial muscular fibers due to the increased internal pressure of the blood, as in mitral or aortic insufficiency. The connective-tissue nodules within the cardiac muscle, however, depend upon a deficiency in the local supply of arterial blood. Simple sclerotic thickening of the coronary arteries, or complete embolism, or thrombosis of a small branch of one of them, is the evident cause of these circumscribed cicatrices. It is certain that the latter may reduce the capacity of the cardiac muscle for work, but, on the other hand, we often find cicatrices of myocarditis without any signs of a previous disturbance in the compensation of the heart. A fuller discussion of this subject will be found in the next chapter.

Pericarditis is not very rare as a result of chronic valvular disease. It is always a dangerous complication, and may cause death. Regarding its origin, we have found that almost all the cases of heart disease complicated with pericarditis show changes in the aortic valves. Hence it does not seem improbable to us that the origin of the secondary pericarditis in such cases is due to a direct invasion of the pericardium by the excitants of inflammation passing from the aortic valves through the walls of the blood-vessel.

3. **Symptoms of Stasis in the Different Organs of the Body.**—As has frequently been mentioned in what precedes, the results of stasis of the blood make themselves manifest in heart disease in various organs. We have already spoken of the important results of blood stasis in the lungs. It remains for us to discuss the symptoms of stasis in the systemic veins.

As soon as the flow of venous blood into the right side of the heart is hindered, the venous stasis is shown by the cyanotic appearance of the patient. This cyanosis may exhibit any degree. In heart disease which is, on the whole, still well compensated, it is recognized only by the practiced eye of the physician as a slight bluish tinge of the lips, the alæ of the nose, the cheeks, or the nails. With the increase of the disturbance of compensation the cyanosis increases, if it be not modified by the co-existence of general anæmia. In mitral disease, especially in mitral stenosis, the cyanosis is usually more marked than in aortic disease. The large veins also become plainly visible as a result of their complete filling, especially the large external jugulars.

A further important symptom which follows the venous stasis is the œdema, the dropsy of heart disease. As we know from general pathology, every venous stasis, if it reaches a certain grade, leads to a transudation of the fluid of the blood from the capillaries. If the lymphatics can no longer carry this transudation away, it collects in the meshes of the tissues and leads to œdema. The œdema of heart disease, therefore, does not appear until the venous stasis has reached a certain degree, and the compensation of the heart disease has become impaired. It first appears in those parts where there is a particularly loose tissue, as in the eyelids and the scrotum, or where mechanical conditions favor its development. The legs usually swell first, especially about the ankles, because here the stasis of the venous blood is increased by gravity. At first, slight œdema appears only temporarily and by day, and disappears again while the body is in bed at night; but, as the disturbance of compensation increases, the œdema also constantly grows worse, especially in the dependent parts of the body, until finally it may reach the highest degree of dropsy. Besides the œdema of the skin, transudations into the internal cavities occur, especially into the abdomen and the pleural cavity.

The patient's distress is decidedly increased by marked œdema, as has already been said. All the motions of the swollen extremities are considerably impeded. Hydrothorax and ascites increase the dyspnœa, the former by compression of the lungs, the latter by upward pressure on the diaphragm. The passage of urine may be rendered very difficult by œdema of the prepuce. Besides that, we must mention that the skin, when very œdematous, is quite apt to become the seat of furuncular and erysipelatous inflammations.

The results of stasis in the internal organs may be best seen in the liver, spleen, and kidneys.

Passive congestion of the liver is manifested by quite a considerable increase in the size of the organ. The lower boundary of the liver dullness extends several fingers' breadth beyond the edge of the ribs, and the lower border of the liver may often be plainly felt. The liver may also be enlarged in cases where there are no other marked signs of passive congestion, such as dropsy. Quite severe pain in the hepatic region sometimes arises from the tension of the capsule of the liver. In the later stages the liver may grow smaller again from the partial atrophy of its cells.

Jaundice often develops in heart disease, as a result of passive congestion of the liver, or perhaps sometimes from a secondary duodenal catarrh. The peculiar mixture of a cyanotic and slightly jaundiced hue of the skin is very characteristic in many cases, especially in mitral disease. It should be added that the yellowish discoloration of the skin in heart disease is probably not always a genuine icterus, but may be occasioned by the deposit of other pigments in the skin.

Passive congestion of the spleen arises if the stasis of the blood extends to the splenic vein. The spleen increases in size and becomes firm and dense. It is often hard to make out the congestion from the increase of the splenic dullness, because percussion of the spleen is uncertain if there be also ascites, hydrothorax, etc. We can often, however, plainly feel the enlarged spleen under the edge of the ribs on the left.

We recognize passive congestion of the kidneys by the changes in the urine. The amount diminishes; the urine becomes darker, more concentrated, of a higher specific gravity, and of greater acidity. A sediment of uric acid or of sodic urate very commonly forms in it. In marked degrees of stasis there is albumen in the urine. The amount of albumen is usually slight, but it may equal one third or one fourth of its volume. Under the microscope we find, in the urine of simple passive congestion, only an occasional hyaline cast, and a few red and white blood-corpuscles. Genuine acute or chronic nephritis, however, may also develop as a complication in heart disease.

We may refer the numerous gastric and digestive disturbances, like loss of appetite, vomiting, constipation, and diarrhœa, from which such patients often suffer, to the stasis in the blood-vessels of the stomach and intestines.

4. **Embolic Processes.**—The slowing of the circulation, and the disturbances in the nutrition of the walls of the vessels, which result from it, often give rise in heart disease to the formation of thrombi. These are situated in the heart itself, on the diseased valves, in the recesses between the trabeculæ, in the auricles, etc., or else they form in the veins, especially in those of the lower extremity. From these thrombi fibrinous plugs may easily be set loose and enter the circulation, and thus give rise to embolic processes in distant organs. Some of the embolisms, whose clinical relations are especially important, have been more fully described elsewhere, and will be mentioned only briefly here.

Embolism of the pulmonary arteries, proceeding from venous thrombi or from thrombi in the right side of the heart, gives rise to hæmorrhagic infarction of the lungs. Its pathogenesis and symptoms have already been discussed in a previous section (see page 247).

Embolism of the cerebral arteries is a common cause of apoplectic attacks, which are not infrequent in heart disease, and which usually lead to hemiplegia. The anatomical cause of the hemiplegia in these cases is the embolic softening of the brain which develops. The details of this are given in the section on cerebral diseases (see page 737).

Embolism of the larger arteries of the extremities, like the femoral and the brachial, is much rarer than the forms mentioned. It leads to embolic gangrene of the extremities, unless an adequate collateral circulation can be established. The skin of the peripheral parts, the fingers or toes, first becomes cool, bluish, and at last, if the circulation be wholly checked, almost black. The gangrene advances slowly, usually occupying weeks. Ulcerations develop as the necrotic portions are thrown off. The affection is extremely painful. The patient soon becomes very miserable from the pain and the septic fever that usually attend the ulcerations, and extensive gangrene almost always ends fatally.

Embolism of the renal artery, with the formation of an ensuing hæmorrhagic infarction, may run its course without symptoms, but sometimes it may be recognized by the sudden appearance of blood in the urine.

Embolic infarction of the spleen is often marked by swelling of the spleen and by severe perisplenitic pains. In other cases it is wholly without symptoms.

Embolism of a mesenteric artery is a very rare event. Its symptoms consist in a sudden intestinal hæmorrhage, in severe colicky pains, general collapse, and peritonitis.

5. **Complications on the Part of the Nervous System.**—The most important complication on the part of the nervous system—embolic softening of the brain—has already been mentioned. We must also state that cerebral hæmorrhage may occur in heart disease. It is especially frequent in aortic insufficiency, either as a result of co-existing atheroma of the cerebral arteries, or perhaps of the abnormally high tension of the walls of the vessels during systole.

Mental disorders have been repeatedly observed in chronic valvular disease. They are the result of the disturbance of the circulation, and the consequent impairment of nutrition in the brain. Hence they usually make their first appearance in the last stages of heart disease, at the same time with the other disturbances of compensation. The psychoses in heart disease most frequently have the character of melancholia, but conditions of excitement also occur.

6. **Secondary Affections of the Joints** are not rare in heart disease. As acute endocarditis develops in the course of acute articular rheumatism, so, on the other hand, rheumatic pains in the muscles and joints, and even acute swelling of the joints, associated with fever, appear in the course of chronic heart disease. Both affections—that of the heart and that of the joints—arise from the same specific cause of disease, and hence they may occur in varying succession.

7. **General Symptoms—Fever.**—In congenital heart disease, as we have said, the child's general nutrition is very backward. In heart disease in adults, however, we by no means always see an injurious influence on the general condition of nutrition. In many patients we even find a remarkably good development of fat. A marked general disturbance of nutrition, such as great anæmia and general emaciation, often develops in the later stages, especially in aortic insufficiency. The wasting is of course often hidden by the œdema.

In general, chronic heart disease runs its course without fever, but periods often occur in the course of the disease when there is a moderate and usually irregular fever. Marked disturbances of the general condition may or may not be associated with it. The basis of the fever is probably an acute exacerbation of the endocarditis, except, of course, in accidental complications. All variations occur, from a mild febrile movement without further symptoms to a severe acute recurring endocarditis (*q. v.*). In other cases the fever is connected with secondary swelling of the joints, or with embolic processes.

GENERAL COURSE AND PROGNOSIS OF VALVULAR DISEASE OF THE HEART.

The course of valvular disease of the heart is in most cases very chronic. It may last for years. So long as there is a complete compensation, the patient feels almost perfectly well; sometimes he even has no misgivings as to his trouble. The slight difficulty in respiration and the incapacity for physical exertion are noticed, but little attention is paid to them, because the patient is used to them. In other cases there is a moderate disturbance for a long time, but it may be borne quite easily if the patient is rational and prudent in his conduct.

We can not make any general statement as to the length of the stage of compensation, because cases differ very greatly in this respect. It depends in part upon the intensity of the heart disease, in part upon the external conditions under which the patient lives, and in part, certainly, upon the different individual capacity for work and power of resistance of the heart. Thus it happens that many cases last for decades, while in others severe sequelæ appear within a few months. External injurious agencies, acting on the patient, are of great influence on the course of heart disease. Severe physical exertion, an injudicious manner of living, intercurrent febrile disease, mental disturbances, care, and anxiety are often followed by unhappy consequences.

If the first signs of disturbed compensation appear, if severe dyspnœa, slight œdema of the ankles, etc., develop for the first time, these symptoms may disappear again completely under proper treatment. Severe disturbances of compensation even, great general dropsy, associated with very weak and irregular action of the heart, may abate, after a few weeks' duration, and the patient may feel quite well again. Exacerbations of the disease may come on several times and as often improve. Finally, of course, the improvement is incomplete. Persistent œdema and the other results of venous stasis ensue, the symptoms constantly increase in severity, especially the dyspnœa, until the patient dies after a long and distressing illness. Immediately before death in heart disease, certain irregularities in the innervation of the heart and in the respiration sometimes develop. Among these the so-called Cheyne-Stokes phenomenon deserves especial mention. It consists in a peculiar periodical variation in the respiratory movements. There will be, first, a complete pause in the respiration (apnœa), and this will be succeeded by feeble breathing gradually becoming stronger, then gradually abating, and finally ending in another complete cessation of the respiration. The patient usually becomes more comatose during the cessation of respiration; his pupils contract. During the hard breathing the patient recovers somewhat, and his pupils dilate again. The chief cause of this rhythmical breathing is probably to be sought in the decided lowering of the excitability of the respiratory centre. During the apnœa a considerable amount of carbonic acid collects in the blood before the respiratory centre is sufficiently stimulated to resume its activity.

In regard to the single forms of valvular disease, aortic insufficiency generally gives the best prognosis, inasmuch as it may be very perfectly compensated for many years, but if severe disturbance of compensation once occurs in this form of heart disease, it gives a very unfavorable prognosis, since, as a rule, we can not re-invigorate the heart. Mitral insufficiency is also quite a favorable form of heart disease, which may be compensated for a long time. Mitral stenosis is decidedly more unfavorable in its prognosis, and is associated with more disturbance; but in all mitral diseases very severe conditions may improve once or even repeatedly. Aortic stenosis is also capable of quite good compensation, and in this respect it is even more favorable for the patient than mitral stenosis, but it often causes persistent cerebral symptoms, such as headache and vertigo, depending on anæmia of the brain.

Whether established valvular disease of the heart is curable is a question which can not be answered unconditionally in the negative. Of course in the great majority of cases it is in itself incurable; only its sequelæ can be prevented or removed to a certain degree. In children and young people, however, cases sometimes do occur, as we ourselves have seen, in which there are all the signs of a pronounced heart disease, but after a long time recovery is complete. Of course it is very hard to decide whether we really have to do with a valvular disease that has been cured, because simple dilatation of the heart, relative insufficiency of the valves, anæmic cardiac murmurs, etc., may easily be confounded with pure valvular disease of the heart.

Among the dangerous intercurrent accidents in valvular disease we must make especial mention of embolic processes, which may occur suddenly and without warning. The different forms of embolism have been mentioned above, and also the occurrence of cerebral hæmorrhage in heart disease. Intercurrent acute diseases, like typhoid and pneumonia, often take a very severe and dangerous course in patients with heart disease, because they make increased demands upon the heart's capacity for work.

TREATMENT OF VALVULAR HEART DISEASE.*

1. *Prophylaxis.*—Our remedies to prevent the development of heart disease are very limited. To avert endocarditis in articular rheumatism by the present method of treating acute rheumatism with salicylic acid is impossible. The probability of the onset of endocarditis may be lessened only so far as the whole duration of the disease is often considerably shortened by salicylic acid.

We can also do little in the way of prophylaxis against the development of heart disease that is chronic from the start, since the cause of the disease is in many cases wholly unknown to us. Those injurious influences deserve the most attention which may favor the development of arterial atheroma and its consequent chronic valvular disease. Undue indulgence in alcohol, too much smoking, and injudicious and luxurious ways of living, are especially to be avoided. The part which these factors play, however, in the development of a pure valvular disease, is certainly much less than their influence upon the development of certain functional and nervous disturbances of the heart (see a later chapter).

[There is evidence that the alkaline treatment of acute rheumatism lessens the danger of cardiac complications (see page 909).]

2. *Treatment of Compensated Heart Disease.*—If we have to treat a heart disease which already exists, but which is at the same time fully compensated, our treatment must be chiefly hygienic. The patient must be made aware of his heart disease without making him needlessly anxious. He must be told that his further good health depends in great part upon his own conduct, his discretion, and his perseverance. The patient must avoid everything which makes great demands upon the heart, or which may have a directly injurious influence on it. All violent bodily exertion, too intense mental work, and also all excesses in eating, drinking, smoking, etc., must be avoided. That the physician's directions will often collide with the demands of the patient's occupation, as well as with his favorite amusements and his habits, should not deter the physician from demanding the fulfillment of his prescriptions, at least so far as possible.

Treatment by drugs is usually unnecessary in compensated heart disease. We do not know a remedy which has a directly favorable action on heart disease. The protracted use of iodide of potassium, Fowler's solution, arsenite of antimony ("granules of antimony"), nitrate of silver, etc., has been recommended. The efficacy of these remedies is not proven. We can always try them, if a mild disturbance makes a prescription desirable and other remedies are not especially indicated. Beyond this, the physician is usually contented with an endeavor to improve the appetite and nutrition of the patient by means of iron, quinine, and bitters. If there is a suspicion that the heart disease may be due to syphilis, a trial of iodide of potash may be made, but a brilliant result from the employment of anti-syphilitic measures is hardly to be expected.

The employment of baths in heart disease deserves special consideration. Numerous experiences go to prove that they are not only well borne by cardiac patients, but that they exercise a peculiarly beneficial and invigorating influence upon the action of the heart. In this regard, the greatest reputation is possessed by the warm mud baths, which are rich in carbonic dioxide, particularly those at Nauheim. Even where there is incipient failure of compensation, there will be marked improvement upon the use of these or similar baths. Patients may also employ at home either simple, or, still better, salt baths, at a temperature of 90·5°–93° (26°–27° R.). That a change of climate may prove beneficial is self-evident. It is particularly appropriate that patients with a tendency to bronchitis or rheumatism should spend the winter South.

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* A brief critical sketch of the mechanical treatment of circulatory disturbances, the so-called Oertel method, will be found in the appendix to the fifth chapter of this section.

3. *Treatment of Disturbances of Compensation.*—As soon as the compensatory activity of the heart begins to be impaired in valvular disease, as soon as dyspnœa and œdema ensue, we must, almost always, resort to digitalis. Digitalis in small doses has the property of reducing the frequency of the pulse, and also of strengthening the individual heart-beats, and thus increasing the blood-pressure. Digitalis is indicated in every case of heart disease if disturbances of compensation appear, and if the pulse also is abnormally small, of abnormally low tension, of increased frequency, and irregular. The desired action of digitalis, then, is to make the pulse slower, more regular, and of higher tension. Under the influence of an increase of the arterial pressure thus produced, the disturbances of compensation disappear in a way that is often surprising; the urine especially bceomes more abundant, the scanty, dark, concentrated urine of passive congestion disappears, the daily amount increases, and the urine therefore becomes clear and of a low specific gravity. The œdema also disappears, the dyspnœa ceases, the head is clear, the general condition is better—in short, a complete compensation of the heart disease may be re-established. This change is sometimes accomplished in a comparatively short time—in a few days or weeks.

The dose in which digitalis is to be prescribed can not be given absolutely, because the amount of the active principle which the plant contains does not seem to be quite alike in different places. This is the explanation of the somewhat different statements as to the dose to be employed. In general, we should begin with small doses. We usually prescribe an infusion of the leaves of a strength of 1 or 1·5 parts of digitalis to 150 parts of distilled water—a teaspoonful every one or two hours. The addition of syrup causes the medicine to decompose more easily, and is quite useless. The favorite addition of diuretics, liquor potassii acetatis, 30 parts, or boro-tartrate of potassium and sodium, four to eight parts added to the infusion, diminishes the strength of the remedy. It is advisable to give digitalis in powder, because we can thus define the dose more accurately. We prescribe powders of one or two grains of digitalis leaves (grm. 0·05–0·1) with seven or eight grains of sugar (grm. 0·4), and give that amount every two or three hours. The drug may also be given in capsules or suspended in water. It is often a good plan to inclose the powder in capsules. The other preparations of digitalis, the vinegar and the tincture, are less active. We usually prescribe the latter when a patient with a moderately persistent disturbance of compensation is to use digitalis in small doses for a long time. We have not yet succeeded in isolating the active principle of digitalis in a durable form. The "digitaline" preparations brought into the market up to the present time are uncertain in their action, and hence are properly but little prescribed.

Digitalis is a remedy which is not free from danger, because there is a stage in its action, which often appears, when it is injurious. The pulse then sinks below the normal, becomes weaker and also irregular and intermittent, if it was previously regular. The patient's general condition becomes bad, nausea and vomiting set in, and a state resembling collapse may arise. Therefore it is a very important rule, never to prescribe digitalis in large doses unless the patient can be observed constantly and carefully. As soon as the first signs of the unfavorable action of digitalis occur, the remedy must be at once omitted. Then we give some strong wine, or strong *café noir*, to prevent the patient's further collapse. Since digitalis is one of the remedies which have a so-called cumulative action, the occurrence of symptoms of poisoning may be quite rapid and unexpected. We do well, therefore, if the desired favorable action follows the exhibition of digitalis, if the pulse has become quieter and more regular, and diuresis has been established, to omit the digitalis. We usually let the patient take the remedy for two to four days, and then omit it. The action of the digitalis continues for several days afterward. Sometimes, however, nausea and vomiting are occasioned by the ingestion of no more than fifteen to twenty grains (grm. 1 to 1·5) of digitalis. The drug must then be stopped, although a decided beneficial effect may be observed even on the day after the nausea. After leaving off the digitalis we usually prescribe a diuretic, like acetate of potassium, squills, boro-tartrate of potassium and sodium, or diuretic herbs. If the pulse again becomes rapid and irregular, we at once resume the digitalis. In such cases, where the patient has used digitalis repeatedly, we must gradually increase the dose. The patient gets accustomed to the remedy, as happens with so many other drugs. There is no maximum dose, and in individual cases we must find out the requisite amount by trial. Many patients finally become genuine "digitalis eaters," and can not exist without large doses—seventy-five grains (grm. 5) or more of the powder a day. In very many cases, unfortunately, the favorable action of the digitalis finally ceases entirely with large doses. The remedy is then no longer "borne," and we must omit it entirely. By that time the last stage of the disease has also usually been reached.

If we have to treat a patient with symptoms of stasis, and the pulse seems neither especially small nor frequent, nor irregular, we may nevertheless try the effect of digitalis, but we must be very cautious in the size of our dose. The question is often hard to decide whether to give a patient with aortic insufficiency digitalis or not. In this form of heart disease there is often a very strong and regular, though of course abnormally frequent pulse, in spite of the most pronounced disturbance of compensation. With this we must not forget that, in spite of the high pulse, the mean pressure in the arteries is low, and hence in most patients with aortic insufficiency we can very well make a trial with digitalis. This is often crowned with success; but still the drug seems to be less often efficient in aortic than in mitral disease.

In some, but fortunately not many, cases, the digitalis is ill-borne from the start, because it excites severe nausea and vomiting. In general the incidental action of digitalis on the stomach is one of its troublesome properties. If the digitalis is not borne in one form, we try another. We give the powder instead of the infusion, or *vice versa*, or we employ the tincture. In such cases we sometimes succeed if we give the digitalis as an enema, using a one-per-cent. infusion.

Caffeine deserves the first mention among the substances having a similar action, which have of late been recommended as substitutes for digitalis by Lépine, Riegel, and others. Given in repeated small doses, a total of fifteen to twenty grains a day (grm. 1·0 to 1·5), it often slows, regulates, and strengthens the activity of the heart, and also increases the arterial pressure. The salicylate of caffeine and sodium is chiefly used, three to five grains (grm. 0·2 to 0·3) of the powder, and also the benzo-citrate of caffeine in the same or smaller doses. The latter is also given subcutaneously. Besides caffeine, we may also mention here adonis vernalis and convallaria majalis; but none of these drugs are as reliable as digitalis. In conditions of cardiac weakness, tincture of strophanthus (ten drops several times a day) is sometimes decidedly beneficial.

[Digitalis is used more commonly in this country in the form of the tincture. The urine affords a good guide as to the safety of the continuance of the drug; as long as the renal secretion is sufficient in quantity, and increasing rather than diminishing, there is no danger of the toxic effects. It is, consequently, a good plan to follow carefully the twenty-four-hour quantity of urine when this can be done.

There are cases in which digitalis must be taken for long periods, but it should then be given only twice a day, with twelve hours' interval between the doses, unless the patient has ready access to his physician; there is then less risk of toxic symptoms. To the list of drugs the action of which is similar to that of digitalis and which may be substituted for it, if it disagrees or is without effect, may be added helleborein and spartein sulphate (gr. j–ij). Fraser reports well of strophanthein; but a more extended use is desirable.

In mitral cases, with or without secondary tricuspid regurgitation, where the cyanosis and other symptoms show that the right heart is engorged with blood which it can not propel onward, the relief afforded by venesection, or by a dozen leeches in the hepatic region, may be very great. Until the veins are relieved either in this way or by free purgation, digitalis and stimulants are useless, and a resort to them results merely in a loss of time, and perhaps in a loss of life which might be saved.]

4. *Symptomatic Treatment.*—Some symptoms which often occur in heart disease demand a special description.

Dropsy is a symptom of venous stasis, and disappears if compensation be restored, spontaneously or by the use of digitalis. Complete rest in bed and elevation of the swollen parts serve as the chief aid in removing the dropsy. Dropsical patients ought also to change their position in bed frequently, if possible, that there may not be too much œdema collected in the dependent portions of the body. It is a good plan to wrap up the swollen arms and legs with flannel bandages under gentle pressure. Mild massage of the œdematous parts may sometimes be of advantage. The amount of liquid ingested is to be limited, if possible. Of internal remedies, besides digitalis and similar drugs, the true diuretics, such as acetate of potash, are to be considered. They are sometimes ordered in combination with digitalis and sometimes alone, particularly when digitalis can not be borne or is not indicated. Calomel is sometimes especially efficient in cardiac dropsy. Its diuretic influence has lately been emphasized by Jendrassik and others. It is prescribed in powders of three grains (0·2 grm.) three to five times a day. Often a very marked diuresis will be caused after one or two days, with rapid abatement of the dropsy. The administration of the remedy is stopped as soon as the diuresis begins. It is also omitted, of course, if stomatitis develops.

In the last stages of heart disease the patient's condition may be particularly distressing from the severe general œdema. It is then justifiable to remove the ascites or hydrothorax by puncture, and to let the œdema drain out by scarification of the skin—little pricks with the point of a knife—in order to procure relief for the patient. The scarification of the skin, however, is dangerous, and is not to be employed without urgent indications, because erysipelatous inflammation, etc., it apt to ensue at the point of incision. We can recommend little silver capillary trocars (the so-called Southey's trocars), to which a thin rubber tube is attached. By the aid of these trocars we can drain off large amounts of serum. We must always use great cleanliness, however, and the utmost disinfection of the skin, wrapping the parts in carbolized jute or salicylated cotton. In patients with heart disease it is not, as a rule, advisable to attack the dropsy by sweating, by hot packs, or pilocarpine.

The dyspnœa of heart disease is usually the most distressing symptom of all. Here, too, our chief task is of course to restore the compensation; but this failing, we must try to relieve the dyspnœa symptomatically. Morphine is most efficient in this respect. In general, morphine is, next to digitalis, the most indispensable remedy in the treatment of severe heart disease. It is usually well borne, and procures great relief, especially if given subcutaneously. If we have to do with the last stage of the disease, we need not spare large doses. Otherwise, of course, caution is necessary. In practice we must often prescribe external applications to the chest, mustard-plasters, hot poultices, and also hot foot-baths with mustard, ashes, etc. In severe cases their action is slight. Acetate of lead in large doses sometimes seems to have a favorable influence in severe dyspnœa, especially with threatening pulmonary œdema. We give the powder, up to a grain and a half (grm. 0·1), every two or three hours, and it is often a good plan to add half a grain or a grain (grm. 0·03 to 0·05) of opium. We can also frequently obtain distinct relief for the patient by a vigorous drastic purge, with compound infusion of senna or gamboge. Inhalations of nitrite of amyl seldom have a beneficial effect.

Palpitation, constant or paroxysmal, is met by applying ice to the cardiac region; sometimes the "heart-flasks," made of tin, act very well. In patients with aortic insufficiency and very excited action of the heart, we may recommend the protracted use of ice. The narcotics are the most efficient internal remedies, especially morphine, which, of course, we should reserve for severe cases. If the palpitation is of a lesser degree, we may try bromide of potassium, or cherry-laurel water, eventually with equal parts of tincture of digitalis, twenty to thirty drops of the mixture two or three times a day.

The subcutaneous use of morphine is again by far the most potent remedy in the anginose attacks, associated with pain and a feeling of distress. We may also use cutaneous irritation, mustard-plasters, etc., ice, and perhaps strophanthus or nitrite of amyl.

We may prescribe bitter remedies—tinctura amara (P. G.), or compound tincture of cinchona—and muriatic acid for the loss of appetite, in case this is not improved by regulating the activity of the heart. Besides that, we must always endeavor to get a regular evacuation of the bowels.

For the attacks of faintness and vertigo, occurring especially in aortic stenosis, as a result of cerebral anæmia, we may prescribe a horizontal position, and stimulants—wine, ether, and Hoffmann's anodyne. If the cerebral symptoms depend upon venous stasis, we try to remove it by ice, mustard-plasters to the neck, and thorough derivation to the intestines.

Especial accidents and complications, like pulmonary œdema, infarctions, or apoplexy, are to be treated according to the usual rules.

CHAPTER III.

MYOCARDITIS.

(*Indurated Degeneration, Myodegeneration of the Heart. Infarction of the Heart. Sclerosis of the Coronary Arteries.*)

Ætiology and Pathological Anatomy.—We have of late learned about a number of affections of the heart in which all the results of disturbance of the circulation may finally appear, without any evidence of anatomical disease of the valves, either during the patient's life or at the autopsy. We have to do here with a lesion of the cardiac muscle itself, or of its nervous apparatus, which reduces the functional capacity of the heart, and thus excites precisely the same disturbances of the circulation as are produced in valvular disease of the heart by purely mechanical conditions.

In a number of cases—but by no means in all, as we shall see in the next chapter—we succeed in detecting striking anatomical changes in the cardiac muscle. The heart is on the whole enlarged, chiefly dilated, though its walls are usually somewhat hypertrophied (*vide infra*). In pure, uncomplicated cases the valves prove entirely normal. If, however, we examine the cardiac muscle closely, we find it studded with whitish, lustrous, indurated spots, arranged irregularly and often very abundant. They consist microscopically of cicatricial connective tissue, while the muscular fibers have wholly or in large part been destroyed. The seat of this induration is chiefly in the left ventricle, especially at the apex and in the anterior wall, but we may also find indurations in other portions of the heart. We often see them appearing through the endocardial or pericardial surface of the heart as dull, slightly sunken places. The papillary muscles may also undergo marked cicatricial degeneration.

The changes in the heart just depicted were formerly considered to be of an inflammatory nature, and hence were given the name of myocarditis; but their development is connected, in the great majority of cases, as Weigert, Ziegler, Huber, and others have shown, with changes in the coronary arteries and their branches. In most of the uncomplicated cases of so-called myocarditis we find marked atheromatous changes in the coronary arteries, usually coincident with a more or less extensive, general arterio-sclerosis (*vide infra*). These changes may lead in circumscribed places to a complete closure of a branch of the coronary artery by thrombosis, and thus give rise to the formation of a true infarction of the heart by cutting off the further blood-supply. These infarctions show, in fresh cases, a decided brownish-yellow, hæmorrhagic color. The finer histological changes consist in a loss of their nuclei by the muscular fibers, and their degeneration into a crumbling, cheesy detritus. By the new formation of connective tissue the peculiar cardiac indurations finally arise, which are therefore to be regarded as true infarction cicatrices.

In many cases of indurated degeneration we can not discover any complete closure of the branches of the coronary artery by thrombosis. Here we have to do only with changes in the caliber of the vessel by the arterio-sclerotic process, and a consequent diminution of the arterial blood-supply of the cardiac muscle. In all places where this diminution of the blood-supply becomes extreme, we also find a gradual deterioration of the muscular fibers, and their replacement by connective tissue. Where the arterial blood-supply is adequate, either directly or from the collateral circulation, the muscles remain intact. This is the reason why we often find sclerosis of the coronary arteries unaccompanied by indurated myocarditis.

Besides the cases of uncomplicated myocarditis just described, we often see the same cicatricial changes in the cardiac muscle combined with valvular affections. The cardiac indurations, then, are either due to a co-existing sclerosis of the coronary arteries, or they stand in a closer relation to the endocardial process. The endocarditis may invade the cardiac muscle immediately. This method of origin of the myocarditis may be easily recognized by the localization and extent of the diseased parts. In other cases the endocarditis may give rise to embolic processes in the cardiac muscle. Thus the embolic infarctions of the heart arise in just the same way as do the thrombotic infarctions above described.

It remains for us to describe a number of sequelæ and combinations of indurated myocarditis. As a result of the extensive cicatricial formation, it may happen that a circumscribed portion of the wall of the heart, usually of the left ventricle, becomes thin and yields to the blood-pressure acting upon it from within. Thus arises a partial protrusion of the wall of the heart, a so-called cardiac aneurism. Such an aneurism, and also an extensive, fresh infarction in the heart, may in very rare cases lead to a rupture of the heart, with effusion of blood into the pericardium and sudden death. A more common occurrence is that sometimes in places where the cardiac cicatrices reach the endocardium, parietal thromboses form in the heart, and give rise to embolic processes in distant organs.

Dilatation of single portions of the heart, especially of the ventricles, depends upon a general yielding of the walls of the heart. If at the same time there is a hypertrophy of the cardiac muscle, we must look for special causes for it. Hypertrophy of the left ventricle usually depends upon co-existing general arterio-sclerosis, upon co-existing contracted kidneys, and the like. Again, it is probably often the case that the same causes give rise simultaneously to the atheroma and to the hypertrophy of the left ventricle (see the following chapter). Hypertrophy of the right ventricle may have its origin in co-existing chronic pulmonary affections, like emphysema. In some cases the hypertrophy of the right ventricle is due to the inadequate action of the left ventricle. If the left ventricle begins to grow weak, the stasis backward from it must extend through the pulmonary vessels into the right side of the heart. In consequence of the increased demands upon the right ventricle this becomes hypertrophied. In cases of indurated myocarditis combined with valvular disease, the hypertrophy of single portions of the heart depends, of course, in part upon the valvular disease.

Regarding the special ætiological factors which lead to sclerosis of the coronary arteries, and thus to indurated myocarditis, they are in many cases as little known as the causes of arterio-sclerosis in general (*vide infra*). Chronic alcoholism and luxurious habits of life are very often the predisposing causes which are to be put in the first rank. Perhaps constitutional syphilis plays a not unimportant part, since it may lead to changes in the coronary arteries, as it has long been known to do in the cerebral vessels. Constant mental excitement is also regarded as a factor; but this must be regarded as much more a cause of the conditions to be described in a following chapter. Heredity, however, is of greater influence, as it seems to play a certain part in the development of arterio-sclerosis. Finally, we must remember that the atheromatous process occurs much more frequently in advanced life than in young people; hence indurated myocarditis is a disease chiefly to be observed in the aged. Men are more frequently attacked by it than women.

Although we have so far spoken exclusively of chronic indurated degeneration of the cardiac muscle, which, as we have said, is not of a peculiarly inflammatory nature, we must also add that there is a true purulent inflammation of the cardiac muscle. This is one of the symptoms of a general, infectious, and especially a pyæmic process, or of malignant acute endocarditis, so that we may refer to the description of these diseases in regard to abscess of the heart.

Clinical History.—We must first mention that sometimes quite extensive cicatricial formation may be found in the cardiac muscle *post-mortem*, without the occurrence of any manifest symptoms referable to the heart during life. We see, then, that the heart may, under some circumstances, undergo quite a considerable loss in its contractile substance without injury.

In many cases, however, the heart's capacity for work suffers so much that the same symptoms arise as in valvular disease. The course of such cases may be very chronic. The symptoms begin quite gradually. The patient first has a slight dyspnœa or palpitation, and a feeling of distress in the chest, but only from external causes, like slight physical exertion. He sometimes suffers from a striking general weakness. He gets tired easily and feels heavy, and more or less incapable of any mental activity. The symptoms gradually increase, and just the same results of disturbance of the circulation appear as in all the other forms of heart disease. The difficulty in breathing becomes more marked, œdema occurs, signs of stasis in the liver, intestines, and kidneys appear—in short, the well-known type of every uncompensated heart disease develops.

Physical examination of the heart shows marked anomalies of the heart's action in all severe cases. The pulse is often irregular in regard to its rhythm and the intensity of its single beats, but the arhythmia may also be wholly absent in spite of the degeneration of the myocardium, as we have often convinced ourselves. The pulse is at first quite strong and full, later it becomes weaker, of lower tension, and at last sometimes very small and scarcely perceptible. It may be increased in frequency, but we quite often notice in chronic myocarditis, especially in the early stages, a persistent slowing of the pulse to 60, 50, or even less, in a minute. With this slowness of the pulse there is also frequently irregularity of the heart's action, especially the appearance of occasional double beats (pulsus bigeminus). Percussion usually shows an increase of the heart's dullness, due to dilatation or hypertrophy of the heart, the increase being either general or chiefly on one side. Auscultation shows the absence of any murmur, and hence the absence of valvular disease. The heart-sounds are distinctly audible, and sometimes quite loud and valvular, but in the later stages often low and obscure. The pulmonic second sound is accentuated, when there is stasis of the pulmonary circulation. In several cases we found the second sound for a long time very plainly divided—reduplicated. We must also mention that sometimes in pure myocarditis a systolic murmur is heard at the apex which is due either to a relative insufficiency of the mitral valve, or to its incomplete closure, as a result of defective muscular action of the left ventricle.

The whole course of chronic myocarditis is in most of its relations precisely analogous to that of chronic valvular disease, so that we need not describe its peculiarities in detail. The patient is alternately better and worse. Severe symptoms of general stasis and manifest weakness of the heart may appear, and, under favorable circumstances, may disappear again. The picture of the disease may be complicated by embolic processes in the brain, the lungs, etc., which usually arise from thrombi in the heart (*vide supra*). Finally, perhaps after the disease has lasted for years, the patient dies from general dropsy or from some intercurrent disease.

We must mention one symptom which has often been brought into special relation with sclerosis of the coronary arteries and with chronic myocarditis. We mean the attacks of so-called angina pectoris, the stenocardiac paroxysms. These attacks consist in a sudden, intense pain in the cardiac region, which shoots into the back, the left shoulder, and the left arm. This pain is associated with a marked feeling of constraint and distress. There is no particular dyspnœa in pure angina pectoris. The attacks last from a few minutes to half an hour, and may be interrupted by entirely free intervals.

We can not deny that such attacks occur in indurated myocarditis, due to sclerosis of the coronary arteries, but, on the other hand, many cases run their course without angina pectoris, and angina pectoris may also occur both in other forms of heart disease and as a pure neurosis. Hence we must not overestimate the significance of angina pectoris in the diagnosis of sclerosis of the coronary arteries. Besides angina pectoris, attacks of dyspnœa (cardiac asthma) and of faintness also occur in chronic myocarditis, the faintness being especially frequent in cases associated with a slow pulse. The dyspnœa is usually due to a sudden weakness of the left ventricle, while the origin of the faintness is not yet fully explained (cerebral anæmia ?).

It is a very important clinical fact that, in not very rare cases, indurated myocarditis is the sole apparent cause of sudden, apoplectiform death. It usually happens in elderly people in comfortable circumstances and good livers, who up to that time have not regarded themselves as really ill; but they have repeatedly had slight attacks of vertigo, of oppression, etc. Suddenly a sort of apoplectic attack comes on, often after some definite cause, after dinner, or after some physical exertion or mental excitement. Death follows in a few moments, or after a deep coma that lasts for several hours or even days. The diagnosis often remains in doubt in such cases, especially if we have not known the patient previously. The autopsy shows, as the sole pathological lesion, a sclerosis of the coronary arteries, with a more or less extensive cicatricial formation in the heart. Apparently in these cases the moment must suddenly arise when the supply of blood to the heart is insufficient, and thus death is caused. Experiments upon artificial closure of the coronary arteries, by Cohnheim and others, agree perfectly with the clinical facts above mentioned. Artificial narrowing of the coronary arteries may also be well borne for a long time, until suddenly both halves of the heart stand still in a condition of diastole. In rare cases sudden death may also be due to embolism of the main trunk of the coronary artery, or, as we ourselves have seen in one case, to the bursting of a focus of myocarditis with hæmorrhage into the pericardial cavity.

Diagnosis.—The diagnosis of chronic myocarditis is by no means always easy and certain. We must first determine that there is a cardiac lesion of some kind. This is usually easy to do from the secondary symptoms of stasis, the condition of the pulse, the heart's dullness, etc. Then the question arises whether we have to do with a valvular disease or with a myopathic disease of the heart. Here auscultation must chiefly decide. The absence of a heart murmur, in spite of other definite signs of heart disease, speaks against valvular disease, but not with complete certainty. All murmurs may be absent in the last stages, especially with a high degree of mitral stenosis, and hence we may easily confuse mitral stenosis with myocarditis, particularly when there is marked arhythmia of the heart. On the other hand, we have already stated that, in pure myocarditis, with the valves intact, functional murmurs may be present, which may lead to an erroneous opinion as to valvular disease. If by long observation we have excluded a valvular disease, we still have left the distinction between myocarditis and pure idiopathic hypertrophy of the heart, or fatty heart (see the following chapters). We hold it impossible to make this distinction with certainty. The conditions of disease mentioned all furnish the same clinical picture of cardiac insufficiency, but during life we can only suspect what finer anatomical relations are the ones which cause this insufficiency, and we can never diagnosticate them with certainty. Irregularity of the pulse occurs both in indurated myocarditis and in pure hypertrophy and dilatation, without discoverable cicatricial nodules in the substance of the heart. A persistently slow pulse, attacks of angina pectoris or cardiac asthma, the evidence of co-existing sclerosis of the arteries of the body, such as the radial, temporal, or femoral, in connection with other symptoms, make the diagnosis of indurated myocarditis probable, but never completely certain. In cases, too, with a sudden apoplectic attack—"heart apoplexy"—it is often impossible to make a certain distinction between it and cerebral embolism, or cerebral hæmorrhage.

Prognosis.—The prognosis is as serious as in any valvular disease. Recovery is impossible, but even extensive cicatricial formation in the heart may probably last for years without causing much disturbance. We must always be prepared for the occurrence of disturbances of compensation, and the manifold sudden accidents to which patients with myocarditis are exposed, but we can not foretell the time of their occurrence.

Treatment.—The treatment of chronic myocarditis must be directed first to the dietetic and hygienic care of the patient. This is of the greatest importance.

For obese persons accustomed to high living, a moderate, simple diet must be accurately prescribed. Alcoholic beverages must be greatly limited or wholly forbidden, and not more than two or three cigars allowed per diem. Moderate bodily exercise is beneficial, and indeed necessary, for the promotion of the circulation and the more rapid diminution of the obesity, but the patient must be warned against too violent exertion, nor can great mental effort be permitted. In summer, a quiet life in the country or some mountain region is to be advised, or, under suitable circumstances, the cautious use of the waters of Carlsbad, Kissingen, Marienbad, or Nauheim. Frequent tepid baths or mud baths may usually be employed even at home with advantage. With disturbance of compensation, and with abnormally frequent, weak, and irregular action of the heart, digitalis and similar remedies are indicated, just as in valvular disease. In cases with an abnormally slow pulse, we may use them, provided great caution is exercised, but we must also be governed by the other prevailing symptoms. In attacks of angina pectoris the subcutaneous injection of morphine is by far the most efficient and often an indispensable remedy. We may also try nitrite of sodium, 1 or 2 parts to 120 of water, two or three teaspoonfuls daily. The inhalation of a few drops of nitrite of amyl is also sometimes beneficial, but more often it has no effect. In cardiac asthma, stimulants like strong black coffee, ether and camphor, and often narcotics, are indicated. Mustard-plasters, cold and warm poultices, hot foot-baths, etc., are also employed. Iodide of potassium and arsenic are especially recommended for continued use in myocarditis. The first remedy sometimes seems to be of service, and is especially to be recommended where there is a suspicion of former syphilis. With regard to all further details we must refer to the preceding chapter.

CHAPTER IV.

IDIOPATHIC HYPERTROPHY AND DILATATION OF THE HEART.

(Over-exertion of the Heart. Weakened Heart.)

Ætiology and General Pathology.—Besides the forms of heart disease already described, cases not infrequently occur which present all the signs of an uncompensated heart disease during life, and which show at the autopsy a hypertrophy of the heart or a dilatation of its cavities, but no other abnormity, either in the valves, in the coronary arteries, or in the cardiac muscle. The cardiac hypertrophy, which involves the left ventricle chiefly, but often both ventricles, can not be regarded as secondary in the ordinary sense of the word, for in the heart itself and in the other organs we find nothing which can call forth a secondary hypertrophy of the cardiac muscle—no valvular disease, no chronic nephritis, no general arterio-clerosis, and no pulmonary emphysema. Hence we term these cases "primary idiopathic" cardiac hypertrophy, in the sense that we can not discover any other primary disease in them. Nevertheless, we must also look in such cases for factors which during life result in increased work for the heart, because only thus can we understand the development of this form of cardiac hypertrophy.

Such factors may in fact often be discovered. In a few rare cases a congenital narrowness of the aortic system plays a part, since by this the heart's work is manifestly increased. The observations upon this point, however, are very scanty, so that we can not yet estimate with certainty the practical significance of this arterial anomaly.

Excessive physical exertion is of much greater ætiological importance. We not infrequently see idiopathic cardiac hypertrophy develop in the hard-working classes—in blacksmiths, locksmiths, pack-carriers, and vine-dressers ("Tübinger heart"). The increased work of the heart upon every physical exertion is here repeated almost every day in the year, and must finally lead to a marked degree of hypertrophy from labor. This is the chief form which has been termed "cardiac over-strain."

Numerous cases of idiopathic hypertrophy of the heart may often be regarded as the result of long-continued gourmandizing, hence the frequent appearance of hypertrophy in the obese, and particularly in great beer-drinkers. The constant excessive ingestion of liquid undoubtedly gives rise to an increase in the amount of blood, and so overburdens the heart. Likewise the abundant ingestion of food occasions a constant slight increase of the blood pressure, and so, again, an increase of the work demanded of the heart. These influences are finally re-inforced by all the consequences of increasing obesity, in its relations to the circulation, which we shall consider more minutely in the chapter upon obesity. Excessive indulgence in beer has certainly the worst effect upon the heart. There is both a large amount of fluid, four and sometimes even eight or ten quarts a day, and a large amount of nourishment. Four quarts (litres) of beer contain about eight ounces (250 grm.) of hydrocarbons, in solution, and therefore capable of complete absorption into the circulation. This explains the great frequency of cardiac hypertrophy which Bollinger has shown to exist at Munich ; but the "Munich beer-heart" is seen with unfortunate frequency in other towns than Munich. The alcohol is probably not a factor in the development of the hypertrophy of the heart, but we may well suppose that it promotes, or at least hastens, the degenerative changes in the cardiac muscle, and particularly in the cardiac nerves, which at last render the heart's action inadequate and the circulation imperfect.

Finally, however, there are always a number of cases in which none of the causes so far mentioned can be discovered. In these cases we are inclined to conjecture that there is an abnormal nervous irritation of the heart, which excites it to increased activity, and hence causes its hypertrophy. The cases which seem to be connected with continued mental excitement in business men, etc., probably belong to this group. We may also recall the cardiac hypertrophy in exophthalmic goître (*vide infra*).

Since the exciting causes above mentioned to explain the origin of the so-called idiopathic cardiac hypertrophy do not by any means constantly result in this condition, we must also assume a special individual, and sometimes apparently a hereditary predisposition, a congenital or acquired weakness of the cardiac muscle. Up to a certain degree a healthy, strong heart can respond to the increased demands made upon its activity. We are even justified in considering a certain amount of hypertrophy in such cases as by no means pathological, just as there is nothing pathological in the hypertrophied muscles of a gymnast ; but experience shows that the relations of the cardiac muscle are different from those of the muscles of the body, for the hypertrophied heart does not permanently fulfill the increased demands put upon it, but it gradually begins to be paralyzed, and becomes insufficient. Hence the English physicians, since Stokes's time, term the cases of cardiac insufficiency, without any apparent coarse anatomical lesions of the valves or muscle, "weakened heart." This term does very well for those cases where the symptoms of cardiac insufficiency have appeared before much hypertrophy has developed. There are pure cases of weakened heart in which the heart seems merely dilated and its walls flabby, and not at all or only slightly hypertrophied. Such a condition shows all the clinical symptoms of a chronic heart disease with disturbed compensation.

It may be mentioned here that dilatation of the heart may be very acute in its development under certain circumstances, if great demands are temporarily made upon a heart which is not capable of very much exertion. Acute dilatation of the heart has been seen in soldiers after a few forced marches. We once saw a case in a previously healthy man who fell into the water and could only with difficulty be saved from drowning. Acute dilatation also occurs in the course of severe febrile diseases, like typhoid, intermittent, or pneumonia, which are sometimes associated with manifest weakness of the heart. In previously diseased hearts, too, acute dilatation may develop after some special cause. These acute dilatations may in many cases be restored to normal dimensions, but they always point to a certain degree of permanent weakness of the heart.

Clinical History.—Idiopathic dilatations and hypertrophies of the heart may certainly exist for a long time without causing the patient any subjective disturbance. The symptoms begin when the heart can no longer respond to the demands made upon it, and when it begins to be paralyzed. Then all the symptoms of cardiac insufficiency arise, in just the same way as in valvular disease and in the severe muscular diseases of the heart. Hence we need not go into the details of the disturbances of compensation again. The whole series of symptoms of stasis, as well as the attacks of angina pectoris and cardiac asthma, described in the preceding chapter, also occur in idiopathic hypertrophies and dilatations of the heart.

The general course of the disease differs considerably in individual cases. Sometimes there is moderate difficulty in breathing for a long time, especially on any physical exertion. The patient often complains of great languor, of nervous irritability, and sometimes of attacks of vertigo and faintness, and a tendency to perspiration. The appetite is poor, and there is very apt to be constipation. The condition may become quite suddenly worse after any marked injurious influence, especially after great physical exertion or mental excitement. The pulse is small, weak, and irregular, the heart-sounds grow feeble, the dyspnœa and oppression in the chest increase, the amount of urine diminishes, and œdema appears in the legs. We now have the complete picture of an uncompensated heart disease. With proper treatment the symptoms may disappear again; but, sooner or later, they return. Death finally ensues from general dropsy or from some complications or intercurrent attacks, among which we may mention embolic processes.

If the patient avoids all injurious influences by a discreet and prudent way of living, the course of the disease may be quite favorable for years. It is not improbable that a number of mild cases may be restored to health, or at least remain stationary.

Diagnosis.—The diagnosis is based on the presence of the ætiological factors, and on the same symptoms as generally point to a disturbance in the heart—namely, palpitation, shortness of breath, acceleration and arhythmia of the pulse, etc. Physical examination in the later stages of the disease gives an increase of the heart's dullness in both directions, usually chiefly to the right. Auscultation permits us to exclude valvular disease by finding the heart-sounds everywhere distinct. We have left, then, only the hypothesis of an idiopathic hypertrophy of the heart or a chronic myocarditis. As we have already said, we consider it impossible to make a clinical distinction between these two diseases, although they are ætiologically and anatomically distinct. We can diagnosticate the enlargement of the heart, its functional disturbance, and the intactness of its valves, but whether the substance of the heart is simply hypertrophied or is studded with the indurations of myocarditis, can only be suspected; it never can be decided with certainty. Arhythmia of the pulse may exist, in spite of the lack of all indurations, and it may be absent in spite of extensive cicatricial formation.

We may often enough find that the autopsy confirms our hypothesis of myocarditis, made from the ætiological factors, from the evidence of atheroma in the external arteries, from the existing cardiac insufficiency, and from certain characteristic symptoms like stenocardiac attacks and sudden death; but just as often, even with extensive clinical and pathological experience, we shall have to admit diagnostic errors and confusion between chronic myocarditis and simple cardiac hypertrophy.

Treatment.—The principles of the treatment of idiopathic cardiac hypertrophy are precisely the same as for valvular disease and myocarditis. We may therefore refer for all particulars to the two preceding chapters, and to the remarks upon the mechanical treatment of circulatory disturbances given at the close of the next chapter.

CHAPTER V.

FATTY HEART.

(*Cor adiposum. Fatty Degeneration of the Heart.*)

Ætiology and Pathological Anatomy.—By the name of "fatty heart" we often mean, at present, two quite distinct conditions of the heart—the one an abnormal deposit of fat in the heart, and the other a fatty degeneration of the muscular fibers of the heart. The first is usually one symptom of great general corpulency. At the autopsy of very fat people we sometimes find the heart entirely inclosed in a thick capsule of fat. The fat is situated chiefly in the external pericardium and beneath the visceral pericardium. It is usually very marked along the course of the larger vessels within the grooves of the heart, but in marked cases the fat also involves the muscular substance. The heart itself is otherwise quite normal or somewhat hypertrophied or dilated. There are sometimes also present sclerosis of the coronary arteries and indurations of myocarditis.

We have already mentioned fatty degeneration of the muscular substance of the heart as a frequent result of valvular disease. In myocarditis and idiopathic cardiac hypertrophy, and in the secondary hypertrophy after chronic nephritis and pulmonary emphysema, we also meet with fatty degeneration. We often find it, as well as fatty degeneration of other organs, in severe acute infectious diseases, in phosphorus poisoning, and in all marked primary and secondary anæmias. Under the microscope we find the muscular fibrillæ studded with little drops of fat, which may be so numerous that the nuclei and the transverse striation of the fibers are quite concealed by them. We often find, besides the fatty granules, albuminous granules, which disappear on the addition of acetic acid ("cloudy swelling" of the cardiac muscle). If the fatty degeneration is of high degree, we can easily recognize it with the naked eye. Beneath the endocardium, especially on the trabeculæ and papillary muscles, we see very fine and delicate yellow points and striæ. With great fatty degeneration, as in phosphorus poisoning and pernicious anæmia, the whole cardiac muscle is manifestly yellow, and also soft and flabby. It is claimed that rupture of the heart may occur as a result of marked fatty degeneration.

In fatty degeneration of the heart the fat comes from the decomposition of albumen in the muscular cells. The occasion of it is probably a defective supply of oxygen, which has either a general cause, as in anæmia and phosphorus poisoning, or a local cause, as disturbed circulation in the heart in heart disease. The details of this are given in the chapter on anæmia (page 928).

Clinical Symptoms.—Fatty degeneration of the heart has no special clinical symptoms. In the conditions under which we know it is apt to occur we can usually suspect it during the lifetime of the patient, but we can not diagnosticate it. We must also mention that the frequently expressed opinion, that fatty degeneration of the heart is the cause of general cardiac weakness, is very often incorrect. In pernicious anæmia there may be quite a strong and a perfectly regular pulse up to death in spite of the most marked fatty degeneration.

We can not say much that is certain in regard to the clinical symptoms of a deposit of fat in the heart. "Fatty degeneration of the heart" always plays a far larger part in popular speech than it does in reality. It is certainly a fact that difficulty with the heart and respiration is very often observed in fat people. Examination of the heart, which, however, is decidedly impeded by the thick panniculus adiposus, often shows in such cases an increase of the cardiac dullness, a small and sometimes irregular pulse, and faint but clear heart-sounds. The disturbance may be very considerable, attacks of angina pectoris and cardiac asthma may come on, and death may follow with increasing dyspnœa and general œdema.

If one has opportunity to make an autopsy in such cases, there will be found no constant, single anatomical change as the cause of the cardiac disturbance, but either idiopathic cardiac hypertrophy (see the preceding chapter), or so-called myocarditic changes with sclerosis of the coronary arteries and the like. Very often there is, of course, a marked deposit of fat upon the heart itself, but the question arises whether this can directly and seriously embarrass the cardiac activity. The fact is that we often have seen similar well-marked cases of fatty heart which during life presented no special cardiac symptoms. There would be more reason in ascribing an unfavorable influence to the fatty infiltration of the cardiac muscle; but here there is probably in most cases a simultaneous atrophy of the muscular tissue.

Therefore we can not associate with the term "fatty heart" any uniform clinical conception. It would be better to speak of the "heart of obesity," that is, of all the manifold injuries to which the heart of obese persons is exposed.

Treatment.—A great part of the disturbance in respiration in fat people depends not upon the cardiac weakness, but on the corpulency itself. The great bulk of the body, and the hindrance to the activity of the respiratory muscles, are very important factors. Treatment directed against the respiratory disturbance must hence chiefly attack the obesity, and thus in many cases we also assist the action of the heart. The detailed description of the hygienic methods of cure to be employed here is to be found in the chapter on obesity (page 996).

In regard to the special treatment of the cardiac symptoms, this does not differ from the rules that obtain in other forms of heart disease.

APPENDIX.

REMARKS UPON THE SO-CALLED MECHANICAL TREATMENT OF CIRCULATORY DERANGEMENTS.

Of late years, particularly since the publication of Oertel's views, so much has been written and spoken about the mechanical treatment of circulatory derangements in heart disease, obesity, and changes in the pulmonary circulation (such as those caused by emphysema, kyphoscoliosis, and the like), that it seems necessary to consider briefly here the chief points of this mode of treatment. There can be no doubt that in all the diseased conditions just named the disturbance of the circulation is one of the main causes of the clinical symptoms. The symptoms dependent upon the venous stasis are repeated in all the diseases which belong in this group, in almost the same way in every case (see their description on pages

295 *et seq.*). Inasmuch, however, as the disturbance may be essentially referred to mechanical influences, above all to the diminution of the difference between the arterial and the venous pressure, and the consequent slowing of the current and abnormal distribution of the blood, it is eminently proper to endeavor by mechanical means, so far as practicable, to improve the circulation. The possible points of attack are as follows:

1. *Diminution of the Body Weight when this has become Excessive from the Deposit of Fat.*—We shall, in a later chapter (page 992), study carefully the injurious influence of obesity upon the circulation. All the measures which lead to the diminution of corpulence, and which are to be carefully discussed later on, may therefore be of the greatest benefit in the treatment of circulatory disturbances in suitable cases.

2. *Diminution of the Amount of Liquid in the Body—"Desiccation."*—This is the point upon which Oertel lays the greatest stress. He believes that he can diminish the amount of blood in the body by withdrawing liquids, and thus lighten the task of the heart and restore the normal circulation. On this view rest the proscription of excessive drinking and the limitation of liquid nourishment, such as soup. We must confess that it is difficult for us to decide upon this point. Numerous well-known physiological experiments prove that the body maintains its amount of blood with great tenacity at a certain constant ratio. Inasmuch as the body is able by means of numerous ways (secretion and diffusion of liquids) to make speedy compensation for variations arising through changes in the amount of water ingested, it is by no means proved that the total amount of blood in patients with circulatory derangements is increased; and if actually there is a retention of fluid in the body (as indeed is certainly the case where œdema has developed), yet the liquid does not collect in the vessels, but in the lymph-spaces of the interstitial tissue, or possibly in the cells of the parenchyma itself. That the total amount of water in the system may be subjected to great variations must certainly be admitted; but that there should be any increase of the labor demanded of the heart, requires that a large amount of fluid should be added to the blood in a relatively short space of time, so that an actual, although extremely temporary, hydræmic plethora should exist. If this process is very frequently repeated, it will surely result in a permanent hindrance to the circulation. However, it is probable that this is the case only in habitual beer-drinkers, and that in such persons the proscription of liquid ingesta is of great value, because the patient is thus kept not merely from water, but also from the injurious and excessive use of beer and wine. Another way in which this limitation of drinking is of still wider practical importance is that a patient who is not allowed to drink while he is eating consequently eats less, and because he is not allowed to drink until after he has finished eating is furthermore, for that very reason, likely to drink less than he otherwise would. What might be called the "psychological importance" of the much-discussed prescription to abstain from liquids ought not therefore to be contested; but in our opinion this is its main value. In lean patients who live temperately the amount of liquids taken does not demand any special attention from the physician.

3. *Strengthening of the Cardiac Muscle and Promotion of Compensatory Hypertrophy.*—That the fulfilling of this indication may be of great importance is certain, and Oertel lays the greatest stress upon inciting the heart to more vigorous contractions by means of suitable bodily exertion, and especially by methodical mountain-climbing, in order by these means to promote as much as possible the development of cardiac hypertrophy. This view is probably fully justified and of obvious utility in many cases of simple cardiac weakness, as seen in anæmia, in convalescence from severe disease, and sometimes perhaps as a con-

genital condition. This may also be true, in many cases, of purely muscular disease of the heart; but as soon as one considers it in relation to those cases of circulatory disturbance where there is actual mechanical hindrance to the circulation, as from valvular lesions, the case seems wholly different. To be sure, one might have an idea that, by increasing the activity of the heart, the hindrance would be more easily overcome. An important consideration, however, seems to us to be that we can scarcely transfer our conceptions with regard to exercise and invigoration from the voluntary muscles to the cardiac muscle. The mechanically dilated heart is minutely regulated by means of an especial reflex apparatus, independently of our volition. We know that every increased demand upon the heart's activity is in most cases directly fulfilled by an increased cardiac effort. Under proper conditions the most marked cardiac hypertrophy may develop in a completely bedridden patient. We must therefore consider carefully whether, in cases of this sort, the further increase of the demands upon the heart is judicious; whether it may not, on the contrary, contribute to a premature exhaustion of the myocardium. It certainly seems to us, and our opinion is fortified by practical experience, that the prescription of increased bodily exertion, such as mountain-climbing, should always be given with great caution, and with consideration of the individual circumstances, if the physician desires to avoid unhappy consequences. We share the belief that a certain measure of bodily exercise is very suitable for patients with valvular and other similar cardiac lesions, but we hold that the benefit lies less in the resultant "invigoration of the cardiac muscle" than in the promotion of the venous circulation occasioned by the motion of the extremities and the deeper inspirations (see page 998). Long mountain trips have a great and unique value for those circulatory disturbances alone which are occasioned by obesity, or associated with simple weakness and slight dilatation of the heart.

4. *Promotion of the Circulation by Massage, Passive Movements, and Gymnastics.*—It can not be doubted that these are suitable means for the promotion of the circulation, especially in the veins. We ought not to overestimate their value, but we are fully justified in the assertion that they may sometimes be employed with decided benefit in circulatory derangements. For particulars as to the carrying out of this method of treatment, we must refer to appropriate monographs.

CHAPTER VI.

NEUROSES OF THE HEART.

1. **Angina Pectoris (Stenocardia).**—Angina pectoris is a group of symptoms which we have already had to mention repeatedly as a frequent complication in different cardiac affections, such as indurated myocarditis, aortic insufficiency, etc. The same symptoms are also seen as a pure neurosis, especially in anæmic persons, or in connection with other nervous diseases, like hysteria, epilepsy, and the psychoses. We know almost nothing as to the exact ætiology of the disease. In quite a large number of published cases excessive smoking has been advanced as an ætiological factor.

The most essential symptom of the paroxysm consists in a severe pain in the cardiac region, shooting up into the left, or, more rarely, into the right shoulder. There is also a general feeling of constraint and anxiety—"præcordial anxiety." The action of the heart is usually somewhat accelerated, either weak and perhaps intermittent, or strong. Respiration is unimpeded. Exceptionally, however, and probably as a result of the pain, it becomes irregular, now hurried and now slow.

The skin during the paroxysm is often pale and cool, while at the end of it there may be profuse sweating. The individual attack lasts sometimes only a few minutes, and sometimes half an hour or more. In many cases the paroxysms return very frequently, almost daily, but in others there may be intervals of weeks or months.

Many theories have been advanced as to the nature of angina pectoris, but none of them have any certain foundation. Since the sensory fibers of the heart rise from the vagus (but partly, perhaps, from the sympathetic also), we usually term angina pectoris a neurosis of the vagus.

The prognosis of the disease is not very favorable. Although life itself is very rarely endangered by the paroxysms, we seldom succeed in permanently preventing their return.

The treatment of the paroxysm is purely symptomatic. Cutaneous irritants, like mustard-plasters to the chest, and foot-baths, are almost always used, but they have but little effect. A subcutaneous injection of one sixth to one third of a grain of morphine (grm. 0·01–0·02) is the best palliative. All the other remedies, like nitrite of amyl, inhalations of chloroform, atropine, coniine, etc., are less reliable. Nitrite of sodium has been especially recommended.

Many remedies have also been used to prevent the return of the paroxysms: arsenic, sulphate of zinc, nitrate of silver, bromide of potassium, quinine, etc. We usually try one of these remedies, but without promising any certain benefit from them. Favorable results are often obtained from electrical treatment—either the application of the faradic brush to the cardiac region, or cautious galvanization of the vagus and sympathetic in the neck, or in the cardiac region. Methodical cold-water cures have also resulted in improvement in some cases of angina pectoris.

Finally, of course, we must pay attention to any underlying diseases, like anæmia and epilepsy, and to the removal of injurious influences which may affect the disease (smoking!).

[In well-marked angina sudden death during a paroxysm is not very rare. In Dr. Arnold, of Rugby, the first attack proved fatal. The patient may, however, live for years—cases of survival for upward of twenty, and one even of thirty, years being recorded. Flint has known recovery to occur. The prognosis depends somewhat on the condition of the cardiac valves and walls; but changes, especially in the latter, may escape detection by any save a very skillful observer. Walshe states that in every one of twenty-four cases he examined during life he found physical signs of changes either in the heart, the aorta, or both. The experience of Balfour and Latham is similar.

With regard to treatment, it is true that morphine, subcutaneously, brings relief; but the duration of the attack is short, the physician is not always at hand, and there are scarcely any circumstances under which it is right to arm a patient with a hypodermic syringe in this any more than in other diseases.

Nitro-glycerine is not only a palliative in most, but also a prophylactic in many cases. To cut short an attack, it can be carried in the pocket; to prevent recurrence, it can be taken twice or thrice daily. The nitrite of sodium seems to act equally well, but has not been so long in use. The nitrite of amyl is serviceable chiefly at the moment of attack. It is put up in glass capsules, one of which can be crushed in a handkerchief and the contents inhaled; it is so volatile that it can be preserved ready for use in scarcely any other way.

The value of the nitrites is greater than the author would seem to allow.]

2. **Nervous Palpitation.**—By "palpitation" we understand the subjective sensation of the movements of the heart. It is usually excited by increased action of the heart, but there is no constant relation between the intensity of the cardiac

pulsations and the subjective feeling of them. We sometimes observe that patients with aortic insufficiency do not perceive the very strong action of their hypertrophied hearts, while in other cases a patient complains of a troublesome feeling of palpitation, although the action of the heart does not appear objectively to be especially increased.

We term cases "nervous palpitation" where the patient complains of palpitation when a physical examination of the heart shows no anatomical change in it. As a rule, in these cases we really have to do with a heart whose action is increased by abnormal nervous influences. In many cases the palpitation arises from slight external causes, which may give rise to little or no palpitation in a healthy person, as, for example, after the slightest mental excitement, after any slight physical exertion, after taking food, after indulging in certain drinks, like tea, coffee, wine, or beer, or in certain positions of the body, as in lying on the left side. Here, then, we have to do with an abnormal sensitiveness of the heart to external irritation, but in other cases there is probably a kind of hyperæsthesia of the patient to the movements of the heart, so that the movements that are of normal strength are felt in a troublesome manner.

The patient rarely complains of continuous palpitation; it usually occurs in more or less sharply defined paroxysms. Very commonly in pure nervous palpitation we have to do with people who, in general, suffer from other nervous, hysterical, and neurasthenic symptoms, or they are anæmic persons, chlorotic girls, etc.; but, on the other hand, nervous palpitation may occur in very full-blooded, "plethoric" people.

The diagnosis of nervous palpitation can be made only when repeated careful examination shows no objective abnormity in the heart. In many cases, as when there are anæmic murmurs, the decision may be quite difficult. We must always pay particular attention to the whole constitution and the general impression which the patient makes.

The prognosis is so far favorable in that the disease is not dangerous. In many cases improvement and final recovery may be effected, but other cases, of course, resist all therapeutic efforts very obstinately.

The treatment must first be directed to improving the patient's general constitution. Anæmic persons are to be given iron, quinine, and strengthening diet. We put full-blooded people, however, on scanty fare, and prescribe for them bitter waters, or a bath cure at Marienbad or Kissingen. Where there is hysteria or neurasthenia, it requires special treatment. We should avoid all the influences which seem to excite palpitation. As a symptomatic indication during an attack we should recommend the patient especially to keep quiet. The use of cold to the cardiac region—cold compresses and ice-bags—often acts beneficially. On the other hand, however, it is to be noted that a tendency to palpitation associated with cardiac weakness may often be allayed by methodical exercise and the invigoration of the cardiac muscle consequent thereupon (see the appendix to the preceding chapter). Among internal remedies we must employ nervines, and in severer cases even narcotics. Among the former we would mention especially ethereal tincture of valerian and bromide of potassium, which have repeatedly done us good service. Digitalis is usually of little value in pure neuroses of the heart, but we may give it as an experiment, say fifteen to twenty drops of the tincture with the same amount of cherry-laurel water.

3. **Tachycardia.**—A peculiar and quite rare neurosis of the heart, tachycardia, consists of an enormous frequency of the pulse, coming on in paroxysms, up to 200 beats and more a minute. We have already mentioned these paroxysms as a rare symptom in mitral and also in aortic disease, but precisely similar attacks may occur as a pure neurosis without any discoverable lesion of the heart. They

have been observed in anæmic and nervous, and also in corpulent persons. In young people the same condition sometimes occurs after diphtheria and other acute infectious diseases (*vide supra*). In men we must consider the possibility of the action of injurious dietetic influences, like drinking and smoking. The individual attack usually begins quite suddenly, by day or by night, sometimes without any cause, but often it is apparently produced by certain exciting causes, especially at times by overdistention of the stomach. The patient feels that the attack is coming, he becomes anxious and restless, and looks pale; but there are not, as a rule, at least according to our experience, any symptoms like præcordial anxiety, dyspnœa, or attacks of faintness. We notice in the heart itself, during the attack, chiefly a great acceleration of the heart-sounds. We sometimes hear indefinite, adventitious murmurs. The action of the heart is often quite regular, but there is not infrequently manifest arhythmia during the attack. Increase of the heart's dullness has been repeatedly observed. In a case of paroxysmal tachycardia, in a patient who had cirrhosis of the liver, we could certainly detect a considerable acute dilatation of the heart in every attack, which disappeared again soon after.

We know little that is definite as to the nature of the attacks. The affection is usually regarded as a temporary paralysis of the vagus.

We may also state here that paroxysmal and constant tachycardia have also been repeatedly observed in anatomical lesions of the cardiac nerves and their centers, in tumors and other affections in the vicinity of the medulla oblongata, and in compression of the vagus in the neck from new-growths, and aneurisms.

The prognosis of tachycardia depends first upon the nature of the underlying disease. We do not know whether a permanent recovery is possible in idiopathic cases, but we can always succeed in improving the condition. The treatment during the attack consists in enjoining complete bodily rest, and in applying ice to the heart. With marked subjective disturbance we should give bromide of potassium, or even, under some circumstances, a small injection of morphine. The best way to guard against the return of the attacks is to give precise hygienic directions, suited to the patient's constitution and manner of life. The continued use of iodide of potassium has sometimes seemed to us to be of service.

SECTION II.

DISEASES OF THE PERICARDIUM.

CHAPTER I.

PERICARDITIS.

(Inflammation of the Pericardium.)

Ætiology.—Pericarditis seldom appears as a primary idiopathic disease. It is usually merely a sequel or a complication of other diseases. Thus, it is observed with particular frequency in the course of acute articular rheumatism, where it appears sometimes alone, sometimes in combination with acute endocarditis. It is not impossible that certain rare cases of apparently primary acute pericarditis belong, from an ætiological standpoint, to acute articular rheumatism—that is, they are excited by the same pathogenic factors which exceptionally attack the pericardium alone, without simultaneous participation of the joints in the disease.

It must be confessed that this view is not actually proved to be correct, although a similar one is also held in regard to many cases of primary acute endocarditis. Cases of secondary pericarditis, unassociated with articular rheumatism, occur, although much less often, in other acute infectious diseases, among which scarlet fever, measles, and pyæmic processes, as well as scurvy and purpura hæmorrhagica, deserve especial mention. In pyæmia the pericarditis is purulent, and in purpura, hæmorrhagic. Among the chronic diseases in the course of which pericarditis sometimes appears, we must mention especially chronic nephritis. Pericarditis has also been observed in a few cases in patients with carcinoma.

A large number of cases arise from an extension of the inflammation from the vicinity. Thus we not infrequently see pericarditis as a result of pleurisy, especially on the left side, and in pneumonia complicated with pleurisy. New growths and ulcerative processes in the œsophagus, in the vertebræ, in the bronchial glands, or in the lungs, also lead at times to perforation into the pericardium and a consequent inflammation. The pericarditis, too, which not very rarely comes on in the course of chronic valvular disease, is probably usually to be regarded as an extension of the inflammation. As has already been mentioned, it is most frequent, according to our experience, in aortic disease, so that we may suspect a direct conduction of the agents of inflammation through the aortic walls into the pericardium. Pericarditis may also develop as a result of myocarditis, abscess of the heart, etc.

Tuberculosis plays a very important part in the ætiology of pericarditis. No small number of apparently primary cases of pericarditis turn out at the autopsy to be tubercular. This apparently comes on in quite an isolated way, or as one symptom of a special localized form of tuberculosis, which we term tuberculosis of the serous membranes. In many cases we can discover the origin of a tubercular pericarditis in the direct extension of a tubercular pleurisy. In apparently primary cases the occurrence of the infection may sometimes be explained by the discovery of a tubercular lymph-gland, which has broken through into the pericardium.

Pericarditis is usually a disease of youth and middle life, but it may also occur in advanced age.

Pathological Anatomy.—Ordinary pericarditis involves the two surfaces of the internal pericardium in either a circumscribed or diffuse manner. Inflammations of the outer surface of the pericardial sac are distinguished as external pericarditis (*vide infra*). The anatomical processes in pericarditis are precisely analogous to those in inflammations of the serous membranes in general, especially of the pleura.

We usually divide pericarditis into fibrinous, sero-fibrinous, hæmorrhagic, and purulent (or ichorous) forms, according to the character of the exudation. The fibrinous and sero-fibrinous forms, with an abundant fluid effusion into the pericardial cavity, are the most frequent, occurring in articular rheumatism, in valvular disease of the heart, etc. Both layers of the pericardium are covered with masses of fibrine, which often show a reticular or villous arrangement (*cor villosum*). Besides that, we find more or less of a fluid effusion which distends the pericardium. The fluid is of a serous nature, and contains more or less numerous flakes of fibrine, and is turbid from the admixture of cells—pus-corpuscles, and, in part, desquamated endothelium. A purulent pericarditis is always the expression of a specific infection of the pericardium. It is seen in pyæmic diseases, as a result of empyema, and in perforation of abscesses, cancers of the œsophagus, etc., into the pericardium. A hæmorrhagic effusion is seen chiefly in tubercular pericarditis. In this we find miliary tubercles, and little cheesy nodules in the inflammatory new growths, besides all the signs of inflammation. The specific tubercular changes are sometimes recognizable with the naked eye, but at other times we have to use the microscope to find them. Hæmorrhagic pericarditis also occurs in general hæmorrhagic diseases, such as scurvy, and in weak and debilitated people, like drunkards.

In long-continued pericarditis the cardiac muscle almost invariably undergoes changes. The heart is usually flabby and dilated, and the muscle often shows fatty degeneration. After the pericarditis has lasted a long time there is often quite a considerable atrophy of the cardiac muscle, which is partly replaced by fat tissue. We have already mentioned the occurrence of pericarditis in connection with valvular disease and degenerations of the myocardium.

In favorable cases of pericarditis we may have a perfect recovery. The so-called *maculæ tendineæ* sometimes remain in the pericardium as residua of a past circumscribed pericarditis. In some cases the pericarditis leads to an adhesion of the two layers of the pericardium to each other, and obliteration of the pericardial cavity (*vide infra*). In many cases a chronic pericarditis finally develops from the acute form, or the whole affection takes a more chronic course from the outset. In this way chronic adhesions of connective tissue arise, and great thickening of the pericardium, but the amount of fluid is usually small. Sometimes the chronic pericarditis is interrupted by an acute exacerbation of the disease.

Clinical Symptoms.—1. *Subjective Symptoms, General Symptoms, and Fever.*—Mild forms of pericarditis may develop, as in the course of an acute articular rheumatism, without causing any subjective symptoms. They are discovered only by a careful physical examination of the heart. In severe cases, however, the pericarditis causes violent subjective symptoms, which of course have in themselves little that is characteristic.

Pain may be felt in the cardiac region, and often in the epigastrium, but it is absent in very many cases. A general feeling of constraint and distress is almost constant in all acute cases of any severity, and so is a feeling of dyspnœa, which may increase to the highest degree of orthopnœa. The patients often complain of headache. In severe cases they become stupid and comatose.

These general symptoms are the direct result of the disturbance of the circulation. Blood pours into the heart slowly, and the diastole is incomplete, because of the increased pressure in the pericardium. Although stasis occurs in the veins, there is less blood in the right ventricle than normal. As a result of this the pressure falls, and the rapidity of the current in the pulmonary circulation diminishes. The left ventricle, also, contains too little blood, and the tension in the medium-sized arteries is considerably lowered. This is the explanation of the patient's dyspnœa and cerebral anæmia. The former is also increased in large pericardial effusions by the mechanical pressure of the distended pericardium on the left lung.

Acute pericarditis is usually associated with fever. This has no special type, and usually keeps at a moderate height—102° to 103·5° (39°–39·8° C.)—but it often exhibits considerable variations. In cases of recovery the fever declines by lysis. Chronic pericarditis may run its whole course without fever.

2. *Physical Signs—Inspection.*—The general hue of a patient with severe pericarditis is pale, but also more or less cyanotic. He has an anxious expression. He lies with the upper part of the body raised, or he sits up in bed. The breathing is usually rapid, labored, and somewhat irregular. The veins in the neck are swollen and prominent. We very often see marked undulating or pulsating movements in the jugular veins, as a result of stasis. The cardiac region seems unusually prominent in all cases with much effusion, and the intercostal spaces there are flattened out. We sometimes detect a slight œdematous swelling of the

chest-wall itself. When there is a large effusion, the action of the heart is only faintly visible, and is sometimes remarkably diffused.

Palpation in the milder cases shows the apex-beat in its normal position and of about normal strength; but if the amount of the pericardial effusion increases, the heart is pushed away from the chest-wall by it, and hence the heart-beat grows weaker until it disappears entirely. In such cases it is sometimes to be felt again if the patient bends forward or lies on his left side. In the rest of the cardiac region we sometimes feel the movements feebly, but they entirely disappear as the effusion increases. In some cases, by laying the hand flat on the chest, we can feel the rub of the rough pericardial surfaces against each other.

The pulse is usually accelerated, and in severe cases it becomes irregular. In every large effusion, as we have already said, the tension and height of the pulse are diminished. In severe cases the pulse sometimes becomes very small and weak, but, when the heart is otherwise normal and strong, it may also remain quite strong—and indeed this condition of the pulse, in contrast to the great weakening of the heart-beat, is sometimes of diagnostic significance. In some cases with a large pericardial effusion we have seen a manifest *pulsus paradoxus*—that is, a diminution or a complete disappearance of the radial pulse on every inspiration.

Percussion shows very characteristic changes if the pericardium is distended by the effusion. The cardiac dullness is then increased, and usually assumes a triangular form peculiar to pericarditis. The obtuse angle of the triangle is found above in the third or second left intercostal space near the left border of the sternum. The lateral boundaries run obliquely to the right and downward to about the right parasternal line, and to the left and downward to the left mammillary line, or beyond. The broad base of the triangle which lies below is usually not to be defined by percussion on account of the adjacent left lobe of the liver. On the border of the dullness we often find a tympanitic resonance due to the retraction of the adjacent lung. The area of the dullness depends, of course, in the first place, upon the amount of the effusion, but we must take special notice that in regard to this the relation is not constant. In old cases of pericarditis especially we sometimes find the cardiac dullness very extensive, while the autopsy detects only a little fluid in the pericardium. This is explained partly by a secondary dilatation of the heart, and partly by a persistent retraction of the lung.

It is an important diagnostic sign of pericarditis that in many cases the still perceptible apex-beat lies within the cardiac dullness, since the pericardial effusion extends farther to the left than the heart itself. It is also worthy of note that the dullness in pericarditis often shows very great changes when the patient changes his position. The dullness is more extensive when the body is erect than when lying down, and when the patient lies on his side it sometimes shows a lateral displacement of several centimetres. The same changes, however, though rarely so marked, also occur in a hypertrophied heart.

The characteristic pathognomonic auscultatory sign of pericarditis is the pericardial friction-rub. This arises during the movements of the heart from the rubbing of the rough and inflamed pericardial surfaces against each other. The friction-rub is absent in pericarditis if the rough surfaces of the two layers of the pericardium are separated from each other by a fluid effusion, or if they can no longer rub against each other from an adhesion of the layers of the pericardium. We usually hear the friction-rub loudest in the neighborhood of the base of the heart, but it may also be heard at other parts of the heart. The quality of the sound is described as scraping, grating, or creaking. The friction-rub may be heard chiefly either during the systole or during the diastole of the heart, but it is in general not often closely associated with the phases of the heart's action. We sometimes find it intermitting frequently, and jerky. The intensity of the friction-rub sometimes varies with the phases of the respiration. It is usually louder on inspiration, but sometimes on expiration. If the patient changes his position, it sometimes alters the intensity of the sound. It is louder when sitting up than lying down, etc. The friction-rub often sounds louder if the stethoscope is pressed firmly against the chest, since in this way the layers of the pericardium are approximated to each other.

The heart-sounds, when the valves are intact, may sometimes be heard as well as the friction-rub, or they may be completely drowned by the loud rub, at least in some parts of the heart. In general, they are weak in every case of pericardial effusion, since their conduction to the ear is impaired. In large effusions where no friction-rub is to be heard, we hear the heart-sounds, especially the first, but only very faintly and obscurely. This condition in connection with the increase of the cardiac dullness is of diagnostic importance. If there is also valvular disease with the pericarditis, the pericardial and endocardial murmurs are sometimes hard to distinguish from each other, but usually the former greatly preponderate.

3. *Sequelæ of Pericarditis.*—A large pericardial effusion may excite special symptoms from pressure on the neighboring organs. Thus we have already said that compression of the left lung must increase the dyspnœa. In many cases we also notice a moderate dullness over the left lower back, from compression of the left lower lobe. In rare cases difficulty in deglutition has been observed as a result of pressure on the œsophagus, and paralysis of the vocal cords from pressure on the recurrent nerve.

In cases of long-continued pericarditis the same sequelæ may develop as in any chronic disease of the heart. The amount of urine diminishes as a result of the low arterial pressure. The venous stasis finally leads to general dropsy and to symptoms of passive congestion in the liver, spleen, and kidneys. We would also state that we have repeatedly met with large effusions in the cavities of the body, especially hydrothorax, without any œdema of the skin. All the symptoms of stasis mentioned, however, are often much less due to the pericarditis itself than to the atrophy and dilatation of the heart which frequently follow it (*vide supra*).

SPECIAL FORMS OF PERICARDITIS.

1. **Pericarditis externa and Mediastino-pericarditis** (*Pleuro-pericarditis*).—By pericarditis externa we mean an inflammation of the external surface of the pericardial sac, which is usually combined with an inflammation of the mediastinal connective tissue and the neighboring pleura, especially over the lingula of the left lung. This form of pericarditis may exist by itself, or combined with internal pericarditis. It is a rare disease, and is most frequently seen as a result of tubercular pleurisy.

The physical signs must differ so much, according to the localization and extent of the process, that we can give few general data in regard to them. There are only a few peculiar signs, which must be noted as characteristic of many cases. In the vicinity of the apex-beat, or at the left border of the cardiac dullness, we sometimes hear a so-called extra-pericardial (pleuro-pericardial) friction-rub. This depends both upon the cardiac movements and upon the respiratory movements. On holding the breath we hear only the murmur due to the pulsations of the heart, while on deep breathing the pleuritic friction-sound is chiefly to be heard. In individual cases there are many modifications, which can not all be mentioned. Another interesting sign, first found by Griesinger and Kussmaul in a cicatricial mediastino-pericarditis, is the so-called *pulsus paradoxus*. This consists of a diminution of the pulse at each inspiration. This condition arises, in part of the cases at least, from the fact that the bands and adhesions of connective tissue at the origin of the aorta mechanically nick into and contract its lumen at every inspiratory movement of the thorax. This explanation, of course, does not suffice for all cases, since the *pulsus paradoxus* also occurs under other conditions, as with large pericardial effusions. In some cases there may be seen a marked swelling of the jugular veins in the neck at each inspiration, at the same time with the *pulsus paradoxus*, since the large venous trunks also undergo a mechanical nicking and constriction at each inspiration. We have ourselves seen a very pronounced slowing of the pulse at every inspiration, in a complicated case of extra-pericardial adhesions (vagus irritation?). We must also méntion that Riegel observed a disappearance of the apex-beat on expiration in some cases where there were bands of connective tissue between the lungs and the outer surface of the heart. At every expiration the bands were stretched more tightly, and hence checked the movements of the heart.

2. **Obliteration of the Pericardial Cavity** (*Adhesive Pericarditis; Adhesions of the Layers of the Pericardium; Concretio seu Synechia pericardii*).—We may have a more or less complete adhesion of the two layers of the pericardium with each other as a result of pericarditis. We can sometimes observe the occurrence of this condition during the course of a pericarditis. Quite frequently, however, we meet with extensive adhesions of the two layers of the pericardium on the living subject, or at autopsies, without being able to gather any history of a previous acute pericarditis. The pericarditis must have occurred here in a chronic way, and without symptoms from the outset.

Even extensive adhesions of the pericardial surfaces may develop and remain entirely without symptoms, and be met with accidentally at the autopsy. In other cases, however, the obliteration of the pericardial sac causes special physical signs and severe clinical sequelæ. Among the first and more important is the systolic retraction of the chest, either limited to the apex or involving a larger area. This is most comprehensible if there is an adhesion of the pericardium with the heart, and also with the chest-wall (Skoda); but we certainly find this retraction at the systole without co-existing extra-pericardial adhesions. It is not, however, an absolutely certain sign of an intra-pericardial adhesion, especially if we have to do with a systolic retraction at the apex alone, since systolic retractions may sometimes occur in other disturbances of the heart's motions; but systolic retractions of the whole cardiac region are, in the majority of cases, a certain sign of pericardial adhesions. The amount of this retraction is often dependent upon the respiration, it being usually more marked on inspiration.

The other symptoms of obliteration of the pericardial cavity are more rare and in their diagnostic significance still more uncertain. Friedreich observed a sudden collapse of the jugular veins at each diastole—the "diastolic collapse"—while they became well filled again at the next systole. He explained this phenomenon by supposing that the conditions for emptying the veins, at the moment of the diastole of the ventricle, were especially favorable, since the chest-wall, which had previously been drawn in by the systole, went back again quickly. Riess described some cases of pericardial adhesions where the heart-sounds had a metallic character from the resonance of the stomach, which had been drawn up. All things considered, we must say that although the diagnosis of pericardial adhesions can be correctly made in many cases, yet the signs given for it are more or less uncertain, since they may be absent in obliteration of the pericardial sac, and they may also be caused by other conditions without such an obliteration.

In the cases of pericardial adhesions which give rise to severe disturbances of the circulation, these are usually not the direct result of the pericardial adhesions, but are due to the secondary changes which develop in the cardiac muscle. Only when extensive extra-pericardial adhesions are also present can there be such a restraint upon the systole of the heart, in a purely mechanical way, as to cause a diminished filling of the arteries, and stasis in the veins. It is usually, however, the secondary atrophy, with fatty degeneration and dilatation of the cardiac muscle, that causes the severe disturbances of the circulation. Such cases give throughout the general impression of valvular disease. Dyspnœa, general œdema, and signs of passive congestion in the liver and kidneys, are the chief symptoms of the disease. It is a striking observation, which other physicians and we ourselves have made a few times, that as a result of obliteration of the pericardial sac, great ascites, sometimes associated with hydrothorax, may develop without any simultaneous œdema of the extremities. In all such cases the diagnosis is usually far from easy. We can sometimes scarcely avoid confounding it with chronic myocarditis, in the absence of all heart-murmurs. If the cardiac muscle remains intact, extensive pericardial adhesions may exist for years without causing the patient the slightest disturbance.

3. **Tubercular Pericarditis.**—Tubercular pericarditis is an important disease clinically, since in many cases it is apparently primary. It may be either quite acute or chronic. The patient falls ill suddenly, or more gradually with indefinite thoracic symptoms, dyspnœa, general weakness, moderate fever, etc. If it is of long duration, there is more or less œdema. When we find on physical examination, in such cases, the signs of a pericarditis, the diagnosis of tubercular pericarditis is probable, if we discover a general "phthisical habit," hereditary predisposition, and also co-existing disease of other serous membranes, especially pleurisy, or more rarely chronic peritonitis. In the latter case the tubercular pericarditis forms one symptom of the so-called tuberculosis of the serous membranes, but, as has been said before, apparently isolated primary tubercular pericarditis does occur (*vide supra*). We have seen such cases repeatedly, especially in old people. In these cases the disease is not easy to diagnosticate. The patient gives one the impression of having heart disease, but the physical signs in the heart are sometimes of a very indefinite nature. Friction-rubs may be entirely absent, on account of adhesions or of large effusions. This leads to confusion with myocarditis, or mitral stenosis. In other cases, of course, all the physical signs of pericarditis mentioned above may be manifest, and a correct diagnosis can be made.

Diagnosis.—From what precedes, it follows that the diagnosis of pericarditis is very easy in many cases, but is very difficult or impossible in others. The most unequivocal sign is the characteristic friction-rub. The practiced ear can often distinguish it from an endocardial sound by its quality. The pericardial sound is a rubbing, grating noise, near the ear; the endocardial is blowing, distant from the ear. The following features may serve as marks of distinction in doubtful cases: 1. We hear the pericardial sounds at first, and also later, over the base of the heart in the vicinity of the pulmonary valve; the endocardial are often loudest at the apex. 2. The pericardial murmurs are not so closely associated with the phases of the heart's action, with systole and diastole, as the endocardial. 3. We find that the pericardial sounds are not transmitted far. A loud rub may be audible at one spot which can not be heard a few centimetres away. Loud endocardial murmurs, however, are audible over almost the whole heart. 4. Sometimes the peculiarity of the pericardial murmur—that it becomes louder when the patient sits up, on pressure with the stethoscope, etc.—may be of diagnostic value. In many cases, too, the loud, functional, so-called anæmic murmurs over the base of the heart may give rise to confusion with pericarditis.

In the cases where pericardial sounds are absent the diagnosis is rendered possible by the characteristic triangular shape of the cardiac dullness, in connection with the character of the apex-beat, the pulse, and the heart-sounds. We have already called attention to the ease with which pericarditis may be confounded with myodegeneration of the heart and mitral stenosis without any murmur. We can not lay down general rules for differentiating these conditions. The more careful the examination, and the greater the personal experience, the more easily can we avoid a false diagnosis.

We have already mentioned the determining factors for the diagnosis of the different forms of pericarditis and their significance.

Course and Prognosis.—Many cases of pericarditis in articular rheumatism, pneumonia, or heart disease, and also many of the rare and apparently primary forms, may recover completely. The disease lasts, in the mild cases, only about a week, in severe cases much longer.

Many cases of pericarditis, however, terminate fatally. The unfavorable issue depends either upon the severity of the primary disease, or upon the intensity of the pericarditis itself. In extensive croupous pneumonia, in valvular disease of the heart, or in severe chronic nephritis, an attack of pericarditis is often the terminal affection—the immediate cause of death. In otherwise healthy people, however, a severe pericarditis with a large effusion may be the direct cause of death, as a result of the impairment of the movements of the heart. The prognosis of every tubercular pericarditis is absolutely unfavorable. The latter can, indeed, run quite a chronic course, but it is hardly ever capable of definite recovery. The prognosis of pyæmic pericarditis is also unfavorable.

In one class of cases pericarditis takes a chronic course from the start, or chronic pericarditis develops from an acute attack. The ultimate prognosis of these cases is usually unfavorable, since the secondary atrophy and dilatation of the heart gradually lead to severe disturbances of the circulation. We have spoken above of the termination of pericarditis in obliteration of the pericardial sac.

Treatment.—Since pericarditis is a severe affection under all circumstances, we must especially see that the patient has perfect rest and care. Extreme caution must be enjoined upon him, especially in the cases where at first the subjective symptoms are slight. We must keep the patient strictly confined to the bed, and not let him leave it even temporarily.

The remedies which are used against pericarditis aim partly at keeping the inflammation in check, and partly at aiding the action of the heart. For the first, the continued application of ice to the cardiac region deserves especially to be recommended. Local blood-letting, ten or twelve leeches to the cardiac region—formerly very often but now more rarely used—may, in otherwise strong and healthy persons, afford great relief in cases with marked subjective symptoms. Painting with tincture of iodine and vesicatories, however, deserve little confidence. Digitalis is our chief means to bring down an accelerated pulse, and to strengthen the heart's action. It is a drug which is most active and most frequently used in pericarditis, and is always indicated when the pulse is rapid and of diminished tension. Of course, the action of the remedy must be carefully watched, as in all cases where digitalis is prescribed. Tincture of strophanthus is also useful. As a palliative, morphine often does indispensable service where the subjective symptoms are marked and the patient is very restless.

If the symptoms are threatening, the question arises whether a large fluid pericardial effusion is the cause of the severe symptoms. In this case the evacuation of the exudation is of course imperatively indicated. The difficulty of forming a correct opinion, however, is very great, because in any individual case it is rarely possible to determine the amount of fluid that may be present. In the first place, we must consider the size of the cardiac dullness and the weakening of the movements of the heart, but both factors may give rise to deception. Hence we always first make an exploratory puncture with a Pravaz's hypodermic syringe. The best point for insertion is, in general, the sternal end of the fourth or fifth [left] intercostal space when the patient is lying on his back. If the exploratory puncture gives a positive result, we make a puncture with Billroth's, Fraentzel's, or some similar trocar. With regard to the details, we will refer to the description of puncture of the pleura. Puncture of the pericardium is always performed by the aid of aspiration. It is less dangerous than might be feared. Even injuries to the heart during the operation have scarcely ever had grave results. The temporary relief to the patient, in cases of successful puncture, is usually very striking, but the permanent results of pericardial puncture are, of course, much less favorable than those of puncture of the pleura, which is chiefly due to the character of the underlying disease. In some cases of purulent pericarditis, drainage of the pericardium has also been practiced after the analogy of the treatment of empyema, but experience on this point is not yet very extensive.

[The experiments of Rotch show that pericardial effusion causes dullness in the fifth right interspace, a sign which he thinks is not produced in cardiac enlargement. So far as is known to the editor, he is the only person who has acted on this observation, and punctured on the right of the sternum for pericardial effusion. In the case referred to, the signs pointed to a very large acute effusion of rheumatic origin. The first puncture was made in the fifth left interspace, but only about an ounce and a half of bloody serum was obtained. The needle was then withdrawn and immediately inserted in the fourth right space near the sternal border with absolutely negative result. The gravity of the symptoms led to two more punctures on the left some days later, one with small though positive result, the other with negative. Absorption and recovery ultimately took place.

The recommendation of the author to make a preliminary puncture with a hypodermic syringe for diagnostic purposes seems unnecessary, just as with pleural effusions.

Roberts has tabulated sixty cases of paracentesis of the pericardium, with twenty-four recoveries.]

If there is a condition of cardiac weakness, stimulants are indicated—strong wine, subcutaneous injections of ether or camphor, or wine of musk. We try to keep up the patient's strength by the best of nourishment.

The resulting conditions of disturbance of the circulation, like œdema, in chronic pericarditis, are treated in the same way as in valvular disease (*vide supra*). Digitalis, in small doses, and diuretics, are the chief remedies.

CHAPTER II.

HYDRO-PERICARDIUM, HÆMO-PERICARDIUM, AND PNEUMO-PERICARDIUM.

1. Hydro-Pericardium.

(*Dropsy of the Pericardium.*)

THE collection of a serous transudation in the pericardial sac, without any inflammatory symptoms in the serous membrane itself, we term hydro-pericardium, or dropsy of the pericardium. Dropsy of the pericardium, which formerly played quite a great *rôle* in pathology, is never a disease of itself, but is always a secondary condition. It may occur in anæmic and cachectic people as a result of hydræmia, but it usually depends upon a local or general venous stasis in the pericardium. In the latter case the hydro-pericardium is one symptom of general dropsy, and hence is found chiefly in heart disease, renal disease, or pulmonary emphysema.

The clinical symptoms of hydro-pericardium are only exceptionally distinct, being obscured by the underlying affection. Large amounts of fluid in the pericardial sac, which may amount to a quart (a litre) or more, must of course impair the action of the heart, weaken the heart-beat objectively, and cause an increase in the cardiac dullness. The distinction from pericarditis is rendered possible by the absence of a friction-rub, but especially by attention to the existence of an underlying disease. In other respects the distinction between a pericardial transudation and an effusion during life is not always easy.

The prognosis and treatment depend wholly upon the nature of the underlying disease. Only exceptionally do we need to puncture, when the exudation is very large.

2. Hæmo-Pericardium.

(*Blood in the Pericardial Sac.*)

In rare cases hæmorrhages occur into the pericardial sac. The source of the hæmorrhage is most frequently an aneurism of the aorta, which perforates into the pericardium. Other causes of hæmorrhage are the bursting of aneurisms of the coronary arteries and rupture of the heart. The latter has been seen after injuries, and also as a result of cardiac aneurism and the cicatricial formations in myocarditis (see Myocarditis). Finally, direct injuries to the heart, especially bullet-wounds, may also cause hæmorrhages into the pericardial sac.

In most cases death occurs in a few moments from compression of the heart, when a hæmo-pericardium comes on. Hence the amount of blood poured out into the pericardial sac is usually not very considerable. Only in the cases where the blood oozes out more slowly can a great distention of the pericardial sac be reached. The diagnosis is only rarely possible. With regard to treatment we can merely note that, in some traumatic cases, the aspiration of the blood has been performed with success.

3. Pneumo-Pericardium.

(*Air in the Pericardial Sac.*)

The entrance of air or gas into the pericardial sac has been observed in rare cases, apart from external wounds, as a result of the perforation of a pyo-pneumothorax, or of some other suppurating process in organs that contain air. Thus cases are known where the rupture into the pericardial sac comes from the œsophagus, as in cancer; from the stomach, in cancer or ulcer; or from the lungs, in tubercular or gangrenous cavities. Since the agents of inflammation enter the pericardium along with the air, a purulent pericarditis almost always develops, besides the pneumo-pericardium, or it may rarely be simply a sero-fibrinous pericarditis.

The most characteristic and striking sign of pneumo-pericardium is the presence of a metallic sound, due to the movements of the heart. Either the heart-sounds themselves, or some existing friction-rub, may acquire a metallic timbre from the increased resonance, or splashing metallic sounds may be produced in the pericardial sac from the movements of the air and the fluid, which may even be heard at a distance from the patient. In regard to diagnosis, however, it is important to know that signs similar to those of metallic resonance in the heart may arise from the stomach, when it is drawn or pushed upward.

In true pneumo-pericardium percussion gives a more or less complete absence of the cardiac dullness. On rod-percussion (see page 269) a metallic sound is sometimes heard, whose pitch may vary somewhat with the phase of the heart's action. If fluid is also present in the pericardial sac besides the air, the dullness caused by this will rise on raising up the patient.

The other symptoms of the disease and the treatment are the same as in a severe pericarditis. The prognosis, however, corresponding to the primary disease, is wholly unfavorable.

SECTION III.

DISEASES OF THE VESSELS.

CHAPTER I.

ARTERIO-SCLEROSIS.

(*Endarteritis chronica deformans. Atheroma of the Vessels.*)

Ætiology.—Atheromatous degeneration of the arteries is chiefly a disease of advanced life, in persons over forty. It is often to be regarded, especially in old people, not as a disease, but as attributable to conditions of senile involution.

Besides age there are also a number of ætiological factors which favor an earlier occurrence and a greater extension of the atheroma. Among these are, first of all, chronic alcoholism; also syphilis, gout, chronic nephritis, articular rheumatism, and chronic lead-poisoning. It is, however, hard to find more positive evidence for the connection between atheroma and the conditions mentioned, although the special connection between alcoholism, and perhaps syphilis and arterio-sclerosis, is made probable by many observations. We must mention that in many families there is a pronounced hereditary tendency to atheroma of the vessels and its results. Men are decidedly more liable to the disease than women.

Pathological Anatomy.—Atheroma is almost exclusively confined to the arteries; only exceptionally do like processes occur in the veins. Among the arteries the aorta is almost always the most intensely and extensively diseased; we also find disease in the iliac and femoral arteries, the brachial, radial, and ulnar, the coronary arteries of the heart, and the arteries of the brain. In some of the other arteries, however, like the gastric artery, the hepatic, and the mesenteric, we very rarely find atheromatous changes.

The atheromatous process is easy to recognize macroscopically. Instead of the normal smooth internal surface, we find more or less numerous irregularities and thickenings on the intima, which appear either more or less gelatinous and translucent, or dense and fibrous, or ossified as a result of calcification, in which case they also feel perfectly hard. In extensive calcification the whole artery is changed to a hard, stiff tube. In many cases we find the surface of the thickenings destroyed—atheromatous ulcers—and covered with masses of thrombi.

Microscopic examination shows that the chief changes are situated in the intima of the arteries. This appears three or four times as thick as normal, partly from the swelling of its elements and partly from the new growth of connective tissue and the deposit of round cells. In the connective-tissue cells of the intima, and in the endothelial cells of its surface, we usually find a marked fatty degeneration, to which the yellowish, translucent appearance of the surface is due. Finally, in the deeper layers there is a complete breaking down of the tissue into a mixture of fat, detritus, and cholesterine crystals, which has given the whole process the name of atheroma [= pulp]. If this destruction extends to the surface, an atheromatous ulcer is formed. In other places, however, it does not reach ulceration, but the superficial layers of the intima become sclerosed, and are finally changed to lamellæ of bony hardness from the deposition of lime-salts. The atheromatous spots on the intima of the vessels often give rise to the formation of parietal thrombi.

The media and adventitia of the arteries also show changes in the later stages of the process. Here, too, we may finally get fatty degeneration and calcification. In other cases, however, there is a marked atrophy of the media.

The immediate result of the atheromatous changes is a loss of elasticity in the walls of the vessels. The ability to resist the blood-pressure is reduced, and this is why diffuse or circumscribed aneurismal dilatations of the vessels so often arise as a result of arterio-sclerosis (see the following chapters).

Another result of extensive atheromatous degeneration of the vessels is an increase of the resistance to the blood-current, and a consequent elevation of the arterial pressure. Furthermore, the loss of elasticity in the coats of the medium-sized and smaller arteries removes an important factor for the propulsion of the blood. The left ventricle, in consequence of these additions to its task, becomes almost invariably hypertrophied in cases of extensive arterio-sclerosis, provided the general nutrition of the patient is still well maintained.

The thickening of the intima in the smaller vessels often causes so marked a diminution of the blood-supply that secondary disturbances of nutrition are not wanting in the various organs. The lumina of the vessels may be still further narrowed, or even completely closed, by the formation of thrombi on such portions of the wall of the vessels as have undergone atheromatous changes. We have already in part learned to recognize the sequelæ which necessarily arise in the various organs, such as indurations in the heart as a result of atheroma of the coronary arteries, and we will return later on to the analogous changes in some other organs, like cerebral softening and certain forms of contracted kidney.

Clinical Symptoms.—In order to decide whether an arterio-sclerosis is present in the living subject, we are of course exclusively restricted to the examination of those peripheral arteries that are accessible to palpation. We must examine, first of all, the radial, brachial, femoral, and temporal arteries. If there is atheroma, we feel the hard and partly bony vessel-wall. In marked cases we have a feeling, especially in the radial, as if we had hold of a goose's neck. We sometimes notice a diffuse dilatation of the femoral arteries. In many cases the marked spiral form of the vessels is very striking, and it is a direct result of the loss of elasticity of their walls and of the increased blood-pressure. The spiral form is most frequently observed in the temporal, brachial, and radial arteries.

Although we can often directly demonstrate atheroma in the vessels mentioned, we must always be cautious in deciding from this that there is also an atheroma of the internal arteries, for the radial arteries often feel very rigid, while the autopsy later on shows little or absolutely no atheroma of the internal arteries. In other cases, however, we find at the autopsy marked atheromatous changes in the arteries of the brain, the kidneys, the heart, etc., although the external arteries during life felt perfectly normal. We see from this how hard it is to make an absolute diagnosis of general arterio-sclerosis.

It is impossible to give a uniform picture of arterio-sclerosis, since its results appear now chiefly in this organ and now chiefly in that, whereby entirely distinct types of disease arise. Hence we must confine ourselves here only to mentioning briefly the most important sequelæ. For the most part, they are described separately in other portions of this work.

In the heart we find a hypertrophy of the left ventricle as a result of the increased resistance to the arterial circulation. This is often apparent during life from the strength of the apex-beat and its displacement to the left, and also from the extension of the area of cardiac dullness to the left. On auscultation, the increased tension in the aortic system is made manifest by the strength of the aortic second sound. The examination of the heart, however, is often rendered difficult by the presence of pulmonary emphysema. On the other hand, we sometimes can not decide how far a manifest hypertrophy of the left ventricle is due to an arterio-sclerosis, and not to other co-existing processes, like contracted kidney. We often find other anatomical changes in the heart besides hypertrophy of the left ventricle. We have already spoken of the important and interesting results of atheroma of the coronary arteries, the formation of the so-called indurations of myocarditis in the heart (see page 308 *et seq.*). Sometimes, from an invasion of the aortic valves by the atheromatous process, we get an insufficiency, or much more rarely a stenosis of the aortic orifice. Finally, we may also mention here that atheroma, especially of the ascending aorta or the arch, is the commonest cause of the formation of aneurism of the aorta.

We have already described the character of the peripheral arteries. The radial pulse is hard and tense, and the wave is either quite large, or, where the tube is very narrow, small. Since the wall of the vessel contracts only slowly, in consequence of its loss of elasticity, the radial pulse is usually sluggish—*pulsus tardus.* This condition is also pronounced in the sphygmographic tracing, which shows a slow ascent, and a still slower descent, of the pulse-curve, and an absence of the elevation in the descending limb of the curve, due to the normal elasticity. The frequency of the pulse is quite different in different cases; it is often rather slow as a result of sclerosis of the coronary arteries (*q. v.*). The pulse is very often irregular as a consequence of changes in the heart. We sometimes find an abnormal delay in the radial pulse, or in the pulse in other arteries, in comparison with the heart-beat, from the lessened rapidity of transmission of the pulse-wave.

Besides the heart, the brain is the chief place in which we observe definite results of arterial sclerosis. The increased tendency to rupture which the atheromatous vessel-walls show, and the co-existing heightened blood-pressure, explain the comparatively frequent occurrence of cerebral hæmorrhages. Cerebral hæmorrhages very often (always, according to some authors) result from little miliary aneurisms, which have formed in the atheromatous cerebral arteries. Atheroma is also the most frequent cause for the formation of foci of softening in the brain, since the arterial changes may give rise to a closure of the cerebral arteries both from thrombosis and embolism. We will later describe in full the clinical symptoms of the affections mentioned.

In the kidneys, too, atrophic processes often develop from the diminution of the blood-supply owing to the narrowed lumina of the vessels, and they lead to a special form of contracted kidney. The origin of the granulated "senile kidney" is in large part due to atheroma of the renal arteries.

Gangrene of the extremities may arise from a plugging of their arteries by thrombosis, or more rarely by embolism. The so-called "senile gangrene" almost always depends upon arterio-sclerosis.

From all this it follows that the type of the disease may be very different in different cases. The symptoms in the vascular apparatus often predominate over all others. The heart, which is simply hypertrophied, or has undergone in part cicatricial degeneration, is finally paralyzed, and then all the symptoms of a chronic heart disease develop—dyspnœa, œdema, etc. If there is also albuminuria, a type of disease is produced which resembles that of contracted kidney. In other cases, however, the symptoms in the brain are especially manifest, either alone or in combination with the other symptoms mentioned.

We must remark, however, in conclusion, that all the results of arterio-sclerosis mentioned may be absent for a long time or altogether. Many people have practically no symptoms at all from their arterio-sclerosis, and reach an advanced age, but we must always consider the possibility of the sudden occurrence of severe symptoms, and make our prognosis accordingly.

There is no question of a special treatment of arterio-sclerosis, since we are not in a position to affect the process by any remedy. In the individual case the treatment is directed according to the symptomatic indications resulting from the sequelæ. Prophylaxis, by avoiding the injurious influences mentioned as ætiological factors, is more important, as this may perhaps prevent, or at least delay, the development of the process.

CHAPTER II.

ANEURISM OF THE THORACIC AORTA.

Ætiology and Pathological Anatomy.—The circumscribed dilatation of an artery is termed an aneurism. The cause of its formation is almost always to be sought in a primary disease of the vessel-wall, which weakens its resistance to the blood-pressure. As we have already said in the previous chapter, it is chiefly arterio-sclerosis which lies at the foundation of aneurisms in many cases. The same factors, therefore, which favor the origin of arterio-sclerosis belong to the ætiology of aneurisms. It is also repeatedly asserted that severe physical exertion plays a part in the ætiology of aneurism of the aorta.

[The occurrence of aneurism in early middle rather than in advanced life shows that too much stress can be laid on atheroma as a cause. That sudden strain often plays an important part in the ætiology can scarcely be doubted, though the cases in which a perfectly healthy aorta yields locally to internal pressure must be very rare. It is highly probable that violent exertion tends to produce changes in the walls of the aorta. The far greater frequency of aneurism in the male sex is notable. Syphilis, gout, alcoholic excess, and lead-poisoning appear to be factors in some cases.]

The size of aneurisms of the aorta varies very much, of course, in different cases. They most frequently are about the size of an apple or the fist; but in rare cases much larger aneurisms are observed. According to their shape we distinguish the more diffuse or spindle-shaped dilatations from the saccular aneurisms (*aneurisma diffusum seu cylindricum, aneurisma fusiforme, et aneurisma sacciforme*). Intermediate forms and combinations of the different forms occur in manifold ways.

As we should expect from its origin, we never find the wall of the aneurism formed of a normal vessel-wall. The intima almost always shows the same changes as are characteristic of arterio-sclerosis, only in a much higher degree. The media, too, is usually changed, and its muscular structure is often fatty-degenerated. The adventitia is usually thickened by chronic inflammatory processes. The media, and sometimes the intima, are in many cases so much atrophied that the wall of the aneurism, at least in part, is formed only of the adventitia.

In the cavity of the aneurism the blood is only partly fluid. We usually find it more or less full of new and old masses of thrombi. The oldest thrombi, which lie upon the wall of the aneurism, are firm, yellowish, adherent to the wall, and sometimes calcified. At other points the thrombi are softened and broken down. The most marked coagulation is usually found in the saccular aneurisms with a narrow entrance, because in this form of aneurism the blood is almost completely stagnant in the aneurismal sac.

Aneurisms of the aorta usually have their seat in the ascending aorta, or in the arch. Aneurisms of the descending thoracic and of the abdominal aorta are far more rare. The following description refers principally to aneurisms at the beginning of the aorta. The other aneurisms will receive a brief separate description farther on.

Clinical Symptoms.—The subjective sensations of the patient, relating directly to the aneurism, are of a very uncertain nature, and are often entirely absent. In other cases there is pain in the region of the aneurism, either only a slight sense of pressure, or very severe and subject to paroxysmal increase. Sometimes, too, the patient feels the beating and pulsation of the aneurism. The remaining symptoms of aneurism may be divided into two groups. The first group embraces those symptoms which are directly related to the aneurism itself, and most prominent in this group are the physical signs. The second group of symptoms includes the resultant phenomena which the aneurism occasions in the circulatory apparatus and by pressure upon the neighboring parts.

1. *Physical Signs.*—It depends entirely upon the position of an aneurism of the aorta whether it causes physical signs or not. Deep aneurisms, which nowhere approach the chest-wall, may, of course, be quite inaccessible to direct examination.

Aneurisms of the ascending aorta, however, and of the arch, often extend so near to the anterior wall of the chest that they cause an abnormal pulsation. We feel this most frequently at the sternal end of the second right intercostal space, or over the upper part of the sternum. The pulsation of an aneurism of the arch of the aorta may sometimes be felt in the root of the neck. It often occurs a moment later than the systole of the heart. In many cases the pulsation is clearly double, analogous to the normal dicrotism of the pulse. We sometimes feel a slight systolic thrill with the flat of the hand. In the rare aneurism of the descending thoracic aorta the pulsating swelling may make its appearance in the back, between the vertebral column and the left scapula. If the aneurism has reached a certain size, the pulsating part protrudes as a tumor. The protrusion is either merely slight, or in many cases it forms a large prominent swelling. It then shows usually a marked pulsation, not only from below upward, but also in a lateral direction, which is of diagnostic significance. In large aneurisms, however, the pulsation sometimes is only very weak, and feels obscure, from the formation of many coagula.

The marked prominence of large aneurisms is possible only because the covering parts, not only the muscles and skin, but also the cartilages and bones, the ribs and sternum, are brought to a gradual atrophy and wasting by the persistent pressure. The skin over large aneurisms gradually becomes thinner and thinner, until finally it may even become necrotic.

In many cases percussion gives a positive result, since the resonance over the aneurism is necessarily more or less dull. The dullness is usually evident in the upper right intercostal spaces, or the adjacent parts of the sternum. Sometimes it even precedes the palpable pulsation, although then its significance is usually still very uncertain. In rare cases of aneurisms of the ascending aorta and of the arch, dullness and abnormal pulsation have been observed to the left of the sternum.

Auscultation gives varying results. In some cases (probably chiefly when many coagula form) we hear nothing at all over the aneurism. In other cases we hear one or two sounds, which are usually the audible heart-sounds transmitted. Perhaps a systolic sound may also arise from vibration of the wall of the aneurism. In other cases, we hear a murmur over the aneurism. A dull and usually not very loud systolic murmur often arises from the formation of eddies in the aneurismal sac. If we also hear a diastolic murmur, it is almost always due to a co-existing insufficiency of the semi-lunar valves of the aorta (*vide supra*).

2. *Sequelæ.*—An aneurism of the aorta by itself probably never causes such an increased resistance to the blood-current as to give rise to the development of a hypertrophy of the left ventricle. In the quite frequent cases where hypertrophy of the left side of the heart exists, it may almost always be referred to a co-existing insufficiency of the aortic valves, and sometimes to very extensive atheroma of the arteries. During life a hypertrophy of the heart may be simulated, because the heart is pushed to the left by the aneurism.

In many cases the signs in the peripheral arteries are important. Marked inequality of the pulse in symmetrical arteries is often an especially valuable diagnostic sign. Either the trunk of an efferent vessel is compressed by the aneurism, or the lumen of the exit of the vessel is itself involved in the aneurism, and hence the opening of the vessel is distorted or contracted, or partly stopped by a coagulum. This readily explains why, in aneurism of the ascending aorta, the radial, and sometimes the carotid pulse, are plainly weaker on the right than on the left, as a result of implication of the trunk of the innominate, while in aneurism of the arch or of the beginning of the descending aorta the opposite condition may obtain. Abnormal differences, too, in the intensity of the pulse in the upper and lower halves of the body may arise under some circumstances.

[W. S. Oliver has described a valuable sign to which he gives the name of "tracheal tugging." In aneurism of the transverse arch with pressure on the left primary bronchus, a distinct downward pull, synchronous with the heart-beat, can be felt by the thumb and forefinger on either side of the inferior border of the cricoid cartilage, when the patient, in the erect or sitting posture and with the mouth shut, raises the chin as far as he can. The late R. L. MacDonnell, of Montreal, has shown that non-aneurismal tumors in this region are not accompanied by this sign, and that pressure on the trachea, from whatever cause, does not produce it. It may be the sole sign of aneurism present, and may then possess the double diagnostic value of indicating both the nature and exact seat of the disease.]

A marked delay of the pulse in the arteries arising below the aneurism is a symptom that is occasionally seen. Thus, we see in aneurism of the arch of the aorta that the left radial pulse is later than the right, and that in aneurism of the descending aorta the pulse in the lower extremities is later than the radial pulse.

We see very striking signs in the veins if the large venous trunks in the thorax, the superior vena cava, or an innominate vein, are compressed by the aneurism. The veins swell in the neck, in the upper extremities, or upon the surface of the thorax, according to the seat of the compression. Local œdema may also be produced in this way.

The respiratory organs are exposed to the pressure of aortic aneurisms in many ways. Compression of the lungs by large aneurisms actually contributes toward increasing the dyspnœa in many cases. This may be still more distressing if the trachea be compressed. Of the two main bronchi, the left bronchus, which lies beneath the arch of the aorta, is more apt to be compressed. This produces the symptoms of a unilateral bronchial stenosis (*vide supra*). The comparatively frequent compression of one recurrent nerve, especially the left, is also of diagnostic importance, as it results in paralysis of one vocal cord. We refer the occasional paroxysms of severe dyspnœa to a compression of the branches of the vagus, but no *post-mortem* lesion has been found.

Very prominent symptoms sometimes arise from compression of the intercostal nerves or branches of the brachial plexus by the aneurism. As a result of this pressure, extremely severe and distressing neuralgias are felt in the nerve territories affected, and sometimes we see motor paresis in the arm.

Finally, disturbances of deglutition arise in many cases from compression of the œsophagus. If this be falsely interpreted, it may lead to a mischievous use of the œsophageal sound. Cases have repeatedly occurred where perforation of the aneurism has been caused by passing a sound into the œsophagus. Hence we must always remember this possibility in practice.

Course and Termination of the Disease.—Aneurisms may remain latent for a long time without causing any symptoms. In such cases a sudden perforation may lead to a speedy and unexpected death.

In the cases which have shown the above symptoms to a greater or less extent for a long time, and often for years, sudden death quite frequently results from rupture of the aneurismal sac and perforation into a neighboring organ. In perforation into the pericardium death follows almost instantly from cessation of the heart's action. In perforation into the œsophagus a fatal hæmorrhage occurs. In perforation of the aneurism into the air-passages, the trachea or bronchi, or into one pleural cavity, two factors, hæmorrhage and suffocation, unite in causing death. In aneurisms which gradually erode the anterior wall of the chest, the perforation is in rare cases external; but here a sudden, immediately fatal hæmorrhage rarely ensues; much more commonly a slowly increasing anæmia develops as a result of repeated slight hæmorrhages which may sometimes go on for weeks. Death then is due to the gradually increasing weakness, or to a final severe hæmorrhage. Perforation of an aneurism into the right side of the heart, into the pulmonary arteries, or the vena cava, is a rare termination. Here death does not follow at once, but severe general disturbances of the circulation, like dropsy, soon arise. In many of these rare cases peculiar physical signs also appear—a venous pulse, a loud systolic murmur over the point of perforation, etc.

If, in patients with aneurism of the aorta, death does not ensue from a sudden perforation, the general type of the disease takes a form similar to chronic heart disease. The aneurism, as we have said, is often also combined with aortic insufficiency. The left ventricle gradually becomes paralyzed, and the well-known disturbances of compensation set in—increasing dyspnœa, œdema, etc. In other cases the patient gradually becomes duller and weaker from the distressing pain, the sleeplessness, and the other symptoms, and dies with the signs of increasing general weakness.

Recovery from aneurism of the aorta scarcely ever occurs.

Diagnosis.—The diagnosis of aneurism of the aorta can in many cases be made with great ease and certainty, but in other cases it is very difficult and even impossible. If the direct physical signs are plain, especially if we feel an abnormal pulsation, we shall not be apt to commit an error; but the diagnosis presents great difficulties in those cases where the aneurism is not accessible at all, or accessible only with great difficulty, where it merely causes indefinite symptoms, pain in the chest, occasional oppression, symptoms of pressure on neighboring organs, etc. A very stubborn intercostal neuralgia, which no remedy can relieve, may be for a long time the only symptom, often misinterpreted, of a latent aneurism. The disease is often not recognized, however, because in such cases we do not generally think of the possibility of an aneurism, and hence we neglect a careful examination of the heart and the arteries, and also the search for other symptoms of compression, like paralysis of the vocal cords; but sometimes, even with the most careful examination, the diagnosis can not amount to more than a suspicion.

The distinction between aneurism and other tumors in and about the thorax sometimes presents difficulties in diagnosis. Mediastinal sarcomata and abscesses, circumscribed empyemas, tumors arising from the sternum, or new growths in the lungs and bronchial glands, may all give rise to confusion. We can scarcely lay down any general rules for diagnosis, since the conditions differ in almost every case. If we feel a swelling, its pulsation is the symptom which points most to an aneurism, but we must be certain that the pulsation is not merely transmitted, but that it really takes place in all directions within the swelling itself. We must also consider the auscultatory symptoms, the condition of the heart and the arteries, and also any symptoms of compression; yet in such cases we can not always make a definite diagnosis.

Treatment.—Many attempts have been made to bring about an obliteration of the aneurism, and thus a recovery. Although the methods of treatment aiming at this have obtained decisive results in the aneurisms of peripheral arteries, their results in aneurism of the aorta are still of a very doubtful character; yet we are always justified in any given case in trying one of the methods recommended.

Persistent compression by a pad can of course be employed only in those cases where the aneurism projects at one part of the chest-wall. The pressure, however, usually causes great pain, and hence is ill borne.

Tying the carotid, the subclavian, or both vessels, has also been repeatedly performed in aneurism of the arch of the aorta, sometimes with apparent success, but oftener without any benefit.

"Acupuncture" of the aneurism (Velpeau) consists in inserting a needle or an iron wire into the aneurismal sac in order to excite coagulation in it. The results obtained by it in aneurism of the aorta are not very encouraging.

Better results are reported from galvano-puncture. Two needles inserted into the aneurism are connected with the poles of a galvanic battery, by which a weak current is passed through the aneurism. Here we must regard the chemical and electrolytic action of the current as well as the mechanical action of the needles.

Injections of chemical substances into the aneurismal sac, in order to produce coagulation, are dangerous, since the coagula caused by them may give rise to emboli. Hence we have abandoned making trial of liquor ferri sesquichloridi and similar substances. We can better recommend injections of ergotine into the vicinity of the sac, two to five grains (grm. 0·1–0·3) of the aqueous extract of ergot dissolved in water or glycerine, injected every day or two. This method was first employed with success by Langenbeck in peripheral aneurisms. Its action depends upon a contraction of the smooth muscles in the wall of the aneurism, caused by the ergotine.

We can expect little action on an aneurism from the use of internal remedies, although favorable results have been repeatedly reported. Acetate of lead, five to ten grains (grm. 0·3–0·6) a day, and iodide of potassium, half a drachm to a drachm (grm. 2–4) a day, are most praised.

The symptomatic treatment of aneurism, which tries to relieve the patient's sufferings, and the dietetic measures prescribed, follow the generally customary principles. In a rupture of the aneurism externally we try to avert the fatal catastrophe by absolute rest, ice-bags, styptic cotton, etc. Treatment is powerless against internal perforations.

[Tufnell's method, so-called, which has given good results in abdominal and peripheral aneurisms, proves sometimes useful in palliating the symptoms and lengthening the course of aortic aneurism. The aim of this method is to diminish the force and rapidity of the circulation, and, if possible, to increase the fibrinous deposit. It is carried out by enforcing absolute rest in the recumbent position, and by limiting the amount of food, especially of liquids. About ten ounces of solid food and eight of liquid are allowed daily, divided into three meals.]

CHAPTER III.

ANEURISMS OF THE OTHER VESSELS.

Aneurism of the Abdominal Aorta.—Its favorite seat is the vicinity of the cœliac axis. In many cases it may be felt through the abdominal wall as a pulsating tumor, over which a systolic sound or a whirring murmur can be heard. The possible symptoms of compression are very numerous. The stomach, intestine, and liver (jaundice) may be implicated. Pressure of the aneurism upon the nerve trunks, or even pressure on the spinal cord after gradual erosion of the vertebræ, with consequent severe neuralgia, paralysis, etc., has been repeatedly observed. Death usually ensues from rupture of the aneurismal sac and internal hæmorrhage.

Aneurism of the trunk of the innominate is rare. Its symptoms are very much like those of an aneurism of the arch of the aorta. If we feel a pulsating tumor, it is usually situated somewhat higher up than the aneurism of the aorta, in the first right intercostal space, or the tumor even extends into the supraclavicular fossa. In rare cases aneurisms of the subclavian and of the carotid have been observed. We have ourselves seen an aneurism of the internal carotid the size of a cherry pressing on the Gasserian ganglion, which caused an extremely severe trigeminal neuralgia lasting for years.

Aneurism of the pulmonary artery may appear as a pulsating tumor in the second left intercostal space. It is usually impossible to distinguish it with certainty from an aneurism of the aorta.

We have already mentioned, in the description of pulmonary tuberculosis, the great importance of small aneurisms of the branches of the pulmonary artery in pulmonary cavities, as a frequent cause of hæmorrhage.

Aneurisms of the arteries of the brain, which are relatively most frequent in the basilar artery and the artery of the fissure of Sylvius (the middle cerebral artery), may cause severe cerebral and bulbar symptoms (see page 693). As has already been mentioned, miliary aneurisms of the cerebral arteries play an important part in the ætiology of cerebral hæmorrhage (*q. v.*).

The symptomatology and treatment of aneurisms of the peripheral arteries belong to the domain of surgery.

CHAPTER IV.

RUPTURE OF THE AORTA.

A RUPTURE of a previously healthy aorta, with fatal hæmorrhage, after violent traumatic influences, has been seen only in a very few cases. In the majority of the very rare cases of rupture of the aorta we have to do with a vessel that is already atheromatous. In some cases a special exciting cause is present, and in others it is absent. We once saw sudden death caused by rupture of the ascending aorta in a young man about twenty-five, who before that seemed perfectly healthy. No trace of atheroma was found; but at the point of rupture there was a slight protrusion and a decided thinning of the wall, which was probably congenital. The formation of a so-called dissecting aneurism, which has often been seen in the aorta, is of anatomical interest. Here only the intima and media are torn. The blood burrows between them and the adventitia or between the layers of the media. Most of the cases of dissecting aneurism of the aorta also result, like rupture of the aorta, in sudden death. In many cases death results from the secondary perforation of the aneurism into the pericardium. On the other hand, a sort of recovery from dissecting aneurism may occur. A secondary perforation takes place into another part of the aorta itself (Boström). Cases of this sort were formerly more than once mistaken for double aorta. If the capsule containing the blood is preserved for a considerable length of time, the symptoms may assume the character presented in ordinary aortic aneurism.

CHAPTER V.

NARROWING OF THE AORTA.

Congenital narrowness of the aorta and its branches is a condition to which Rokitansky first, and later Virchow, have directed attention. We find this anomaly especially in those, mostly women, who during life have shown the signs of persistent chlorosis. Sometimes such persons are backward in their whole development; they retain a puerile habit, and show a defective development of the genitals. They often suffer from palpitation, faintness, and a tendency to hæmorrhages. In many cases the heart is also small, but in others it is dilated and hypertrophied. Valvular disease of the heart has been repeatedly found combined with general narrowness of the arterial system. During life this anomaly of the vascular system may sometimes be suspected, but it can never be recognized with certainty.

Narrowing of the aorta at the point of insertion of the ductus arteriosus is a lesion observed in rare cases, whose origin probably always falls in the period directly after birth, and is associated with obliteration of the fœtal ductus arteriosus. Other congenital anomalies of the heart are usually present at the same time. If the narrowing of the aorta is not very marked, it may be properly equalized by a secondary hypertrophy of the left ventricle and the development of a collateral circulation. The latter is brought about by dilatation of the anastomoses between the first intercostal artery, the dorsalis scapulæ, the subscapularis, and the transversalis colli on one side, and the lower intercostal arteries, which come off from the descending aorta below the narrowing, on the other. Anastomoses are also formed between the mammary and the superior epigastric on one side and the lumbar and femoral arteries on the other. During life the dilated arteries are prominent, in part abnormally distorted, and perceptibly pulsating, especially the dorsales scapulæ, the subscapulars, the mammaries, and the epigastrics. In some cases a systolic murmur has been heard over some of these vessels. The pulse in the arteries of the lower extremities, the femoral and popliteal, is very weak and scarcely perceptible.

In many cases the collateral circulation is so complete that the person affected may feel no subjective disturbance at all, and may attain an advanced age, but in other cases disturbances of the circulation appear sooner or later, and the patient finally succumbs to dropsy. Sudden death from rupture of the heart or of the aorta has also been observed.

DISEASES OF THE DIGESTIVE ORGANS.

SECTION I.

Diseases of the Mouth, Tongue, and Salivary Glands.

CHAPTER I.

STOMATITIS.

(*Inflammation of the Mouth.*)

Ætiology.—Inflammation of the buccal mucous membrane is not infrequently the direct result of mechanical or chemical causes. As mechanical causes we may mention particularly the sharp edges of broken or carious teeth. Chemical irritation may come from highly spiced food, or from tobacco-chewing or excessive smoking. Very intense inflammation is caused by acids, alkalies, and the like, attacking the mucous membrane. Mercurial stomatitis is also of practical importance. It is caused by mercurial poisoning, or not infrequently by the therapeutic employment of the drug. The stomatitis attendant upon the cutting of teeth in children will be discussed below.

In many instances stomatitis comes from a direct propagation of inflammation from neighboring parts. It thus forms a frequent complication of pharyngeal catarrh, and less often of rhinitis.

Infection plays an important part in the ætiology of stomatitis. The local inflammation may be merely part of a constitutional infectious disease, as in measles, variola, and syphilis. Stomatitis is still more frequently a complication of some severe and protracted illness, where the mouth is not properly attended to and cleansed. The bits of food and the mucus quickly begin to decay. Great numbers of fungi and bacteria invade the buccal cavity, and excite inflammation in its mucous membrane.

Scorbutic stomatitis will be considered under scurvy.

Clinical History.—The usual symptoms of an inflammation of mucous membrane—namely, redness, swelling, and increased secretion—are exhibited in stomatitis. The redness is usually most intense on the inside of the cheeks and on the gums. Indeed, we have the special name—gingivitis—for inflammation of the latter. The swelling is best shown by the indentations made by the teeth in the cheeks and the edges of the tongue. The tongue and gums are smeared with mucus. There is often considerable salivation. If the inflammation is more active, we find a purulent coating on a greater or less portion of the membrane. The tongue is almost always thickly coated. If we scrape off a little of the coating and put it under the microscope, we find a great abundance of pavement epithelium, in part fatty-degenerated, pus, micrococci, bacilli, occasionally mold-fungi, and remains of food. White spots made up of epithelium may also be formed elsewhere than on the tongue. Here and there little vesicles appear which burst and leave superficial ulcers.

The local discomfort of severe stomatitis is by no means trifling. There is burning pain, which interferes with taking food, and usually the processes of decomposition occasion a constant bitter or disgusting taste in the mouth, as well as a foul and offensive breath.

The duration of the disease depends on the nature of the immediate cause or the character of the primary disorder. Usually a stomatitis which gets well in one or two weeks is called acute, and a more tedious attack, chronic. The chronic form is seen in topers and inveterate smokers. It may last for years, with the symptoms described above, only milder. (For lingual psoriasis, *vide infra*).

Treatment.—If the inflammation is considerable, the diet must be liquid. Sometimes cold drinks are most agreeable, sometimes lukewarm. Often the pain is relieved by taking from time to time a sip of iced water or a bit of ice; but it may happen that the patient will prefer to rinse the mouth with lukewarm water. The important indication, to keep the mouth as clean and pure as possible, is best met by having the mouth frequently rinsed out with a one- or two-per-cent. solution of carbolic acid, a two-per-cent. solution of chlorate of potash, or one or two teaspoonfuls of a one-per-cent. solution of permanganate of potash to a glass of water. In children who can not do this, the mouth is to be carefully washed or sprayed. If the gums are spongy, they should be painted with a mixture containing equal parts of tincture of myrrh and tincture of rhatany. If there are superficial ulcers scattered about, it is sometimes an excellent plan to touch them lightly with lunar caustic, to hasten their healing.

Chronic stomatitis is often very obstinate, resisting all sorts of treatment for a long time. The first thing is to remove any such injurious agencies as tobacco or bad teeth. It is recommended to swab out the mouth with a solution of corrosive sublimate (1 to 5,000) or of lunar caustic (1 to 30–50). A well-known household prescription is to chew bits of rhubarb.

CHAPTER II.

ULCERATIVE STOMATITIS.

(*Stomacace.*)

Ætiology.—By ulcerative stomatitis is meant a severe disease of the buccal mucous membrane, with superficial necrosis and the consequent formation of ulcers. The abnormal processes are not in all cases identical, and their cause may vary. Still it is probable that infection is the important factor, at least in most cases. The disease has repeatedly been epidemic, chiefly among soldiers in barracks or on a campaign, and among the inmates of jails. It also occurs in children, principally at the time of the second dentition; and in such cases, also, the evidence of contagion and of endemic influences is often very striking. Mercurial stomatitis, if severe, always assumes the form of ulcerative stomatitis. (For the scorbutic form, see page 959.)

Symptoms.—The disease usually attacks the gums of the lower jaw first, gradually spreading thence to neighboring portions of the lips and cheeks. The tongue and palate are generally not very much affected, though often the seat of a simple catarrhal inflammation.

Inspection shows that the mucous membrane in the places mentioned has a thick, soft, purulent coating. The gums are swollen, spongy, and red, and bleed easily. In severe cases the incisors become loose, and may fall out. There is usually profuse salivation. The lymph-glands at the angle of the lower jaw and on the chin are generally swollen. The breath is very offensive, poisoning the air of the whole room.

The local discomfort of the patient is the same as in simple stomatitis, only much worse. It is very difficult to take nourishment. In many cases there are marked constitutional symptoms. The patient feels very weak and languid. There may be moderate elevations of temperature, particularly in children. Now and then severe symptoms of constitutional sepsis have followed the disease.

The course of ulcerative stomatitis is favorable in the great majority of cases. With good treatment and nursing, the ulcers gradually clean up, and at the end of one or two weeks recovery is complete. Exceptionally, the disease may be more chronic. The most frequent way in which recovery is delayed is that the disease extends to the periosteum of the lower jaw, causing necrosis of small portions of the bone, which must be expelled before the patient gets well.

The **treatment** does not differ essentially from that of the milder forms of stomatitis. The mouth must be still more carefully and more frequently cleansed and disinfected. A solution of potassic chlorate (1 to 30) is the favorite mouthwash. Some authors strongly recommend the simultaneous internal administration of this remedy; but we must employ it cautiously in children, as it has repeatedly caused poisoning. For children two or three years old we ought not to give over fifteen to thirty grains (grm. 1–2) in a day.

As to prophylaxis, we should mention that all patients who are using mercury should employ a gargle of potassic chlorate faithfully from the beginning of treatment, in order to prevent the occurrence of mercurial stomatitis. If salivation occurs, the mercury must be stopped.

CHAPTER III.

APHTHÆ.

(*Aphthous Stomatitis.*)

APHTHÆ is a name given by physicians to several entirely distinct diseases. Many doctors call every disease aphthæ in which there are white spots upon the buccal mucous membrane. It is thus frequently confounded with thrush. German mothers often apply the same name (*Schwämmchen*, or fungus) indifferently to thrush and to aphthæ.

There is a special form, known as Bednar's aphthæ. In new-born children white patches are not infrequently found lying symmetrically on both halves of the hard palate near the alveolar processes, and persisting till about the third month. These plaques are not syphilitic, although often thought to be. They are probably merely due to the tongue pressing upon the thin mucous membrane during nursing. Generally they do no harm; but in marantic, ill-cared-for children, they may develop into quite deep ulcers. In that case, repeated cauterization with a five-per-cent. solution of argentic nitrate is required.

The genuine aphthæ are roundish spots upon the mucous membrane, grayish white, and of small size, unless made larger by the confluence of several into one another. They usually have a narrow, red areola. They are most numerous on the edges and dorsum of the tongue and on the frænum, but they also occur on the lips and cheeks. The attempt to remove the white spot with forceps never succeeds, but does cause bleeding. In addition to the genuine aphthæ there are almost always the signs of a common stomatitis, which may be mild or severe. The white spots are in part due to a thickening and opacity of the epithelium, and in part are said to be caused by the formation of a fibrinous exudation, which penetrates the most superficial layers of the mucous membrane.

The disease occurs chiefly in children, and at the time of the first dentition. The child is usually restless, often somewhat feverish, and evidently suffers pain when nursing. Generally there is considerable salivation. The lymph-glands may be a little enlarged. Herpetiform vesicles may appear on the lips. The disease is not rare in adults. Many individuals seem especially liable to it, and very frequently have little, white, and often very painful spots here and there on the tongue or elsewhere in the mouth. These have a tendency to develop into superficial ulcers. They may prove very troublesome, from their frequent recurrence and the hindrance they cause to speaking and mastication.

The prognosis, except as just described, is always favorable. There is usually complete recovery in a week or two. The treatment of children consists in carefully washing out the mouth with cold water, and in administering potassic chlorate. Of a mixture consisting of three parts of potassic chlorate, twenty of syrup, and a hundred of water, we may give a dessertspoonful every two hours. If the spots do not disappear, we can paint them with a five-per-cent. solution of sulphate of zinc, or a solution of borax (1 to 30). If some of the places are especially painful, particularly in adults, we may touch them with lunar caustic, when they usually are soon cured. In the constantly recurring form, treatment sometimes proves almost unavailing. Local cauterization, which unfortunately in such cases is often of no benefit whatever, and the use of a mouth-wash containing chlorate of potash or carbolic acid in weak solution, may be employed, and, furthermore, some physicians strongly recommend the internal administration of iodide of potash. Calomel also appears sometimes to have a beneficial influence.

In conclusion, as to ætiology, infection is a not unlikely cause, if we consider that small epidemics or endemics have repeatedly occurred. Lately attention has been called to the possibility that the milk of cows suffering from hoof-and-mouth disease may be a source of infection. That this may happen seems indubitable; but how frequently it occurs, future observations must determine. The cause of the above-described chronic form is unknown.

CHAPTER IV.

THRUSH.

(*Soor. Muguet.*)

Ætiology.—Weak and artificially nourished children are particularly liable to this disease; but it also attacks adults who are suffering from phthisis, carcinoma, and severe typhoid or typhus fever. In it, grayish-white deposits are developed upon the buccal and pharyngeal mucous membrane. The microscope shows these collections to be fungi; there are a multitude of oval spores, or conidia, and a tangled mass of long mycelium threads. Until lately the fungus of thrush was called *oïdium albicans*, and was held to be identical with the *oïdium lactis* found in sour milk. A more recent investigation of Grawitz has rendered it very probable, however, that the thrush fungus is a budding fungus, and is closely related to the comb fungi (*mycoderma vini*,* or *saccharomyces albicans*, according to Rees). It seems to be a sort of transition form between the budding fungi and the thread fungi, inasmuch as it appears both in a form resembling yeast and also in the form of long thread-like mycelia. At any rate, the thrush fungus is widely distributed, for the development of thrush upon the mucous membrane of the mouth and throat is a frequent phenomenon. Nursing-bottles and the nipples used upon them are probably not infrequently the agents by which the disease is conveyed.

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* Mycoderma vini is a fungus which is found associated with the formation of acetic acid out of alcohol, that is, when alcoholic beverages "sour."

Symptoms.—The mucous membrane of the tongue, cheeks, and soft palate is usually somewhat red and swollen. Upon it we see at first small white spots, which may gradually spread. Microscopic investigations have shown that the fungus develops first in the middle layers of the epithelium. From this starting-point it grows not only upward, but also downward into the mucous membrane. If the growth is abundant, it is easy to scrape off the upper layers, and make a diagnosis by aid of the microscope. In exaggerated cases the growth may even extend from the pharynx into the upper part of the œsophagus and the entrance of the larynx. But we never find thrush in the larynx itself, or the nostrils, or the stomach—briefly, in no place where there is cylindrical epithelium.

As a rule, thrush is accompanied by a more or less severe stomatitis. The fluids of the mouth have an acid reaction. Nursing, or chewing, and swallowing, are painful. Still, it is a question whether the stomatitis is due to the fungus, or whether it prepares the territory for the fungus to settle in. Nursing infants, who suffer from thrush, often have diarrhœa or marasmus at the same time, which latter affections are more probably the cause than the result of the thrush. If vigorous and healthy sucklings are attacked by thrush, the disease is usually quite harmless, quickly vanishing if proper cleanliness is maintained. In sickly children, particularly if bottle-fed, the appearance of the disease is very ominous. In adults, thrush is seldom seen except when there is great general prostration; and it is therefore, to a certain extent, an unfavorable symptom.

Treatment.—To prevent the development of thrush in children, the mouth must, if possible, be wiped out, each time they drink, with a cloth wet in cold water; and if adults are very ill, they require equal attention in this regard. As soon as we see the first traces of the disease, we should touch the parts attacked with a brush wet in aqueous solution of borax (1 to 30) or of sodic carbonate (1 to 20). Honey should not be added to the borax solution, as is often unwisely done. If the thrush has once got a vigorous start in the mouth of marantic children, or of adults suffering from an incurable disease, it must be confessed that we often fail to check its growth.

CHAPTER V.

GLOSSITIS.

(Parenchymatous Inflammation of the Tongue.)

INFLAMMATION of the true lingual parenchyma is rare, although the tongue's mucous surface is frequently involved in the various diseases of the mouth.

1. **Acute parenchymatous glossitis** is the name given to an inflammatory infiltration of the whole or a part of the tongue, usually ending in abscess. The most frequent cause is the sting of a bee or wasp, or it may follow burns or severe cauterization. In the rare instances where it is apparently spontaneous, it is probable that some little wound has afforded ingress to the inflammatory poison.

The symptoms of acute glossitis are very violent in the severer cases. The tongue is enormously swollen, so as sometimes to protrude from the mouth. It has a thick, soft, purulent coating, and often presents excoriations and ulcerations.

The subjective symptoms are very disagreeable. The patient has violent pain. Talking and eating are almost impossible. There is usually catarrhal inflammation of the rest of the mouth. The cervical lymphatic glands are swollen. The salivation is profuse and very annoying. In many cases the tongue swells so much as to cause dyspnœa and more or less suffocation. There is usually fever.

Treatment consists in the employment of ice, which the patient should keep constantly in his mouth, if possible. Very great relief follows deep scarification done in a few places where the swelling is greatest. As soon as fluctuation is obtained, we must give exit to the pus. This is usually followed by a rapid abatement of the discomfort, and complete recovery. It is the exception that the increasing dyspnœa necessitates tracheotomy.

2. **Glossitis Dissecans.**—This is a chronic disease, of rare occurrence and unknown ætiology. It causes the gradual development upon the surface of the tongue of a number of deep fissures and indentations, giving the organ an uneven and ragged look. The pain is due to the frequent presence of excoriations and ulcers in these fissures.

The trouble is not intrinsically dangerous, nor does it need special treatment. We should prescribe cleanliness and the use of some disinfectant mouth-wash. Ulcers, if present, must be touched with lunar caustic.

3. **Lingual Psoriasis. Leucoplacia.** (*Tylosis; Ichthyosis linguæ et oris.*)—This, again, is a superficial disease, the ætiology of which is unknown. It consists in localized hyperplasiæ of the epithelium of the tongue, sometimes conjoined with similar spots upon the cheeks and lips. Usually the tongue gets to look like a map ("*lingua geographica*"). The disease generally lasts years, but causes discomfort only when extraordinarily severe. Still, it may cause a hypochondriac endless anxiety, especially if he takes it to be syphilitic.

This last statement applies still better to a peculiar disease allied to psoriasis. It is called leucoplacia, and affects the mucous membrane of the tongue and mouth. Usually it causes the appearance, on the lateral borders of the tongue, of dull-whitish spots, which have the look of scars, and are generally somewhat notched. As a rule, the cheeks display at the same time similar white spots, which are evidently due merely to thickening of the epithelium. Certain spots may disappear, but are sure to be replaced by others, so that, as far as has yet been observed, the disease must be regarded as incurable. Still, it is not of great importance, for in many cases the local discomfort is very slight. If the indentations along the sides of the tongue become cracked or ulcerated, then there may be great pain. The cause of leucoplacia is not yet known. It certainly is not syphilitic, although the disease is said to be especially prone to attack those who have at some former time been infected with syphilis. This does not apply to all cases. Nor is excessive smoking connected with the disease; we have seen leucoplacia in women. Treatment is, as we have said, usually unsuccessful. Still, thorough cleanliness and good care of the mouth may avert any great discomfort. We may try the effect of painting the spots with a five-per-cent. solution of chromic acid. The chief importance of knowing the disease is to prevent our confounding it with syphilitic affections, and thus to save the patient groundless apprehension and needless mercurialization.

CHAPTER VI.

NOMA.

(*Water-cancer. Cancrum oris.*)

Noma is a gangrene of the cheek, apparently of spontaneous origin, and attacking chiefly feeble and sickly children. The disease is rare. It may be primary, but is usually a sequel of severe diseases, like measles, scarlet fever, typhus and typhoid fevers, and pneumonia. Now and then it has been observed in adults. *A priori*, it is extremely probable that noma is due to some parasitic micro-organism; but the matter has not yet been minutely investigated. It deserves mention that noma is said to occur with much greater relative frequency in moist regions along the coast—for example, in Holland—than among us in Germany.

The disease begins, without any evident occasion, in an insignificant spot of gangrene on the inner surface of the cheek—that is, in the mucous membrane. It is usually situated near the corner of the mouth. Externally, the parts are soon swollen by collateral œdema, and the whole cheek gradually becomes hard and infiltrated. At first, all we see upon the mucous membrane is a dirty-greenish spot not much larger than a silver dime, but soon the whole cheek and the neighboring parts are one mass of gangrene. Bits of dead tissue come away, and foul-smelling ichor flows continuously into the mouth. The collateral œdema may finally pervade that entire half of the face. The neighboring lymph-glands are always greatly swollen.

This condition is almost always accompanied by fever, often reaching or exceeding 104° (40° C.). The general health may indeed for a time be astonishingly little affected; but gradually prostration comes on, or even general sepsis develops, with fever, stupor, and delirium. Frequently lobular pneumonia, which may have a gangrenous character, is produced by the inhalation of sloughing bits of tissue; and often the ichor, being swallowed, excites violent and offensive diarrhœa. The local discomfort is not really very considerable in most cases, compared to the severity of the disease. There may even be no pain felt whatever.

The prognosis is almost always fatal. Death sometimes occurs suddenly from collapse. Sometimes it comes at the end of three or four weeks, from a gradual sinking of the bodily powers. Recovery has been seen in only a few cases: there is a line of demarkation formed, the sloughs come away, and a slow convalescence follows, leaving extensive and usually very disfiguring scars behind.

Treatment must have for its chief object to check further extension of the gangrene, by removing all parts that are already destroyed. Local cauterization with concentrated hydrochloric acid, or fuming nitric acid, or lunar caustic, or chloride of iron, is usually futile. It is probably the best way to remove all the gangrenous portion by means of Paquelin's thermo-cautery. At least in the early stages of noma we may hope something from this method of treatment; but if the case is far advanced, we can hardly expect to accomplish much.

We should also disinfect the mouth as thoroughly as possible. The most efficient means is to syringe it out with solutions of salicylic or carbolic acids, or permanganate of potash, and to dust it with iodoform. We should do our best to maintain the patient's strength.

CHAPTER VII.

PAROTITIS.

(*Mumps*).

Parotitis, or inflammation of the parotid gland, appears not only as a peculiar, primary, infectious disease, usually epidemic, but also as a secondary complication of numerous other severe diseases. These two forms should be considered separately.

1. **Idiopathic, Primary Parotitis** (*Epidemic Mumps*).

Ætiology.—The disease occurs in epidemics that, although not very frequent, may be quite extensive. Here and there a sporadic case is seen. Children and young adults are most liable to it. Nursing infants enjoy a marked immunity, as well as elderly persons. Males are much oftener attacked than females.

There can be no doubt that mumps is a specific infectious disease; but we possess no minute knowledge of the infectious agent. Still, it is natural to suppose that the infectious matter reaches the gland by way of Steno's duct.

Numerous observations support the view that the disease is directly contagious. The period of incubation seems to vary. On the average, it is about fourteen days.

Clinical History.—There may be a prodromal stage of one or two days, with mild feverish symptoms. The disease itself begins with swelling of one parotid gland. The swelling is directly below and in front of the lobe of the ear, which is gradually pushed upward. In the next few days the swelling rapidly increases, and it and the collateral œdema of the cheek and floor of the mouth may become very considerable. The face is much distorted, but often makes a very comical impression, especially as everybody knows how harmless the disease is. In most cases the other gland also swells a few days later.

An abscess hardly ever forms in genuine mumps, nor does the swelling often become very hard. Generally it has a somewhat doughy consistence. The corresponding portion of skin is usually pale and shiny. The submaxillary gland not infrequently swells also in addition to the parotid, and this may occur upon one or both sides of the neck. Penzoldt has observed cases where the submaxillary and sublingual were swollen, but not the parotid. We ourselves have seen a swelling of the submaxillary precede the parotitis.

The local discomfort is moderate in most cases. There is difficulty in chewing, swallowing, and talking. Often quite a severe stomatitis develops, with foul breath.

There often seems to be no fever whatever. Sometimes there may be a moderate elevation of temperature, seldom exceeding 102·5° (39° C.). Only occasionally has there been a case with grave typhoidal symptoms.

Complications.—It is not rare for men to have a swollen testicle, which may be quite painful, but usually subsides in a few days. Resultant suppuration is a rare occurrence. Double orchitis is rare. In boys this complication is much more exceptional than in adults. Some observers have mentioned analogous swellings of the female genitals and mammæ, but this is doubtful.

The prognosis of epidemic parotitis is, as we have said, almost always favorable. The trouble seldom lasts more than a week or ten days, when the swelling goes down, and the patient completely recovers.

The diagnosis is easy. The only thing to exclude is swelling of the lymph-glands, and they never have exactly the same location as the parotid.

Special treatment is hardly necessary. Children should be kept in bed.

Usually some salve, like vaseline, is applied to lessen the feeling of tension. If resolution is tedious, we may paint the swelling with iodoform collodion (1-15) or with tincture of iodine; or we may prescribe iodoform ointment (1-15). If there is orchitis, the testicle must be elevated, as by a suspensory bandage. If the pain and swelling are marked, an ice-bag should be applied.

2. **Secondary Parotitis,** or "*Metastatic Parotitis.*"—This secondary form may be a complication of any grave disease. In most cases it is due to inflammatory agents generated by decomposition of matters in the mouth, which agents reach the gland through Steno's duct. It was formerly the universal belief that the infection was metastatic, being conveyed through the blood-vessels; but it is doubtful whether this does occur. It is probable that the pyæmic form is in many instances thus produced. Secondary parotitis is most frequently observed in typhus and typhoid fevers. It is also seen occasionally in all other severe acute diseases, and in phthisis and carcinoma.

The parotid gland swells, just as in the primary disease. It is, however, much oftener of excessive size, and in the majority of cases suppurates. If one has an opportunity to make an autopsy on such a case of secondary parotitis in its early stages, the cross-section of the swollen gland presents a large number of rather small discrete abscesses. These finally unite to form one larger abscess, which usually discharges outward, or into the external auditory meatus. Sometimes the parotid suffers from gangrenous inflammation, and there is extensive sloughing. If such a case finally gets well, still, as a rule, some permanent injuries have been inflicted: there is facial paralysis, due to destruction of the facial nerve, or deafness, caused by an extension of the inflammation to the middle ear.

The treatment of secondary parotitis is that of any phlegmonous inflammation. We may at first try to scatter the swelling by ice or iodoform ointment, but this usually fails. As soon as fluctuation is detected, the spot must be incised, and a drainage-tube inserted. The prognosis depends chiefly on the nature and course of the original disease.

CHAPTER VIII.

ANGINA LUDOVICI.

The name *angina Ludovici* is applied to a rather rare phlegmonous inflammation of the floor of the mouth. Its starting place seems to be the submaxillary gland, at least in most cases. It may be primary, or a complication of other severe acute diseases.

Angina Ludovici usually begins with swelling in the neighborhood of the submaxillary gland. The swelling rapidly increases, and comes to involve the whole floor of the mouth and the anterior surface of the throat. It causes great discomfort. Talking, chewing, and swallowing are almost impossible. There is usually fever, and in many cases we even find the symptoms of general sepsis. There may be great dyspnœa, due either to compression of the larynx or to œdema of the glottis. The final result in some cases is an extensive sloughing of the soft parts. This has the special name of *cynanche gangrœnosa*. In other cases an abscess forms, and points outward or into the oral cavity. The swelling is sometimes, though seldom, reabsorbed.

The prognosis should always be guarded, for severe constitutional symptoms and a fatal ending are not infrequently seen, particularly if the patient has a weakly constitution. There may also be repeated exacerbations and relapses.

Treatment.—At the commencement of the disease we may make the attempt, in suitable cases, to check the process by local depletion and by ice; but, as soon as suppuration or gangrene begins, the case becomes a surgical one. Now and then the threatening asphyxia demands tracheotomy.

CHAPTER IX.

ANOMALIES OF DENTITION.

(Difficult Dentition.)

THE processes of dentition play so important a *rôle* in the disorders of childhood that we feel obliged to discuss the subject, at least briefly.

The first appearance of any of the milk-teeth usually takes place when the child is seven to nine months old; it may, however, occur either earlier or later than this period. As a rule, the two lower central incisors are cut first; then the upper central incisors appear, a few weeks later, and next the lateral incisors of the upper jaw. In the beginning of the second year come the lower lateral incisors, and almost simultaneously the four anterior molars. The four canine, or "eye" and "stomach" teeth, are cut in the second half of the second year; and last of all comes the eruption of the four posterior molars. The first dentition is therefore completed by the end of the second or in the beginning of the third year, with the development of all the twenty milk-teeth. The accompanying diagram (Fig. 33), after Vogel, represents the order in which the separate teeth appear. In the fifth or sixth year the milk-teeth begin to be replaced by the permanent teeth of the second dentition.

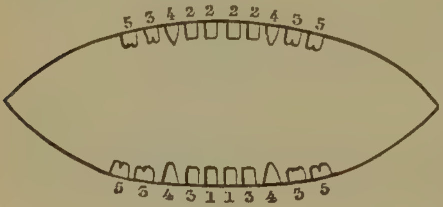

FIG. 33.

"Trouble with teething," however, almost invariably refers to anomalies of the first dentition.

Noticeable delay in teething is frequent in weakly, and particularly in rachitic children. In such cases, sometimes, all the teeth are not cut till the end of the third year.

On the other hand, it sometimes happens that certain teeth appear very early, or even are present at birth. If an abnormally early tooth is only loosely inserted in the gums, it should be removed with the forceps; for it interferes with nursing, and injures the opposing surface of the mouth. But if the tooth is firm in its place, we let it be.

During the eruption of the teeth there is in every child considerable redness of the mucous membrane and an increased flow of saliva. The child evidently feels an itching in the mouth, and therefore a constant desire to bite something. This simple catarrh is sometimes accompanied by a slight rise in temperature. Occasionally there is a severe stomatitis, with which thrush may be associated. These troubles should be treated as already described.

In consequence of the salivation, and the large amount of saliva which is swallowed, in which the various processes of decomposition are apt to develop, we often see gastro-intestinal diseases in teething children. In most children a temporary and mild diarrhœa occurs. We should be particularly careful at this period about the child's nourishment, and in treating any marked gastro-intestinal symptoms. Experience shows also that teething children are unusually liable to simple or even capillary bronchitis, and catarrhal pneumonia.

Nervous disturbances are often referred to dentition. The most important symptom of this kind is eclampsia. The attacks are sometimes called "teething-convulsions." Although the laity go too far in ascribing all sorts of nervous disorders to teething, still experienced specialists do recognize the possibility of such an origin for many cases. Some of the convulsions may in fact be regarded as reflex (*vide infra* the chapter on the convulsions of children, page 779).

When the upper canines, or "eye-teeth," are being cut, there is sometimes a unilateral purulent conjunctivitis, which is perhaps to be explained as an extension of the inflammation by way of the antrum of Highmore and the nostrils.

Eczema and other cutaneous eruptions have been often ascribed to dentition; whether justly, is doubtful.

There is, of course, no special treatment for difficult dentition; and the various disturbances which it may indirectly produce are to be treated on general principles.

SECTION II.

DISEASES OF THE SOFT PALATE, TONSILS, PHARYNX, AND NASO-PHARYNX.

CHAPTER I.

SORE THROAT.

(*Tonsillitis. Angina.*)

Ætiology.—Acute inflammation of the soft palate and tonsils, in its various forms, is one of the commonest of diseases. Almost everybody has had personal experience with it. It is chiefly a disease of early life, being infrequent after the thirty-fifth year. Individual predisposition to it varies greatly. There are persons who have one or more attacks almost every year, while with others attacks are rare and insignificant. In many instances exciting causes have evidently been potent. Chief among these is catching cold; the sufferer has had wet feet, or has been talking in a damp, cold atmosphere. Most cases, therefore, occur in cool weather, although now and then attacks may occur in the hottest days of summer. Again, direct injuries of the pharynx may produce the disease, e. g., the smoky atmosphere of inns, combined with loud talking or shouting; the inspiration of poisonous vapors; cauterization of the mucous membrane with concentrated acids, alkalies, and other chemical agents; and burns.

[Especially when tonsillitis recurs in an individual, or attacks several members of the same household about the same time, it is well to have the drainage carefully examined. The precise connection between bad drainage and sore throats we do not know, but that they are often connected there seems to be no doubt.]

Frequently the inflammation is due to extension from neighboring parts, as in coryza, laryngitis, and stomatitis. In many cases both affections are simultaneous results of one common cause.

Finally, sore throat may be a symptom of many acute infectious diseases, such as scarlet fever, measles, small-pox, and erysipelas. It is also very probable that at least some of the primary cases of sore throat are to be regarded as a special acute infectious disease; but this has not yet been really proved.

To distinguish between an inflammation of the soft palate by itself and an affection of the tonsils is not practicable. In most cases the tonsils are the stronghold of the disease; less often we find the inflammation limited to the soft palate.

Clinical History.—The most important subjective symptom of sore throat, and that by which it is usually first recognized, is the difficult and painful deglutition. The pain is sometimes manifest before any objective changes are to be seen. It may in a severe case be very violent and distressing. The pain has a "darting" character, or sometimes is "burning"; and it is most acute whenever the patient swallows, although in well-marked cases it seldom entirely intermits. Swallowing is not only painful, but it is laborious; it requires more than usual effort and time. The patient feels constantly as if he had to swallow a big lump. This sensation is worse if the tonsils are swollen. It is a matter of experience that not infrequently an "empty" swallowing hurts more than swallowing a liquid or some half-solid substance.

Talking is also difficult. Every word may be painful, so that the patient expresses his wishes as briefly as possible. Even in a mild case, speaking for any length of time will produce a burning pain in the throat. The impaired mobility of the soft palate often prevents the complete cutting off of the nasal passages in talking, so that the voice has a nasal twang; and often it sounds as if the patient were talking with his mouth full: he has the "voice of sore throat."

Further local discomfort results from the mucus and saliva collecting in the mouth. Salivation is not infrequent, probably as a result of the stomatitis usually present. In other cases the patient complains that his mouth feels dry and sticky. Frequently there is a persistent bad taste in the mouth, and the breath is disagreeable.

With these local disturbances, more or less severe constitutional symptoms are almost always conjoined. Indeed, these latter may begin a day or two earlier than the local symptoms. The patient is indisposed, languid, has anorexia and headache. The general disturbance may be surprisingly great in comparison with the slight objective changes in the tonsils.

There is fever in most of the well-marked cases; it may even be quite high. Temperatures of 103° or 104° (39·5°–40° C.), or even higher, are not rare. Sore throat can not be said to have one particular type of fever. Usually the fever appears rather abruptly, remains high for several days, with an occasional slight interruption, and then falls with equal abruptness to normal again.

The entire attack usually lasts only a few days, seldom more than a week. Even where a person is quite ill for several days, convalescence is almost always rapid and complete—that is, if the patient has a good constitution.

Special complications are very infrequent, except that the neighboring parts—the larynx, mouth, and throat—are not seldom involved. Herpes labialis is quite frequent. Beyond this there is nothing to mention.

VARIOUS FORMS OF SORE THROAT.

The symptoms thus far mentioned are much the same in all cases of sore throat, varying only in intensity and duration. But the objective changes to be observed in the soft palate and tonsils are noticeably different in different cases. Whether the ætiology differs also we have no certain information. In some instances it seems probable that it does.

We shall distinguish five chief varieties of acute sore throat. Transitional forms are, however, by no means rare. Genuine diphtheria, which is a specific, acute, infectious disease, and has already been discussed, does not need to be brought up again here.

1. **Catarrhal Sore Throat** (*Simple Catarrhal Inflammation of the Mucous Membrane of the Soft Palate*).—There is a more or less vivid reddening of the mucous membrane, either uniform or in patches. The swelling is most marked

in the pillars of the fauces and the uvula. The surface of the tonsils is likewise reddened; their size may be somewhat increased, or remain unchanged. The mucous membrane of the palate and uvula may be covered here and there with a thin layer of muco-pus, which can easily be wiped off. The tonsils may present small, superficial erosions, scattered about. These little ulcers are apt to lie at the openings of the follicles. The small "blisters" which are often seen on the mucous membrane of the soft palate may be caused in various ways. Either they are mucous glands or solitary follicles, swollen; or, rarely, they are real vesicles filled with a clear fluid and produced by a raising up of the epithelium. The cervical lymph-glands are usually but slightly swollen, if at all.

This is the common and mildest form of sore throat; and it may be over in a day or two. In some instances, however, it causes considerable local and general discomfort; but the disease seldom lasts longer than five to eight days.

2. **Follicular Tonsillitis.**—In this form there is not only more or less catarrhal inflammation of the soft palate, but a decided swelling of one or both tonsils. On the reddened surface of these swollen bodies are whitish-yellow spots, varying in number from two or three to ten or more, and corresponding to the follicles. These spots are often seen to be plugs projecting from the openings of the follicles. It is usually easy to press out the pasty contents of the follicle, represented by the white speck, with a spatula. The microscope shows it to consist of numerous epithelial cells and pus-corpuscles, bacteria, and detritus, and sometimes there are also crystals of the fat acids and cholesterine. The pus-corpuscles may so predominate that we may have small follicular abscesses, which, on being opened, leave superficial ulcers behind. The parenchyma of the tonsil is swollen with a serous and cellular infiltration, increasing the bulk of the part. The trouble is usually bilateral, though often more marked and extensive on one side than on the other. In the severer cases the cervical lymph-glands are swollen.

The clinical symptoms do not differ essentially from those of the other forms. The attack may be mild or severe. Usually the contents of the follicles are discharged after a few days, and the tonsils become normal again. Yet the contents may be retained some time, and become calcified. It is not a rare thing to find such plugs in the tonsils of those who are subject to sore throat. Timid and hypochondriacal individuals are sometimes badly frightened by expectorating these old chalky plugs, which they believe to be "tubercles"! From an ætiological standpoint, follicular tonsillitis is in most cases entirely distinct from genuine diphtheria; still it is noteworthy that precisely at the time of diphtheria epidemics follicular tonsillitis is remarkably frequent. It might, after all, be possible that the mildest degrees of diphtheria present themselves in the garb of an apparently simple follicular tonsillitis. The latter is very probably an infectious disease.

3. **Tonsillar Abscess** (*Parenchymatous Sore Throat*).—In this form the swelling of the tonsils is the most striking symptom. They may be more than twice their natural size. The anterior pillars of the fauces are pushed forward and become convex. The swelling extends so far toward the median line that the tonsil touches the uvula; or, if the affection is bilateral, the two tonsils press against each other, grasping the uvula between them, or pushing it forward. The soft palate is very much reddened, particularly at first. Its surface is usually thickly covered with mucus. If this be wiped off, the mucous membrane is seen to have a moist, œdematous luster. The mucous membrane of the tonsils not infrequently suffers a superficial necrosis. Follicular and parenchymatous tonsillitis are often combined.

In well-marked cases of abscess the local discomfort is usually great. The patient is in a pitiable condition; he can neither talk, nor swallow, nor gargle.

The few words which he painfully utters have in an extreme degree the nasal quality of the "voice of sore throat."

In the milder cases the trouble seldom lasts but a few days before the swelling goes down, and the discomfort and usually rather high fever gradually abate. In other cases, however, a tonsillar abscess forms, usually on only one side. The mucous membrane bulges out more and more in one spot; fluctuation is detected; and, finally, the abscess breaks. With the discharge of the pus the pain is relieved very rapidly, or it may vanish at once. The rest of the tonsil soon regains its former size, and in a few days the patient is well. Relapses are possible, but rare.

Parenchymatous or (as it is called) phlegmonous sore throat, in which the soft palate and not the tonsil is chiefly affected, is infrequent. Its usual cause is some severe external injury, like burns, and cauterizations with concentrated acids or alkalies. The swelling extends deep down into the submucous tissue. The uvula may have the diameter of one's finger. There is intense hyperæmia. Sometimes there are hæmorrhages into the mucous membrane: this is called hæmorrhagic sore throat.*

There are also a peri-tonsillar and a retro-tonsillar abscess, which from a clinical standpoint are not essentially different from the more common form. They are almost always unilateral, and are due to a suppurative inflammation of the peri-tonsillar connective tissue, lying between the tonsil and one of the pillars of the fauces, usually the anterior one.

4. **Necrotic Tonsillitis,** or *Necrotic Sore Throat.*—In this form the tonsils are chiefly involved. The pillars of the fauces and the uvula are but slightly affected with a simple catarrhal inflammation. The tonsils are, as a rule, moderately swollen, seldom attaining great size. The mucous membrane covering them presents a whitish or grayish-white discoloration, often quite extensive, and most marked on the side toward the uvula. These spots are often erroneously said to be a white "coating"; but a more careful investigation shows that there is in reality a necrosis. The process may be superficial; sometimes it reaches quite deeply into the structure of the mucous membrane. It is not possible to pull off this white matter, as one can loosen croupous membranes, although little bits may perhaps be scratched off with a spatula or a pair of forceps. These particles are found, on microscopic examination, to be made up merely of detritus, bacteria, epithelium, and pus-corpuscles. The necrosis is almost invariably confined to the tonsils, and a sharp boundary-line separates it from the reddened and inflamed pillars of the fauces. After a few days the slough may come away, leaving behind an ulcer, which, though usually shallow, has sometimes a considerable depth. This generally cleans up rapidly. In severe cases, however, the floor of the ulcer consists for a number of days of a dirty necrotic material, which comes away only gradually. The worst cases may be properly called "gangrenous tonsillitis."

Necrotic tonsillitis is almost always attended by considerable fever and marked constitutional disturbance. Children particularly seem very ill in the first days of the attack. The cervical glands are usually swollen, but seldom as much so as in genuine diphtheria.

Despite the rather ominous commencement, the disease does not last a great deal longer than the other forms of sore throat. It seldom continues more than five to eight days before a speedy convalescence begins.

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* Another form with the same name occurs where there is violent tonsillitis with necrosis or gangrene. There is also a necrotic, hæmorrhagic sore throat accompanying scurvy, leukæmia, and analogous diseases.

The necrotic tonsillitis is distinguished from the follicular form by the greater area of the white or grayish-white spots. Still it should be particularly noted that sometimes combinations of these two varieties, or transitional forms, occur.

Ætiology.—In our opinion, necrotic tonsillitis is in many instances an entirely different disease from genuine diphtheria. On the other hand, our experience in several epidemics of diphtheria in Leipsic and Erlangen has taught us that in all probability mild cases of genuine diphtheria of the tonsils do occur, and that these objectively resemble necrotic tonsillitis. This is the only way to explain what other physicians as well as ourselves have often observed—namely, that necrotic tonsillitis appears not infrequently in families at the same time with severe cases of genuine diphtheria. Besides, these cases of necrotic tonsillitis are sometimes followed by the characteristic "diphtheritic" paralysis.

[The reader will observe that the author distinctly admits the frequent impossibility of distinguishing between his necrotic and croupous forms of sore throat and diphtheria. It is furthermore stated that the follicular form occurs in combination with either or both of the necrotic and croupous forms, the vague nature of which is thus apparent.

A membrane or membraniform layer which is not confined to the tonsils, or which is seen on the soft palate or pillars of the fauces alone, should be regarded with great suspicion. A few days' isolation can do no harm in such a case, and may save bitter regrets.

Similar deposits limited to the tonsils consist not infrequently of follicular secretion which has coalesced; careful examination will show the follicular origin in these cases.

A protest should be entered against the use of the term "diphtheritic sore throat," so often applied to severe simple inflammations as well as to mild or doubtful cases of diphtheria. A case is either one of diphtheria or it is not. It is our duty neither to excite needless alarm nor to encourage a false security. In doubtful cases the only safe way is frankly to express the doubt and prepare for the worse alternative.]

Diagnosis and Prognosis.—It is never very difficult to recognize a sore throat, and a little practice makes it easy in most cases to decide what particular variety is present, if we examine the objective changes carefully. It is very important in practice to distinguish diphtheria from the benign forms of inflammation. Follicular and necrotic tonsillitis are very frequently mistaken for diphtheria—an error which explains the success of a large number of remedies said to cure diphtheria. Many physicians call every case of sore throat, where there is anything white to be seen, "diphtheria." Certainty in diagnosis of genuine diphtheria can only be gained by practice; no description, however complete, can take the place of personal observation. It may be a help to remember that in both follicular and necrotic tonsillitis the white spots are usually limited to the tonsils, while in croupous sore throat the deposits are generally from the very first also situated upon the pillars of the fauces and the uvula. The white spots of the follicular variety can generally be recognized by their arrangement. The plugs are seen projecting from the follicles. In necrotic sore throat there is never a separable croupous membrane with its characteristic histological structure, but there is simply a superficial necrosis of the mucous membrane and parenchyma. In doubtful cases the condition of the cervical lymph-glands is not unimportant; as a rule, they are much more affected in diphtheria than in the benign cases. Inasmuch as we have seen that the milder forms of tonsillitis may be ætiologically related to diphtheria, it is well in practice to make a guarded prognosis, even in cases of follicular and necrotic angina, particularly in children. And we should always isolate the patient from other children.

Treatment.—These troubles run so favorable a course that active treatment is very seldom needed. The gargle usually prescribed generally gives the patient more discomfort than relief. The most common prescriptions are: Solutions of potassic chlorate (5–10: 300), of alum (5–10: 500), or of borax (10: 300); salt and water; and weak solutions of carbolic acid or of permanganate of potash. To paint the parts is a useless and now almost obsolete proceeding. Inhalations of spray are better, with alum, tannin, or carbolized water. It is beneficial to put a cold wet compress around the throat. Children must be kept in bed, and adults are generally forced to go to bed, if the constitutional symptoms are well marked.

In parenchymatous tonsillitis, ice will sometimes alleviate the pain. Often, however, the patient can not bear it. If there is evident fluctuation, we can make an incision with a spear-pointed bistoury, after guarding a portion of the blade with sticking-plaster. Great relief follows; and, even if there is no abscess, scarification of the tonsils, if they are excessively swollen, usually lessens the pain. The operation causes little discomfort.

We may add that if a person is subject to sore throat, he can lessen his liability to attacks by hardening his skin through the use of cold baths.

CHAPTER II.

CHRONIC HYPERTROPHY OF THE TONSILS.

Chronic hypertrophy of the tonsils occurs not only in those who have had repeated attacks of tonsillitis, but also in cases where no occasion for it can be found. Even in childhood there may be well-marked hypertrophy, which must be due to a congenital predisposition.

The condition is at once revealed by inspection. There may be no signs whatever of any acute or chronic inflammation, or there may be an accompanying chronic pharyngitis. The tonsils bulge out in two great lumps. They may be so large as to touch the uvula on each side. Histologically, there is a genuine hypertrophy of the organ—that is, an increase of all its component tissues.

In many cases, where the swelling is moderate, there is no discomfort. The possessor of the tonsils is not aware that they are enlarged. In other cases the hypertrophy proves of clinical importance, inasmuch as all forms of sore throat are found to occur more frequently if the tonsils are enlarged, and to cause more trouble when they do appear. The hypertrophied organs may also be the seat of a chronic catarrh, which by extension gives rise to chronic nasal catarrh, catarrh of the Eustachian tubes, or hoarseness.

If the hypertrophy is considerable, the local discomfort may be quite marked. Swallowing is rendered difficult, if not painful. Frequently there is evident dyspnœa. The patient has to breathe through his mouth, and sometimes when asleep snores and snorts in a way to frighten one. Children are particularly apt to suffer in this manner. Many instances of *pavor nocturnus*, or "night terrors," in children are referable to this cause. We have already mentioned that cases of bronchial asthma sometimes seem to be connected with hypertrophy of the tonsils (see page 171).

Treatment.—The attempt to reduce the enlargement by applying lunar caustic, tincture of iodine, etc., usually fails. If there is much distress, if the patient is subject to frequent sore throats, or if the hypertrophy of the tonsils keeps up a chronic nasal or pharyngeal catarrh, then the simplest remedy is to remove the tonsils. The operation is free from any danger. The extirpation can be accomplished either with the tonsillotome or with scissors and forceps. The latter way is the simpler, and is almost equally easy.

CHAPTER III.

CHRONIC PHARYNGITIS.

Ætiology.—It is not practicable to distinguish between chronic catarrh of the soft palate and of the pharynx, for, as a rule, the two are combined. Sometimes the condition is the result of repeated acute attacks; sometimes—and probably oftener—it is due to persistent, injurious, local influences. A large number of cases originate in bad habits, or in abuse incident to certain vocations. Examples are seen in smokers, drunkards, singers, preachers, teachers, and men who work out-doors. In talking and singing, the soft palate is strained; or the disease is excited by breathing cold or impure air, or by such chemical irritants as alcohol or tobacco. In many cases, chronic pharyngitis follows chronic rhinitis or chronic laryngitis. The general passive congestion due to cardiac disease or pulmonary emphysema may sometimes promote the development, or prolong the existence, of a chronic pharyngitis.

Symptoms.—The local discomfort is often slight. The patient gets used to it, and does not mind it except when there is some exacerbation. It becomes a more important matter if the calling of the patient is interfered with, as in a preacher, singer, or teacher.

Deglutition is seldom impaired. There is often, however, a constant feeling of dryness, or burning, or scratching in the throat. The patient has to clear his throat frequently, and often acquires an habitual, short, sudden cough, which may be dry. The uvula becomes so long that its tip rests on the tongue or the posterior wall of the pharynx; and this gives rise to a peculiar and disagreeable sensation of tickling. All these uncomfortable feelings are temporarily increased if anything affects the throat unfavorably; and they are generally at their worst on rising in the morning, apparently because the mucous membrane has become dry, or a collection of tough muccus has formed during the night. Every one knows how drunkards have to hem and cough mornings, so that often they almost strangle or vomit.

On inspection, we generally find the mucous membrane reddened. Very often a number of dilated and tortuous veins are visible both on the soft palate and in the back of the throat. Of equal frequency is the appearance of numerous small gray projections, corresponding to swollen follicles or hypertrophied mucous glands. This is called granular pharyngitis. Small follicular ulcers are not infrequent. Exceptionally there are more extensive catarrhal ulcers. The mucous membrane of the posterior wall of the pharynx may present patches of opaque or thickened epithelium, giving the surface a grayish-white appearance.

Frequently chronic pharyngitis is combined with chronic laryngitis, evidenced by hoarseness; or with posterior nasal catarrh, or catarrh of the Eustachian tube, producing deafness and ringing in the ears.

VARIETIES OF CHRONIC PHARYNGITIS.

1. **Chronic Catarrh of the Naso-pharynx, or Chronic Posterior Nasal Catarrh.**—This has the same ætiology as the ordinary form. It is practically important because the nose and ear are frequently involved.

The anatomical changes are essentially those already depicted under chronic pharyngitis. The region affected can not be seen by direct inspection, so that accuracy in diagnosis requires the use of a nasal speculum (see particulars in the works mentioned on page 125). The ordinary examination of the throat may reveal a condition which is quite characteristic of posterior nasal catarrh: a collection of muco-pus, or of firmly adherent dry crusts, rests upon the posterior wall of the pharynx, and can be seen to extend upward toward the naso-pharynx.

The local discomfort is somewhat similar to that experienced in chronic pharyngitis. There is a scratchy feeling, or a feeling as if there were a foreign body in the back of the throat, accompanied by a constant desire to blow the nose, hawk or cough. Dried and decomposing secretion often causes extremely foul breath. There is often also vertigo, and occipital headache.

In many cases the nostrils are stopped up. The posterior opening of the nostrils is closed in part by the swelling and hypertrophy of the mucous membrane, and in part by the accumulated secretions. The patient, therefore, usually has to breathe through the mouth. The ear is frequently involved. The catarrh extends into the Eustachian tubes and the tympanic cavity, or the opening of the tubes is occluded with the secretions. For a detailed consideration of the deafness, tinnitus, etc., thus produced, consult works on otology.

2. **Pharyngitis Sicca**, or "*Dry Atrophic Catarrh of the Throat and Naso-pharynx.*"—This name is applied to an atrophy of the mucous membrane, which sometimes is spontaneous and sometimes is a sequel of chronic pharyngitis. The whole mucous membrane of the pharynx and the naso-pharynx (seen with the rhinoscope) seems pale, smooth, and perfectly dry, and has a peculiar luster, as if varnished. Here and there tortuous veins project from the general anæmic surface.

If an opportunity is afforded to examine the mucous membrane microscopically, it will be found that the atrophy involves all the elements of the tissue, though the follicles and mucous glands suffer most.

This condition may not cause any symptoms, but, in many cases, the patient suffers constantly and considerably. The chief trouble is a feeling of dryness in the throat, rendering deglutition difficult or even painful. There is also a constant desire to clear the throat. The secretion hawked up may be scanty and tough or more abundant, and it is often tinged with blood. Actual coughing may also be due to pharyngeal trouble ("throat-cough"). Talking is often rendered difficult, the voice grows weak and it becomes easily tired. In severe cases there is considerable general debility. Not infrequently pharyngitis sicca is associated with atrophic rhinitis (q. v.), but it occurs also in cases where there is no nasal disease.

The disease is most frequently seen in the elderly, but it also occurs in children and young persons. It is especially common in ill-nourished individuals or in those who are suffering from such diseases as tuberculosis or chronic nephritis.

3. **Hypertrophic Catarrh of the Pharynx and Naso-pharynx.**—An opposite condition of hypertrophy sometimes results from chronic catarrh. The changes consist mainly in hyperplasia of the lymphatic tissue, and they are usually termed "adenoid growths." The choanæ and the posterior extremity of the nasal septum may be almost completely hidden by these growths, as they extend down from the roof of the pharynx in grayish-red, uneven masses. In many cases the hypertrophy seems to originate chiefly in Kölliker's "pharyngeal tonsil."

The adenoid growths are especially common in childhood. The symptoms consist in a change of the voice (which loses its reverberating quality and becomes nasal), frequent snuffling and hawking, and a tough mucous secretion, often tinged with blood. Not infrequently there is headache. Of greater importance is ear-trouble, which is often occasioned by the growths.

An accurate diagnosis requires rhinoscopy. Positive results are also often obtained by palpation. The index-finger, being passed backward and bent upward, can touch the protuberances and the enlarged pharyngeal tonsil in the naso-pharynx.

Prognosis.—The prognosis in all forms of chronic pharyngeal catarrh should be somewhat guarded, for all severe cases are very obstinate and can seldom be permanently cured. Success may be depended upon only in cases where all unfavorable influences can be completely removed. We may afford great relief, but there will be a persistent tendency to acute exacerbations and to relapses long afterward.

Treatment.—Many of the milder cases never apply to a physician. The patient uses some domestic remedy or gargle, or becomes so accustomed to the disagreeable sensations that he does not consider it necessary to do anything in particular about them.

The treatment of a well-developed case requires great patience and persistence on the part of all concerned. If there is some underlying disease, such as pulmonary or cardiac disease, that must be treated. All exciting causes must be avoided. Energetic local treatment is also indispensable. This has been greatly elaborated by specialists, and for the many details we must refer to their writings; but the following remarks will meet the requirements of ordinary practice:

Gargles are seldom satisfactory, for they never reach farther than the soft palate. Inhalations are better; we can use solutions of alum or tannin, or, in mild cases, of common salt. Still more efficient is the painting of the entire surface of the pharynx with some concentrated solution. The physician usually has to perform this, although some patients learn to do it for themselves. Proper solutions are: Argentic nitrate, five or ten per cent.; tannin, eight to twenty per cent.; tincture of iodine, either pure or diluted; or iodized glycerine, composed of pure iodine, parts 1·5; iodide of potassium, 5; glycerine, 500. These applications must reach all the diseased surface. If the naso-pharynx is involved, the brush must accordingly be bent upward, to reach that region. For this a mirror may be needed. It is very important to make the applications to the mucous membrane itself, freed from any interposing secretions.

In the treatment of chronic posterior nasal catarrh the nasal douche (*vide* Diseases of the Nose) plays an important part. It should be used two or three times a day. It not only removes the collected secretions, but is a means of making local applications. The instrument is merely a fountain-syringe. The nozzle must be of a size to fill the nostril completely. The force of the current should always be moderate, and the patient's head should be sharply flexed forward. The fluid used—the best is a one-per-cent. solution of sodic chloride or bicarbonate—must have about the temperature of the body. Other medicated solutions must be very weak, such as sulphate of zinc, 1 to 1,000.

The insufflation of powders into the throat can be made through any small glass tube, three to six times a week. Alum or tannin may be used, either pure or mixed with equal parts of pulvis gummosus [P. G., made of gum-arabic, three parts; licorice-root, two parts; and sugar, one part]. For the naso-pharynx, a bent tube of glass or hard rubber is to be introduced through the mouth. There are numerous "insufflators" to be had at the instrument-makers.

Many baths enjoy a great reputation for the cure of chronic pharyngitis. Besides Ems, there are Reichenhall, Kreuznach, Salzungen, the cold sulphur springs, such as Weilbach, and many others. Good results are also achieved in Kissingen and Marienbad, if these places are favorable to the patient's general constitution.

In pharyngitis sicca, the nasal douche with a one-per-cent. salt solution is to be recommended. It is sometimes also beneficial to paint the parts with solution of argentic nitrate, iodized glycerine, etc. Many irritating influences which do harm in common pharyngitis seem sometimes actually to benefit this form—such as smoking and taking snuff.

In treating the hypertrophic forms of pharyngitis and the adenoid growths, cauterization with nitrate of silver suffices for the milder cases alone. A thorough and permanent cure can be achieved only by removing the growths with the galvano-cautery.

[To Hooper, of Boston, belongs the credit of showing that a radical operation for the removal of adenoid growths of the naso-pharynx can safely be done under anæsthesia. The blood does not, as was feared, run into the air-passages, but either into the stomach or outwardly. Anæsthesia thus renders it possible to scrape away with the finger or tear away with forceps the hypertrophied tissue at one sitting, and to do it in the child before secondary changes have taken place in the facial expression, the jaw, the ears, or the form of the chest.]

CHAPTER IV.

RETROPHARYNGEAL ABSCESS.

RETROPHARYNGEAL abscess is formed by a suppurative inflammation of the connective tissue lying between the posterior wall of the pharynx and the spinal column. It is a serious disease, although a rare one. If unrecognized, it proves fatal in many instances; while, if a correct and timely diagnosis is made, the patient can usually be easily cured. It is commonest in childhood, and before the second year. It almost always appears as a primary, acute disease, without any special cause being evident. Probably the agents which excite the inflammation penetrate into the tissue from the pharynx. The idea that the inflammation originates in the small lymphatic glands which lie in front of the vertebræ lacks proof as yet.

The disease attacks not only weakly children, but those who have been perfectly healthy and vigorous. The child grows restless and fretful, and does not nurse well. Apparently, deglutition soon becomes painful, but one can not be certain about this except in older children. Generally, the respiration quickly takes on a peculiar stertorous character, particularly during sleep. Mucus collects in the mouth and throat. Upon swallowing, there is often regurgitation through the mouth or nose, or some of the food gets into the windpipe and causes violent coughing. The lymph-glands of the jaws are usually somewhat swollen, and the neighboring parts may seem slightly œdematous. After a week or two the dyspnœa gradually increases. Respiration becomes more and more laborious, with loud rattling, and the signs of stenosis. The jugular veins become distended, the lips cyanotic, and portions of the thorax are retracted during inspiration. The voice is feeble, and may be hoarse and indistinct.

The correct interpretation of these symptoms, which are common to various disorders, requires a careful examination of the throat. It must be confessed that this has its difficulties in an infant. Still, we can sometimes see distinctly a swelling in the posterior wall of the pharynx. This may be either in the median line or on one side. All doubt is removed by digital examination, in making which, however, we must insert a wedge between the teeth, to avoid being bitten. The finger detects fluctuation.

The diagnosis once established, the abscess must be opened at once. We should not delay, even if the dyspnœa has not yet become extreme. To use the finger-nail for the purpose, as has been recommended, is permissible only in an emergency. As a rule, incision is made with a bistoury, of which all but the point is guarded with sticking-plaster. The left index-finger is placed upon the abscess, and used as a guide. Meanwhile, the child's head is kept upright, and, as soon as the cut is made, bent over forward. The pus pours out in abundance. It is advisable to syringe out the mouth repeatedly with lukewarm water. The threatening symptoms vanish almost instantly upon the escape of the pus. Exceptionally, the abscess refills and requires a second incision.

If the trouble is not correctly diagnosticated, or if the abscess is not opened promptly, the patient may suffocate. Or the abscess may burst spontaneously; then there is either speedy recovery, or asphyxia from the pus filling the larynx. In some instances, where a retropharyngeal abscess has not been properly treated, the pus has gravitated far down into the neck and posterior mediastinum. The recognition and incision of the abscess may prove very difficult if from the start it is situated lower down in the throat than usual.

Analogous to this acute idiopathic abscess of which we have been speaking is the chronic abscess due to caries of the cervical vertebræ. This should not be opened unless there is danger of asphyxia.

Retropharyngeal abscesses sometimes occur in pyæmia or other severe acute infectious diseases, but have hardly any interest except to the pathologist.*

SECTION III.

DISEASES OF THE ŒSOPHAGUS.

CHAPTER I.

INFLAMMATION AND ULCER OF THE ŒSOPHAGUS.

Ætiology and Pathology.—The various forms of œsophageal inflammation and ulceration are not of very great clinical importance. The processes are seldom of a severe grade, or, if so, they are generally a part of some complicated disease, to which they seldom contribute prominent symptoms. Very likely the milder forms of inflammation occur frequently, but the symptoms are hardly ever characteristic.

A simple catarrhal inflammation of the œsophageal mucous membrane may be caused by swallowing substances which are injurious mechanically, chemically, or from their temperature. It may also occur in the general infectious diseases, such as typhoid and typhus fevers, and the acute exanthemata. Any inflammation of neighboring tissues may extend into the œsophagus. Chronic catarrh is seen in heart disease, from the passive congestion. It is also found in the vicinity of other chronic œsophageal diseases, particularly cancers and diverticula (*vide infra*).

The acute catarrh is distinguished by not having the usual increase of secretion. The epithelium grows spongy, as a rule, and is cast off more rapidly than usual, so as to suggest the name of a desquamative catarrh. It is in only a few cases that the scanty mucous glands become swollen and look like papules upon the surface of the membrane; this form is called follicular catarrh. In limited areas the desquamation may be complete, giving rise to small catarrhal erosions. Likewise, the swollen follicles may break down into small follicular ulcers.

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* Tuberculosis of the pharynx is spoken of in the chapter on pulmonary tuberculosis, p. 226. New growths in the mouth or pharynx belong to the domain of surgery.

In chronic catarrh there is a moderate increase in the secretion of mucus, and a marked thickening of the epithelium. In very protracted cases actual papillomata may finally be formed. In some cases ulcers are seen.

Croupous and diphtheritic inflammations of the œsophagus are very rare. We have already said that the specific pharyngeal diphtheria frequently extends into the larynx, but only exceptionally into the gullet. Still, we have ourselves seen in a child a stricture in the upper third of the œsophagus, which was said to have been a result of a severe attack of diphtheria. Isolated cases of diphtheritic œsophagitis have also been seen in connection with severe infectious diseases, such as typhus, typhoid, small-pox, cholera, pyæmia, and pulmonary tuberculosis, as well as in the course of Bright's disease and cancer. In variola it is not unusual for pocks to appear upon the œsophageal mucous membrane.

A purulent, phlegmonous œsophagitis now and then attacks the submucous layer. It may be either diffuse or circumscribed. The mucous membrane is dissected up from the muscular layer by the pus, and pushed inward, so as to diminish the lumen of the œsophagus more or less. Most of the cases end by the discharge of matter into the tube, when complete recovery may ensue. If the mucous membrane, however, has been extensively undermined, Zenker states that a fissure-like cavity may be left, even after healing has taken place. Its walls grow smooth, and finally acquire a layer of fresh epithelium.

Purulent œsophagitis is caused either by the presence of foreign bodies in the œsophagus, or by purulent inflammation in neighboring parts, as in glandular abscess, vertebral abscess, or laryngeal perichondritis. It has now and then resulted from the action of concentrated acids and the like upon the mucous membrane.

The action of corrosive poisons (corrosive œsophagitis) is to cause necrosis of the tissues, which in its turn produces inflammation. The inner surface of the œsophagus is converted into a rotten, hæmorrhagic, sloughing mass, of a dirty gray or almost black color. The muscular layer itself may be partly destroyed. If death does not occur speedily, the necrosed portions come away, leaving extensive purulent ulcers behind. These, if they heal at all, cause large cicatrices and stenosis.

Symptoms.—The milder cases, as we have stated, produce almost no distinctive symptoms. Possibly there may be pain along the œsophagus, or at some one point in it, during deglutition. In a more severe case the pain may be great; but the other symptoms are usually too grave for this to excite special attention. Laborious deglutition, and the feeling as if the food were inclined to stick in the throat, result from implication of the muscular layer. A diagnosis of the particular form of œsophagitis is attainable only when the ætiology guides us to it.

Treatment must be purely symptomatic. No solid food should be taken. The pain is to be allayed by bits of ice, or by morphine.

CHAPTER II.

DILATATION OF THE ŒSOPHAGUS.

1. Diffuse Dilatation.

DIFFUSE, spindle-shaped dilatation of the œsophagus is observed as a result of stricture of the cardiac orifice. At first the muscular coat hypertrophies as the orifice contracts, and is able to overcome the obstruction, so that there is no dilatation; but as soon as the muscles are paralyzed, and food collects behind the stricture, the dilatation begins and keeps on increasing. The ectasis is greatest at the lower end of the tube, as is natural from its mode of origin, and gradually diminishes upward.

There have been a very few well-substantiated instances of this diffuse spindle-shaped dilatation, without any demonstrable stenosis of the cardiac orifice. Their cause is unknown. Sometimes the walls of the œsophagus may have been rendered more yielding and less contractile by a precedent inflammation or other disorder. In other cases, some chance bend or distortion of the lower end of the œsophagus may have produced a mechanical obstruction. In some cases the exciting cause is said to be a blow on the chest, or the lifting of a heavy weight.

The symptom of this condition, when well developed, is a chronic difficulty in deglutition, lasting perhaps for years. The patient himself feels that most of the food he eats does not reach the stomach, but lodges higher up. Usually the food is soon afterward vomited, or rather gulped up. When there is stenosis of the cardiac orifice, the explanation of these symptoms is easy. It is much harder to explain the almost equal dysphagia where there is dilatation without stenosis. Sometimes a localized bulging of the wall causes the food to collect in that spot, and thus to obstruct the lumen. As is to be expected, the partial or complete hindrance to the ingestion of food results in marasmus.

If there is stenosis, it can easily be detected with the œsophageal sound, and all the symptoms thus explained. In the rare cases, however, of diffuse dilatation without stenosis, the use of the sound does not give us so much information. If the instrument passes readily into the stomach, we may safely exclude stricture; but, in one case of our own, we made an erroneous diagnosis of a diverticulum, because the sound sometimes glided readily into the stomach, and sometimes could not be passed. A pocket must have been formed at the lower end of the dilated tube, in which the sound caught.

The treatment is directed chiefly to the satisfactory nourishment of the patient; for the œsophageal trouble itself is dangerous only as its prevents the taking of food and leads to starvation. We do not speak of the initial lesion, if there be one, which causes the dilatation. If nourishment can be given through a stomach-tube, the patient almost invariably shows a rapid improvement, which lasts as long as the artificial feeding can be kept up. If, however, any cause prevents the introduction of the tube, we must resort either to nutrient enemata (*vide infra*)—and these will not support the system indefinitely—or we must make a gastric fistula. In the latter case the prognosis depends on the success of the operation and the nature of the original lesion.

2. Diverticula.

Ætiology and Pathology.—Circumscribed pouches in the wall of the œsophagus are termed diverticula. They are divided into two essentially distinct varieties, according to their mode of origin. Zenker has given them the names of pressure and traction diverticula.

The diverticulum due to pressure is extremely rare. It is caused by pressure upon the mucous membrane from within, by which some abnormally weak spot is forced outward. All cases that have been carefully examined thus far have shown that, histologically, the wall of the diverticulum is not the distended but otherwise unchanged wall of the œsophagus, but is composed exclusively of the mucous membrane and the thickened submucous coat. We are therefore obliged to suppose that the mucous membrane is pushed out like a hernia through some gap in the muscular coat. It is only about the neck of the diverticulum that any muscular fibers are found.

The original factor, therefore, in the occurrence of a pressure diverticulum is apparently to be sought in some circumscribed lesion of the muscular coat. As a result of several observations, it is established that a foreign body, sticking in the throat, may separate some of the muscular fibers and push the mucous membrane through the gap thus formed. Or a severe injury leads to a trifling rupture of the muscular coat, and then the food, as it is being swallowed, presses out the mucous membrane at this weakened spot. There are still many other cases where the true origin of the diverticulum remains obscure.

As soon, however, as the formation of the pouch has once begun, there are many influences to make it grow larger. Each successive bit of food, as it glides by, presses upon this yielding and inelastic spot. Gradually a little sac is formed, in which bits of food lodge. These exercise a constant pressure upon the walls of the pouch, and by their weight drag it bodily downward. The larger the pouch, the more it holds, and consequently the more it grows. Thus a pressure diverticulum of the smallest size originally may gradually attain to a diameter of four inches or more. The general shape of the diverticulum may approach the hemispherical, or it may be more cylindrical or pear-shaped.

It is remarkable that, with very rare exceptions, these pressure diverticula are always situated at the beginning of the œsophagus, or rather between it and the pharynx, and almost invariably affect the posterior wall. The pouch hangs, therefore, in front of the spinal column. It pushes out through the lowest fibers of the inferior constrictor of the pharynx; and the feebleness of this muscle is apparently a potent factor in determining the precise point of origin.

The cases thus far seen have been almost all in men, and at a rather advanced age. A few cases have occurred in children.

Traction diverticula are much more common, but in most instances have little interest except for the pathologist. They are not infrequently found unexpectedly at the autopsy. Rokitansky, and later Zenker, have given explanations of their occurrence: some tissue, which has formed adhesions to the œsophagus, contracts and gradually pulls out the œsophageal wall in the shape of a funnel. Bronchial glands are apt to be the seat of the contractile change. These glands are situated near the bifurcation of the trachea, and accordingly the traction diverticula occur oftenest at this level. There may be two or three in one subject. They are rarely over a third of an inch in depth. From within, the mucous membrane, much wrinkled transversely, is seen to be drawn toward the apex of the diverticulum. The wall of the latter consists either of the mucous membrane alone, bulging out like a hernia, or of the mucous membrane covered by the muscular layer. Inasmuch as children quite often suffer from suppuration and caseation of bronchial glands, with subsequent shrinkage, we see why traction diverticula are frequent in children.

Clinical History.—The large pressure diverticula always cause grave symptoms, for they obstruct more and more each day the passage of food. At first there is scarcely any disturbance. Gradually, however, deglutition is impeded. A portion of the food lodges in the pouch, and is either wholly or in part regurgitated, though perhaps not immediately. Decomposition is apt to take place in the contents of the diverticulum, giving rise to foulness of the breath and to nausea. The danger reaches its climax when the distended sac presses sidewise upon the œsophagus and closes its lumen, so that no food reaches the stomach. After protracted strangling and vomiting, the material may be in part ejected, and the patient enabled once more to swallow.

Of course the symptoms in individual cases depend upon the mechanical conditions present, and may vary greatly. Patients contrive all sorts of manipulations, by which they manage to get at least some portion of their food down. Such individuals may maintain a tolerable degree of nutrition for years, although they scarcely ever are in a normal condition. But at last some cause or other renders the amount of food ingested inadequate; whereupon a rapidly progressive marasmus sets in, and the patient will inevitably starve to death unless some relief is afforded.

The most valuable objective evidence in these cases is gained by the use of the œsophageal sound. If the sound enters the sac, its passage is impeded. If it happens to slip by the mouth of the diverticulum, it glides readily into the stomach. This varying result may sometimes be obtained at one sitting by repeated trials, and is of the greatest importance in making the diagnosis.

In some instances where the sac was large, a tumor in the neck has been observed at one side of the trachea, appearing after eating and disappearing when the sac emptied itself. Symptoms due to compression of the recurrent and phrenic nerves and of the blood-vessels have been noticed in some cases.

Auscultation of the œsophagus during the act of swallowing has been practiced, and, of late, attempts have been made to examine it with a speculum. Whether these methods of investigation will prove valuable for diagnosticating diverticula, experience must determine.

The traction diverticula are usually of no clinical importance. They do not affect deglutition at all, and their size is too limited to permit any great accumulation of food in them. There is but one way in which they are dangerous: the apex of the funnel may undergo ulceration and perforation. A foreign body, like some bit of food, produces necrosis of the wall, by what is probably at first a purely mechanical irritation. The tissue ulcerates; and then the inflammation may gradually progress till it causes a severe and usually fatal illness. The most frequent event is perforation into a bronchus, followed by the aspiration of food and pulmonary gangrene. Or the perforation takes place into the pleural cavity, exciting an ichorous empyema. In other cases the pericardium or a large vein has been perforated. Many a case of apparently spontaneous pulmonary gangrene or purulent inflammation of the anterior mediastinum or empyema has been found at the autopsy to have been brought about in the way above indicated. These occurrences are fortunately, however, exceptional.

Treatment.—The only possible way of treating the large pressure diverticula successfully would be by operation. Perhaps surgery will some day win victories in this domain. In the mean while our efforts are confined to sustaining the patient. If he can not swallow, we must try to feed him through a tube. As long as this is possible, starvation is averted. The best way is to have the patient pass the tube himself. He will find out how best to avoid the sac and reach the stomach. If food can no longer be given in this way, there remain two alternatives: rectal feeding (*vide infra*), or making a gastric fistula. As to the latter, there has been thus far very little practical experience, because cases are so rare.

The traction diverticula admit of no special treatment. If the events above mentioned occur, we must endeavor to meet the indications of the individual case.

CHAPTER III.

STENOSIS OF THE ŒSOPHAGUS.

Ætiology and Pathology.—Contractions of the œsophagus occur with such relatively great frequency that they are the most important of all its disorders. They originate in various ways. By far the commonest cause is ring-shaped carcinoma of the tube. The new growth in the mucous membrane encroaches more and more upon the lumen of the œsophagus, until finally it fills it. Carcinoma will be discussed at length in the next chapter. We shall here confine our attention to its purely mechanical action in causing stenosis.

Œsophageal tumors other than cancer are very rare. Fibrous pedunculated polypi have been observed a few times. They usually originate in the lowest portion of the anterior wall of the pharynx, hanging down into the œsophagus, which they may thus obstruct.

A second cause of stenosis is the contraction of cicatrices of the œsophageal wall. The most frequent occasion for this is the extensive ulceration caused by caustic poisons, such as concentrated acids or alkalies. If the victim escapes a speedy death, he is almost certain to have extensive scars formed in the wall of the œsophagus. These scars radiate irregularly in all directions, and, contracting, may almost completely close the tube.

Ulcers from other causes, resulting in stenosis due to the scars they leave, are among the greatest rarities. Syphilis has been the well-established cause in some instances, and Quincke has described a few cases where there were ulcers at the lower end of the œsophagus analogous to the round ulcer of the stomach, or "ulcer due to digestion" (*vide infra*). These ulcers also may eventually produce cicatricial stenosis.

A third and rare cause of stenosis of the œsophagus is compression from tumors external to it. Such swellings may originate in the thyroid gland, or in the lymph-glands of the neck or the anterior mediastinum; or the swelling may be due to a vertebral abscess or an aortic aneurism. This form of stenosis is seldom extreme, for the portion of the tube pressed upon is usually limited.

Next on the list after stenosis due to compression is usually placed what is called intermittent dysphagia (*dysphagia lusoria*). This term is applied to the difficulty in swallowing which is said to be caused by an anomaly in the course of the right subclavian artery. The artery is given off as the last branch from the arch of the aorta, and runs toward the right side just behind or just in front of the œsophagus. It seems, however, *a priori* improbable that the feeble pressure of this vessel as it pulsates should impede deglutition; nor has it yet been proved to do so. It would be more natural to believe, what was indeed the original explanation of the phenomenon, that a large morsel of food passing down the œsophagus compresses the vessel and thus excites uneasiness and palpitation.

Stenosis due to foreign bodies belongs to surgery. It need not be said that the clinical symptoms differ greatly in different cases. Not only the obstruction, but also a possible laceration and consequent inflammation are to be considered. Occasionally thrush has been abundant enough to cause pronounced symptoms of stenosis.

Above the point of stenosis, no matter how the condition arose, if only it is well developed and has lasted a certain length of time, the circular fibers of the muscular coat are more or less hypertrophied. This hypertrophy is due to the increased force required to propel the ingesta downward. In many cases the tube is also diffusely dilated above the stenosis.

Symptoms.—The effect of every œsophageal stenosis is to render deglutition difficult. If the case is a mild one, the patient experiences nothing more than a moderate pressure in the œsophagus upon swallowing. He feels that the morsel is longer than usual in reaching the stomach. Very soon he notices that solid food and large morsels can be swallowed only with difficulty. Accordingly, he is gradually led to confine himself to a liquid diet, takes only small mouthfuls, and always washing down any solid food with a swallow or two of liquid. The narrower the stenosis, the more he is troubled. Finally, even liquids can be taken only slowly and in sips.

It must not be thought that the dysphagia just described is due exclusively to the mechanical obstruction of the lumen. Sometimes a patient is almost entirely unable to take nourishment, and yet at the autopsy no adequate mechanical obstruction is found. The dysphagia must therefore be due to some lesion of the muscular coat of the œsophagus. The impaired contractility of the muscular coat at the affected spot is always a potent factor in impeding deglutition.

As soon as the dysphagia has become considerable there is usually regurgitation of food. At first only a portion of the food comes up, but at last all of it. If the tube has become dilated above the stenosis, food may collect for some hours, and then be regurgitated, mixed with an abundance of very tenacious mucus. We saw a case of this kind where the patient could fill the sac above the stricture with quite a large amount of fluid without a drop reaching the stomach. If he bent his head sharply forward, the collected fluid would run out again through his mouth. It was not until the pouch was completely filled that a small amount of liquid would trickle through the stenosis into the stomach.

Although the dysphagic symptoms above described generally imply œsophageal stenosis, the diagnosis can not be really established without using a sound. Upon introducing this, it is usually easy to detect the obstacle, which may either allow the instrument to pass, with a noticeable jerk, or else prevent its further progress. By measuring the length of the portion introduced before the stenosis is reached we can learn its position. On the average, the entire distance from the teeth to the cardiac sphincter is in adults sixteen inches (40 cm.); from the teeth to the beginning of the œsophagus, six inches (15 cm.); and consequently the length of the latter is about ten inches (25 cm.). If we succeed in passing a smaller sound through the stricture, the feeling as we move it back and forth will give us some idea of the length of the stenosis, or will detect the existence of several lying one below the other, etc. If the end of the sound can be moved about very freely above the stenosis, we may conclude that the tube is dilated there.

Hamburger has employed auscultation of the œsophagus for diagnostic purposes. If we listen behind, to the left of the upper dorsal vertebræ during deglutition, we hear a gurgling sound, due to the act of swallowing, extending down the tube to the stenosis, but no farther. Then come all sorts of sounds, some of them caused by the fluid trickling slowly through the narrow part, and some caused by regurgitation. In general, the results obtained by auscultation are rather variable and uncertain.

Having established the fact of the existence of a stenosis, we have next to determine its nature, which is our chief guide to prognosis and treatment. In certain instances the history of the case gives us the needed information. The diagnosis of cicatricial stricture can hardly be made unless the patient himself tells us of being burned or injured by caustic poisons. The previous history is likewise of great importance if the stenosis be due to foreign bodies or to syphilis. If no decisive ætiological factor can be elicited, we must carefully examine the neck and thorax, with regard to the possible existence of a swelling compressing the œsophagus. In cases where an aortic aneurism has acted in this way, a rhythmical movement has sometimes been communicated to the free end of a sound introduced as far as the stenosis. If the physical examination does not reveal a compressing tumor, and particularly if the stenosis has developed gradually and in an elderly person, we are almost compelled to assume that there is cancer of the œsophagus. This is, after all, by far the most frequent cause of œsophageal stricture. If the new growth has ulcerated, a little portion of it may adhere to the end of the probe, and, on microscopic examination, render our diagnosis of carcinoma certain.

The prevailing characteristic in stenosis of the œsophagus is inanition, increasing as the dysphagia increases. The patient gets to be very much emaciated, and so feeble that he can not leave his bed. The temperature is subnormal; for weeks it keeps at 95° to 97° (35°–36° C.). The pulse grows very small and slow, being 40 to 60 per minute. The heart-sounds are soft. Respiration is superficial and slow; and toward the close of life short pauses occur after expiration, before inspiration begins. The stomach and intestines are so empty that the abdomen is very concave, while the abdominal walls usually feel tense and resistant. In all cases where the nature of the stenosis precludes the possibility of cure or improvement, death results from increasing exhaustion, the lamp of life gradually flickering out.

Prognosis and Treatment.—In prognosis the main factor is of course the nature of the stenosis. If it is due to foreign bodies or to cicatrices, it may be completely cured. In stenosis from other causes it is often possible to produce considerable improvement, at least temporarily. The final result must be confessed to be usually unfavorable, as we should expect from the nature of the original trouble.

The treatment is chiefly mechanical. We shall not speak of operations for the removal of new growths, etc. What we do refer to is a methodical and gradual dilatation of the stricture. Its results are sometimes brilliant, particularly in cicatricial stenosis. Other varieties, like the stenosis from cancer, may sometimes undergo considerable though but temporary improvement with this treatment.

The best instrument to employ is the flexible, so-called English, œsophageal bougie. It is made in all sizes. If the stenosis is very narrow indeed, we may have to resort to catgut at first. Whalebone bougies, with olive-shaped ivory tips of various sizes to screw on the end, are also good, except that, being stiffer, there is more danger in using them. For introduction of the bougie, the patient should be seated, with the head slightly extended backward. The first two fingers of the left hand are introduced into the throat and guide the instrument, previously well oiled, over the back of the tongue and the epiglottis into the œsophagus. Of course, no violence must be used. Otherwise a perforation might occur if there were a soft, broken-down cancer, or an aortic aneurism. However, such a misfortune is very exceptional.

The use of the bougie is almost invariably beneficial if the stricture can be passed. The patient generally finds that he can swallow easier than before, and will himself request a repetition of the performance. If the patient is an intelligent person, it is advisable to have him introduce the bougie himself. Patients often acquire even greater skill with it than the physician has. The bougie should be passed regularly once a day, or, at most, twice daily; and in favorable cases we shall be able gradually to increase the size. If so, the symptoms speedily abate, and, with the increased ingestion of food, the patient gains flesh very fast.

If the stenosis is extreme, and, although it admits the bougie, does not allow of sufficient nourishment, we must pass a tube into the stomach through which to introduce liquid food. Milk is the best food to choose. Raw eggs, sugar, wine, etc., may be mixed with it. The various infant's foods and Hartenstein's "leguminose" are also excellent. Their consistence is favorable for the purpose, and they supply a considerable amount of nourishment in a small bulk.

If this means also fails us, two alternatives are left, unless we are to resign our patient to death by starvation: (1) operative interference, œsophagotomy if the stenosis is high up, otherwise gastrostomy; (2) rectal feeding.

For information about the operations we must refer to surgical literature. As to feeding *per rectum*, its results are never brilliant. It is indeed probable that life may be by this means somewhat prolonged, but not indefinitely. But the moral effect is very valuable, when the patient could otherwise receive no nourishment whatever. The sufferer feels that something is being done to avert absolute starvation.

The simplest materials for the nutrient enemata are milk, eggs, and wine; to which we may add pepsine and pancreatine in the hope of promoting absorption. Leube's pancreatic meat emulsion is still better, although more troublesome in its preparation. Leube's directions are as follows: About five ounces (150 grm.) of meat, cut very thin and then minced finely, and about two ounces (50 grm.) of minced pancreas (from the calf) free from fat, are to be stirred with about three ounces (100 grm.) of lukewarm water until the mixture has the consistence of gruel. Before it is injected, the rectum should be cleansed by an enema of plain water. One such enema is to be given daily.

CHAPTER IV.

CANCER OF THE ŒSOPHAGUS.

Ætiology and Pathology.—Cancer is the most important and most frequent affection of the œsophagus. We have already mentioned in the preceding chapter that often stenosis is the result of carcinoma in the œsophageal walls.

Little is known about the ætiology. It has been often maintained that mechanical, chemical, or thermic irritation of the mucous membrane may result in the development of cancer; but this is not certain. It receives some support from the remarkably frequent occurrence of œsophageal cancer in hard drinkers. Now and then the patient himself will allege a perfectly definite cause for his disease, such as the lodging of a foreign body, or the swallowing of a very large or very hot morsel. Still it is hardly possible in any particular case to decide how much value such statements have. It has been maintained that the carcinoma sometimes develops in the scars of old ulcers. This is of interest when we recall the similar fact in regard to gastric carcinoma (*vide infra*).

Œsophageal cancer follows the general rule in being most frequent in elderly people—somewhere between forty and sixty years of age. The male sex is decidedly more often attacked than the female.

As we might expect from the histological character of the epithelium lining the œsophagus, primary cancer here is invariably composed of pavement cells. The new growth may be either hard, firm, and fibrous, or it may be soft, succulent, and but scantily supplied with connective tissue. The first variety corresponds to the "scirrhus" of older writers, and the second to "medullary" cancer. Usually the new formation encircles the entire tube like a ring, extending three to ten centimetres longitudinally. Exceptionally a still larger portion of the œsophagus is involved, sometimes almost all the mucous membrane. The tumor is usually seated in the lower and middle thirds of the œsophagus, being much rarer above.

Symptoms and Complications.—In the great majority of cases the symptoms are those of a gradually increasing stenosis, with its results. We may therefore refer to the preceding chapter for most of the particulars. There are, however, exceptional cases where the carcinoma is flat and entails no dysphagia, or so little that œsophageal trouble may not be suspected. We have repeatedly seen cases of extensive secondary hepatic cancer, or of pulmonary gangrene (*vide infra*), where the real primary disease was a flat cancer of the œsophagus which gave no clinical signs of its existence, and was therefore not diagnosticated.

It is characteristic of the stenotic symptoms produced by œsophageal cancer that sometimes a considerable and apparently spontaneous amelioration occurs. This is the result of an ulceration of the new growth. It crumbles away, as the result of superficial disintegration. The tumor is transformed into an ulcer, and one can easily understand how this may result in a temporary improvement in deglutition.

Important clinical symptoms may result from conditions secondary to the new growth. The cancer may extend to neighboring organs. Not infrequently the cardiac extremity of the stomach is thus involved. Sometimes such a tumor may be felt in the epigastrium; but in most cases there is nothing to indicate that the stomach is attacked.

The neighboring parts of the trachea or bronchi are sometimes affected, and important symptoms result from such a complication. If perforation occurs, an almost certain result is the inhalation of food or of decaying bits of the tumor, with consequent pulmonary gangrene, and, as a rule, speedy death. The disease has also been observed to attack the pleura, and end in perforation. The same is true of the pericardium and the aorta. A few instances are known where the vertebræ have been involved, the spinal cord compressed, and paraplegia thus induced. We have ourselves seen one such case.

Quite frequently the recurrent nerve is affected, and a paralysis of the vocal cords is produced, which can be detected by the laryngoscope. This nerve lies so close to the œsophagus that it is peculiarly exposed to injury from the new growth itself, or from any inflammatory process which may be set up around it.

Metastatic cancer in distant organs is not infrequent, and may give rise to important symptoms. It attacks most frequently the liver and the lungs. The kidneys, pancreas, bones, and brain are also liable to it.

Pulmonary gangrene must be mentioned as a relatively frequent complication, and one which has serious consequences. We have already stated that it may result from perforation. A still more frequent cause is the inspiration of decaying masses vomited or regurgitated by the patient.

Clinical History, Termination, Prognosis, and Treatment.—The disease is incurable. Operative removal has never been successful. The entire duration of the disease seldom exceeds a year, or a year and a half. At the end of this period the patient dies either from lack of nourishment or as a result of some one of the complications above enumerated. Treatment is purely symptomatic. Temporary improvement may be obtained by mechanical treatment of the stenosis. The particulars about this may be found in the preceding chapter.

CHAPTER V.

RUPTURE OF THE ŒSOPHAGUS.

MEDICAL literature records a small number of cases which prove that the sudden rupture of the œsophagus in persons previously perfectly well is possible, although of course very rare. The first and most famous instance was described by Boerhaave in 1724.

The symptoms, according to the observations thus far reported, usually commence with sudden nausea and vomiting, during or shortly after a hearty meal. There is simultaneously an extreme, general collapse. There is pallor of the face and extremities, cold perspiration, and an extremely feeble pulse. Sometimes the patient feels a sudden darting pain in the chest. Almost invariably an extensive emphysema overspreads the neck and thorax. Death results in a few hours, or at latest in a few days.

The autopsy reveals a tear in the œsophagus, invariably situated in its lower half. It may be five centimetres long, and is almost always longitudinal. Food has usually escaped into the surrounding tissues, in which case a secondary purulent inflammation exists, if death was not immediate.

Zenker has attempted to explain this remarkable phenomenon by a supposition which is really very plausible, namely, that œsophagomalacia always precedes these so-called spontaneous ruptures. The cause of this softening of the œsophageal walls is probably the action of gastric juice escaping into the tube and attacking a surface which, through some temporary disturbance in the circulation, has lost its normal powers of resistance.

CHAPTER VI.

NEUROSES OF THE ŒSOPHAGUS.

1. **Spasm of the Œsophagus.**—In rare instances œsophageal disturbances are observed, which appear to result from spasmodic contraction of its muscular coat. Nervous and hysterical subjects are particularly apt to present temporarily the symptoms of extreme stenosis, for which there can be no anatomical basis. Such cases are termed "spastic stenosis" of the œsophagus, or "œsophagismus." It is, of course, possible that there may exceptionally be some real lesion at the foundation of the trouble, and that the spasm is the reflex result of an ulcer or inflammation affecting the œsophagus. It is even affirmed that the reflex influence may sometimes originate in distant organs, such as the uterus. The dysphagia is usually attended by a painful sense of constriction in the throat and chest. The bougie comes upon an obstruction, which usually soon yields. The diagnosis is confirmed by the easy passage of the bougie after the spasm is over. Other important factors are, the character of the symptoms as a whole and the other attendant nervous and hysterical disturbances. Some authors also explain the "*globus hystericus*"—that feeling as if a lump were passing up or down in the throat and chest—as a spasm of the œsophagus.

2. **Paralysis of the Œsophagus.**—Of this subject we have little accurate knowledge. It is not improbable that an extensive bulbar paralysis, affecting the muscles of the pharynx and larynx, may sometimes involve the œsophagus; although such a disturbance hardly ever gives rise to prominent symptoms in this disease.

Ziemssen asserts that sometimes the œsophagus seems to participate in post-diphtheritic paralysis, when extensive.

SECTION IV.

Diseases of the Stomach.

CHAPTER I.

ACUTE GASTRIC CATARRH.

(*Acute Gastritis.*)

Ætiology.—As the gastric mucous membrane is not open to direct inspection, we usually have to base our conclusion that an acute gastric catarrh exists, upon the analogy of the symptoms with those which we have seen in other mucous membranes. The pathology of gastric catarrh has thus far been very little studied, for the disease almost always ends in recovery; and in those cases which, on account of some other disease, do terminate fatally, the signs of catarrh become very indistinct. Nevertheless, we are fully justified in assuming that a catarrh of the gastric mucous membrane is the cause of most of the brief gastric disturbances which occur. That there may be other acute disorders of the stomach, in which there is no anatomical change whatever, but merely some functional anomaly ("dyspepsia," in the narrower meaning of the word), is possible, but has not yet been proved.

The most frequent cause of acute gastric catarrh is some direct injury of the mucous membrane; the injury may be thermic, as by taking food too hot or too cold, or mechanical, or chemical. The last is the most important. It is irritation that produces the frequent cases of catarrh which follow over-eating, or the ingestion of improper, indigestible, highly spiced, or very sour articles. In the same way arise the disorders caused by excess in alcohol, or by taking certain medicines, as well as a large portion of the milder cases of poisoning from all sorts of injurious substances.

A special importance attaches to the ingestion of decaying substances. The incautious use of tainted meat or fish may be followed by relatively severe forms of acute gastric catarrh. The products of decomposition act as chemical irritants upon the mucous membrane; and the ferments and putrefactive agents likewise continue in activity after reaching the stomach, and thus contribute to produce the inflammation.

It is universally assumed that catching cold may excite gastric catarrh; but the importance of this factor can seldom be demonstrated.

Individual predisposition to the disease varies greatly. Weakly children are peculiarly liable to it; also anæmic persons, fever patients, convalescents from severe diseases, and chronic invalids whose general nutrition and vigor are impaired. Such persons may be affected by things which the strong and healthy would not feel at all. The probable explanation of this especial liability to the disease is that, under the circumstances enumerated, the physiological functions of the stomach are not a little restricted. It has been experimentally proved that in fever, and in most anæmic or weakly persons, the secretion of the gastric acids is subnormal in amount. Digestion is thus considerably protracted. The contractions of the muscular coat of the stomach, which are set up by the normal gastric secretions, are also rendered less vigorous. And, further, it is probable that the muscular fibers themselves may participate in the general debility. Thus it happens that the food is not carried on into the duodenum, but remains undigested in the stomach. It undergoes abnormal decomposition to a certain extent, and acts both as a mechanical and as a chemical irritant upon the gastric mucous membrane.

Symptoms.—The most constant subjective symptom is anorexia. In many cases the very thought of food excites disgust. What the patient does eat tastes flat, and he is therefore very eager for piquant dishes, highly spiced or very sour. Thirst is often present, and a feeling of dryness in the mouth.

The subjective gastric sensations are seldom those of marked pain. The usual complaint is of constant pressure and fullness. Sometimes the patient is conscious of the peristaltic movements of the stomach. He has "rumbling" of the bowels.

There is nausea, and often even vomiting. The vomitus consists for the most part of undigested food, with which mucus, and sometimes bile, is mingled. Eructations of gas or liquid are frequent.

Physical examination reveals little. The epigastrium may be somewhat prominent as a whole, and may be tender on pressure. The tongue is almost always thickly coated and dry. The breath is usually disagreeable, and there is a persistent flat or bitter taste in the mouth.

In all well-marked cases of acute gastric catarrh the general health is considerably impaired. The patient is languid, and disinclined to exertion. Moderate elevations of temperature, with chilliness and feverishness, are not infrequent. Now and then there is a decidedly typhoidal condition, with marked nervous symptoms, such as intense headache, vertigo, and dullness. The term "gastric fever" is applied to such cases, and it is possible that they may sometimes be the result of a constitutional infection. It is also probable that toxic influences are exerted by the abnormal products of the fermentation which takes place in the stomach. For example, Senator mentions sulphuretted hydrogen as thus generated. A short time ago Litten described a few cases in which at first there were such dyspeptic symptoms as nausea, vomiting, flatulence, and a coated tongue, but which soon gave evidence, by restlessness, headache, great muscular weakness, and somnolence, of rather severe nervous disturbance in addition. The breath had a marked "fruity" odor; and, on adding chloride of iron to the urine, a strong reddish color was developed, as in the so-called acetone-reaction. So that it seems probable that an auto-intoxication had occurred, somewhat resembling diabetic coma.

Chief among complications are the intestinal symptoms, which are frequently coincident with the gastric disorder. Constipation is the rule. There may be diarrhœa. The gastric catarrh may by extension involve the duodenum, and give rise to jaundice. Sometimes herpes appears upon the skin. This fact argues for the infectious nature of many cases of gastric catarrh.

The course of acute gastric catarrh is invariably brief. Usually after a few days there is complete recovery.

Treatment.—If, at the beginning of the disease, there is reason to suppose that the stomach is loaded with undigested food, an emetic is indicated. If it is desired to avoid the irritative action of an emetic upon the gastric mucous membrane, a subcutaneous injection of one sixth grain (0·01) apomorphine may be given.

In most cases, however, emetics may be dispensed with. The treatment in such cases consists mainly in a strict regulation of the diet, allowing only such things as milk porridge or iced milk. Of internal remedies, ten to fifteen drops of dilute hydrochloric acid in half a wine-glass of water sometimes seems beneficial; while in other cases, where there are sour eructations and vomiting, alkalies are to be given. We may order as much bicarbonate of soda or as much magnesia as will rest on the point of a pen-knife. The so-called stomachics and bitters are also often prescribed, such as compound tincture of gentian or tincture of rhubarb. If the vomiting is obstinate, the best remedies are bits of ice, sips of cold seltzer water, or small doses of opium. If the bowels are constipated, Carlsbad salts, Hunyadi water, or rhubarb may be given.

CHAPTER II.

CHRONIC GASTRIC CATARRH.

(*Chronic Gastritis. Chronic Dyspepsia.*)

Ætiology.—The same injurious influences which produce acute gastric catarrh if frequently recurring, excite the chronic form of the disease. The most potent causes are improper diet and the improper use of alcohol. Individual predisposition is important in this as well as in the acute disease. Sometimes it even seems to be a matter of inheritance. It is not a rare thing to find that a large number of the members of a family have a "weak stomach."

Chronic gastric catarrh is not always a primary affection. It may be secondary to some other disease. All diseases which result in passive hyperæmia of the portal system, such as cirrhosis of the liver or thrombosis of the portal vein, are not infrequently followed by gastric catarrh; and when the disease occurs in persons afflicted with chronic cardiac or pulmonary disease, it is to be regarded as in part due to stasis.

Pathology.—The macroscopic changes in the gastric mucous membrane are in most cases very slight. Usually it is coated with a layer of tough, grayish-white mucus, in which is suspended a greater or less amount of detached epithelium. The membrane is red, unless rendered gray by excessive pigmentation. The changes are almost always most extensive in the pyloric region.

If the catarrh is of long duration, there may be still further changes. The mucous membrane often seems smooth and atrophied. The glands are narrower and shorter than normal, and the connective tissue between them is increased in amount. Other cases present hyperplasia of the mucous membrane. The internal surface of the stomach is thickened and mammillated. The hyperplasia in these cases affects chiefly the tubes of the glands in the mucous membrane, although the submucous layer may also be considerably thickened.

Symptoms.—The symptoms of disturbed gastric digestion, or dyspepsia, to be observed in all sufferers from chronic gastric catarrh, are referable to the following functional anomalies:

The secretion of the gastric juice depends upon the maintenance of the circulation of the blood in the gastric mucous membrane. This is merely in accordance with a general rule. It is easy, therefore, to comprehend that any inflammation, disturbing as it does the circulation of the blood, must modify the gastric secretions. We may also assume that a deficiency in the secretion of the gastric juice is the chief factor in producing the dyspepsia which accompanies both the primary and inflammatory catarrh and that resulting from venous stasis. The possible diminution in the production of pepsine is probably not of great importance in this connection; for, like any other ferment, a very small amount of it would prove efficient under favorable circumstances. What seems of more importance is the diminished proportion of hydrochloric acid in the gastric juice of patients with chronic gastric catarrh—a condition which has been repeatedly proved to exist. The processes of digestion are thereby not a little lowered and impeded. It is self-evident that in cases where there is actual atrophy of the mucous membrane and of the glands in particular, the normal processes of gastric secretion may be still more seriously impaired.

A further unfavorable result of the imperfect digestion dependent on the lack of hydrochloric acid is that the undigested food is very apt to undergo abnormal fermentation and decomposition. These fermentative processes give rise to lactic, butyric, and acetic acids, and to alcohol. The direct cause of their production is that the ferment-producing spores are not destroyed by the gastric juice, as they would be under normal conditions. On the contrary, they find in the food stagnating within the stomach the most favorable conditions for the development of their activity. These products of abnormal fermentation in their turn irritate the gastric mucous membrane and prolong the catarrh.

Another factor contributes to the disturbance of the normal digestive processes, namely, the excessive secretion of mucus. As mucus has an alkaline reaction, its presence neutralizes a portion of the acid in the gastric juice and lessens the potency of the latter. The mucus also does harm in a purely mechanical way. It envelops all the ingesta, and thus to no small degree prevents the gastric juice from reaching them. Morsels completely covered with mucus can remain for quite a long time in the stomach without being digested.

A very great importance attaches to the motor disturbances which the stomach suffers in chronic catarrh. The normal peristalsis is of course an essential factor in the process of digestion. It is its function to bring all portions of the contents of the stomach successively into adequate contact with the mucous membrane. Thus alone is the uniform digestion of the entire mass rendered possible; and, as physiology has taught us, the gastric secretions are continually being stimulated by the contact of the still undigested food with the lining membrane. Lastly, it is the peristalsis again which pushes on the already digested food into the duodenum, and thereby prevents the useless accumulation of matter within the stomach.

Now, gastric peristalsis is undoubtedly much impaired in catarrh. One reason for this is the actual harm done the muscular coat. Every decided inflammation renders the muscular layer œdematous, and thus impedes its activity. In all cases where food accumulates in undue amount in the stomach, the muscular coat is gradually so stretched as to become still more incapacitated for its proper functions. A factor which is perhaps of still greater importance than these lesions is the diminished vigor of the normal excitants of the peristaltic movements. We learn from physiology that the chief part in arousing peristalsis is played by the normally constituted gastric juice, and more especially by the acid therein contained. It follows that all influences which modify the amount or composition of the gastric juice must have the remote effect of diminishing the gastric peristalsis. The resulting evils can be inferred from what has been already stated. Digestion is impaired, and abnormal decomposition promoted; and each effect becomes in turn a cause of further derangement—a condition of things which we are constantly meeting in the pathology of digestion.

It remains to be stated that absorption is impeded in chronic gastric catarrh. It has been shown in Ludwig's physiological laboratory, in Leipsic, that no inconsiderable portion of the peptones manufactured in the stomach are absorbed by the blood-vessels in its walls. Now, as the circulation is disturbed by the inflammation, we should expect the absorption of peptones to be thereby diminished. The peptones remaining in solution within the stomach and not absorbed have been shown by experiment to disturb and delay the transformation of fresh albuminous substances into peptones. The diminished peristaltic action also tends to prolong the time during which the peptones remain in the stomach; and numerous observations teach that normal peristalsis has a direct influence in promoting absorption, so that, if it be diminished, absorption is checked.

We have therefore a whole series of factors, all of which contribute to disturb the normal processes of digestion. We have discussed them at some length, because they serve to explain not only the digestive symptoms of chronic gastric catarrh, but those of almost all the other gastric diseases.

If we now proceed to the symptoms which lead us to a diagnosis of dyspepsia or chronic gastric catarrh, we find first anorexia. This symptom is common to all gastric disorders. There is sometimes a moderate appetite, but it is soon changed to a feeling of repletion upon the ingestion of even a slight amount of food. In other cases there is actual dislike for any form of nourishment; the patient eats little, and prefers highly spiced, piquant dishes. There is often a persistent bitter, flat, or otherwise abnormal and disagreeable taste in the mouth.

Subjective sensations in the region of the stomach are rarely entirely absent. As a rule, there is a feeling of fullness or pressure and of dull pain. These troubles may either be constant, or occur after meals.

The eructation of gas is a very frequent and annoying symptom. Often a sour liquid comes up with the gas ("sour stomach"). The gas is a mixture of atmospheric air and the abnormal gaseous products of the decomposition which takes place in the stomach. Hydrogen, carbonic dioxide, and occasionally inflammable gases, such as marsh gas, have been detected. The sour masses regurgitated irritate the œsophagus, and give rise to a sharp, burning sensation, known as "heartburn."

In many instances the feeling of nausea is superseded by actual vomiting. This almost always takes place either directly after eating or an hour or two later. The vomitus consists mainly of undigested food, usually mixed with a large amount of mucus. The chemical reaction may be either neutral or strongly acid. If acid, however, it is often not made so by hydrochloric acid, but by the acetic, lactic, or fatty acids generated by the abnormal fermentation going on within the stomach. That the contents of the stomach have an acid reaction is no proof that the gastric juice has peptonizing properties, for none of these other acids have nearly so much efficiency in promoting digestion as has hydrochloric acid. They may even at times check the process. A small amount of blood may be mixed with the matter vomited, but need not excite apprehension. Microscopic examination reveals little that is characteristic. The cells of the yeast fungus are frequently found, and also the *sarcina ventriculi* (see Fig. 34). We may add that this latter has apparently no connection with the abnormal processes of fermentation.

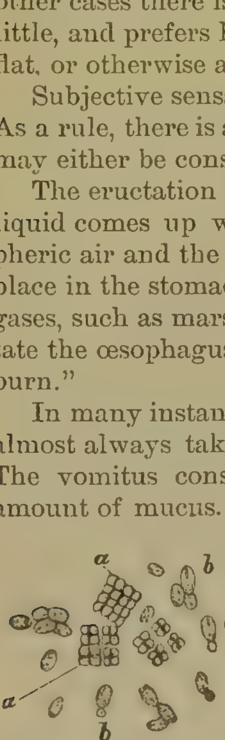

FIG. 34.—*a*, Sarcina ventriculi. *b*, Yeast-cells.

A peculiar kind of vomiting is extremely frequent in the chronic gastric catarrh of drunkards. We refer to the well-known morning vomiting of a watery fluid, which is usually alkaline in reaction, and apparently consists at least in part of saliva that has been swallowed. (*Vide* also page 357.)

The physical examination shows malnutrition (*vide infra*), but not much that is distinctive of the disease. The center of the tongue is often coated, while its point and edges are red. Considerable salivation not infrequently exists.

The epigastrium seems in many cases unchanged. Sometimes the stomach is distended, and somewhat tender on pressure. On palpation, a splashing can sometimes be both heard and felt. Its presence usually indicates dilatation of the stomach (*q. v.*).

EXAMINATION OF THE CONTENTS OF THE STOMACH BY MEANS OF THE STOMACH-TUBE.

The examination of the contents of the stomach with the aid of the stomach-tube is much more important than the mere external examination of the stomach, which usually affords little information. This newer method of research was introduced mainly by Leube, and by its means we are able to learn about many of the processes of digestion, and to obtain a somewhat clearer insight into the especial forms of digestive disturbance which may be present. The use of the stomach-tube for this purpose has indeed certain inconveniences for the patient, and therefore it will not, of course, be used immediately in every case. If, however, we have to deal with any obstinate disorder of the stomach, investigation by means of the tube is the more desirable because we may thus obtain data valuable not only from a diagnostic but also from a therapeutic point of view.

The first question which can be determined with the aid of the stomach-tube relates to the time occupied in digestion, or, in other words, to the motor power of the stomach. The best tube to use is the soft catheter of Nélaton, supplied at its lower end with sufficiently wide openings. Numerous experiments on healthy human beings have shown that under ordinary circumstances gastric digestion is ended in six or seven hours at the latest, so that we should find the healthy stomach completely empty six or seven hours after the last meal. Indeed, frequently this is the case much earlier, say three or four hours after a meal. The investigation of this point in disease of the stomach is usually carried out in the following way: The patient is given, either in the morning fasting or at noon, a so-called test-meal; this consists either of flesh, such as minced beef-steak, with a roll, or merely of a roll with some tea. Six or seven hours after this meal the stomach is rinsed out with lukewarm water. In normal digestion the rinse-water is almost perfectly clear, and contains at most a few small bits of mucus and the like, while in dyspepsia there are still present in the stomach numerous remnants of the ingesta. This latter condition demonstrates with certainty the presence of disorders of digestion; while the sufficiently rapid emptying of the stomach is in most cases a sign of normal digestion, although it may sometimes exist despite some few deviations from perfect health.

We must therefore endeavor to make further investigation as to the quality of the gastric juice and the digestive power of the stomach, and for this purpose also the stomach-tube is indispensable. The result of attempts to obtain a perfectly pure and undiluted gastric juice has, to be sure, thus far been unsatisfactory; still, we are able to get most of the reactions in a satisfactory way from the total gastric contents—that is, from the gastric juice mingled with portions of food. For this purpose we must, of course, draw out the gastric contents at a time when the secretion of the gastric juice has reached its highest point, and, accordingly, the tube is introduced about an hour or an hour and a half after the test-meal. By coughing and pressing the patient is usually able to squeeze out a sufficient amount of the gastric contents through the tube, and this by filtration is separated from the remnants of the food. The filtrate thus obtained may now be tested in various ways. If, however, we can obtain none of the contents of the stomach by mere expression, it is necessary to gain our result by pouring some fluid into the stomach and siphonage. For the investigation of the quality of the gastric juice it is in general preferable to give the so-called test-breakfast, consisting of a roll and tea, because if we give a larger test-meal the secreted hydrochloric acid is at once and completely combined with the abundant nitrogenous substances present. In that case the test for free hydrochloric acid proves negative, even when the stomach has secreted hydrochloric acid in abundance.

The investigation of the gastric juice thus obtained relates, first, to its richness in acids, and in particular free hydrochloric acid, and, secondly, to its digestive power.

The digestive power of the gastric juice is tested in the following way: A thin shaving of coagulated egg albumen is allowed to stand in the gastric juice for sixty to ninety minutes. If the gastric juice possesses normal efficiency, the albumen will be completely dissolved at the expiration of this time. Under pathological conditions the gastric juice requires a much longer time for this, or it may not digest the albumen at all until a few drops of hydrochloric acid are added.

Testing the gastric juice in relation to the acids it contains has become, as the result of numerous investigations, a matter of great importance during the last few years. In the first place, we must determine whether the juice contains any free acid at all or not. This is best done by means of a solution of tropæolin. Even slight amounts of acid change the yellow color of tropæolin to red. Red Congo-paper is also to be recommended for demonstrating free acids. This takes on an evident blue coloration upon the application of a drop of acid gastric contents. Both reactions take place only if free acids are present. They are not produced by acid salts.

If the gastric juice contains free acid, the next question is whether this free acid is hydrochloric or lactic. Hydrochloric acid is certainly the acid which is by far more important and absolutely necessary to digestion, while lactic acid is probably entirely dependent for its creation upon the digestion of the starches in the stomach. In the case of normal digestion lactic acid alone is found during the first half-hour, the amylolytic stage of gastric digestion. Then the hydrochloric acid begins to appear along with the lactic acid. The latter grows less and less abundant, and at the end of an hour there is usually found hydrochloric acid alone without any lactic acid. The most certain and best means for the demonstration of free hydrochloric acid is the phloroglucin-vanillin test of Günzburg. The solution is composed as follows: Phloroglucin 2, vanillin 1, absolute alcohol 30 parts. A few drops of this solution are mixed in a porcelain evaporating dish with a few drops of the gastric juice which is to be examined, and warmed over a flame. If there be free hydrochloric acid present there at once appears at the edge of the heated liquid a beautiful red border. The test is very easy to carry out, and extremely sensitive. The second reaction for free hydrochloric acid is with methyl-violet. Its solution has a violet color, which changes to blue upon the addition of a small amount of hydrochloric acid, while lactic acid does not produce this change in color. For the direct demonstration of lactic acid, on the other hand, we may use Uffelmann's chloride of iron and carbolic test. Upon the addition of a drop of liquor ferri perchloridi to a two-per-cent. solution of carbolic acid, there is at once developed a steel-blue color. Upon adding the gastric juice to this mixture the blue color will at once change to yellow, if lactic acid be present.

According to the later investigations of Cahn and Von Mehring, we must add that the failure of the reactions for hydrochloric acid does not authorize us to assert that there is actually a complete absence of hydrochloric acid in the gastric juice. The authors just named have demonstrated hydrochloric acid where the methyl-violet reaction has failed. Nevertheless, the negative result of the test, which, it seems, must be caused by the mixture of foreign matter with the gastric juice, does possess practical importance, inasmuch as the reaction can always be demonstrated if digestion is normal.

If we now return from this digression to the conditions present in chronic gastric catarrh, we find that in this disease digestion is often decidedly delayed and incomplete. The gastric juice usually contains free hydrochloric acid, but the amount of it is often appreciably diminished. Indeed, there are severe cases of chronic gastric catarrh where the secretion of hydrochloric acid seems to be permanently almost *nil.* This is probably to be explained by the final occurrence of an atrophy of the mucous membrane. The appearance of abnormal organic acids in the gastric contents, such as butyric and acetic acids, points to processes of abnormal fermentation in the stomach.

With regard to those disturbances which are associated with an increased secretion and an abnormally large amount of acid in the gastric juice, see the appendix to this chapter.

Of the other organs, the intestine most frequently suffers in chronic gastric catarrh. The combination of gastric disease and intestinal disease is not infrequent. In almost all cases of chronic gastric catarrh the bowels are irregular. Habitual constipation is the rule, but sometimes there is diarrhœa. If much gas is generated in the stomach, the intestinal canal often becomes implicated, and tympanites and flatulence develop. There may be catarrh of the duodenum leading to jaundice.

The urine is often only feebly acid in reaction, and therefore frequently deposits a large amount of phosphates. The diminished acidity is probably in part a result of the deficient secretion of acid by the stomach, though excessive vomiting (compare the chapter on dilatation) may also have some influence, because of the acid thus lost.

It has been often maintained that eczema and other chronic cutaneous diseases are sometimes referable to gastric catarrh, but the fact has not yet been definitely established.

There is a more evident relation between chronic gastric catarrh and certain nervous derangements. The disease has a notable influence on the spirits. In numerous instances it is accompanied by decided hypochondriasis. There may be other nervous symptoms, such as headache, vertigo, and dullness or listlessness. Vertigo in particular is a familiar concomitant of the disease ("*vertigo a stomacho læso*"). It is possible that in many cases these symptoms are toxic, resulting from the absorption of the abnormal matters generated within the stomach (*vide supra*, page 373). In the majority of instances, however, they are due to the neurasthenic and hypochondriacal condition of the patient (see the chapter on nervous dyspepsia).

Whenever the catarrh is of any duration and severity, the general nutrition is seriously impaired. The diminished appetite and the imperfect digestion and absorption of what is eaten contribute to produce a gradual and considerable loss of weight. The fatty and muscular tissues atrophy. The skin grows dry and harsh, and usually has a dirty-pale color.

Individual cases differ greatly in the combination of symptoms they present and in their course. The anorexia, gastric oppression, eructations, vomiting, and other important disturbances already mentioned, exhibit the greatest diversity in their intensity and their grouping. In the milder cases, loss of appetite and moderate local uneasiness may be the only symptoms. Frequent vomiting is seen only in the severer cases. The disease often lasts for years, especially if the patient neglects himself. In most cases there are frequent remissions and exacerbations, usually dependent upon external causes.

The disease is not intrinsically fatal, but the general debility consequent upon it may indirectly shorten life.

Diagnosis.—The diagnosis is based upon the existence of chronic gastric symptoms unattended by evidence of any more serious lesion, whether of the stomach itself or of some other organ. For example, the possibility of gastric ulcer, cancer, or dilatation is to be considered. This elimination of other diseases is very important. It is not at all exceptional to meet in practice cases that have been carelessly diagnosticated as chronic gastric catarrh where a more thorough examination or the further progress of the case leads to an entirely different conclusion —chronic pulmonary or cardiac disease, chronic nephritis, or one of the other gastric affections above mentioned. It should therefore be our invariable rule to reach the diagnosis by exclusion, after making a careful and complete physical examination, in order to detect any other possible disease to which the gastric symptoms might be referred. The important differential diagnosis between chronic gastric catarrh and so-called nervous dyspepsia will be discussed hereafter.

Treatment.—If the disease seems to be merely symptomatic—the result, for instance, of venous stasis due to chronic cardiac, pulmonary, or hepatic disease—our efforts must, of course, be chiefly directed to the relief of the original trouble.

Treatment of the primary form, on the other hand, must always begin with regulation of the diet. Such vague injunctions as "to be cautious" or "to avoid indigestible articles of food" are useless. The patient must have a perfectly definite bill of fare prescribed for him; nor can any universal one, suitable for all cases, be drawn up. In each individual instance the individual circumstances must be considered. The personal experiences of the patient himself are by no means to be disregarded. One man may be quite unable to digest what is well borne by others, and *vice versa*.

In the first place, certain foods must be utterly forbidden to such patients as do not themselves avoid whatever disagrees with them. All articles must be prohibited which may irritate the mucous membrane, either mechanically or chemically. This includes all the coarser sorts of vegetables or fruits, containing a large proportion of indigestible cellulose; and all dishes that are very sour, strongly salted, or highly spiced. Potatoes, farinaceous food, and all substances mainly composed of hydrocarbons, must also be interdicted; because almost all the abnormal fermentative processes, the evil consequences of which have already been considered, are promoted by the hydrocarbons. Fat is also harmful. It impedes digestion by protecting the contents of the stomach from the action of the gastric juice, in a purely mechanical way; and then, being changed into the fat acids, it causes sour eructations and pyrosis. It is important that all forms of alcohol should be forbidden. Fleischer and others have proved by actual experiment that even small amounts of alcohol prolong and hinder the digestive process. An injunction to abstain totally is usually better obeyed in a severe case than the advice to be very moderate in the use of liquors. The food should neither be very hot nor ice-cold.

In determining what the patient may be allowed to eat, we are to consider, as already mentioned, his own personal experience as well as our more general knowledge. An intelligent patient will often be himself the best judge of what agrees or disagrees with him. The following foods are very easy to digest: Milk, soft-boiled or raw eggs, broths, and certain artificial preparations, chief among which stand the Leube-Rosenthal meat solution and the meat peptones which have been lately put upon the market. The brain and sweetbread of calves are easily digestible; also birds, such as pigeons, chickens, and partridges, and thin shavings of raw meat or raw ham. Gradually we may proceed to somewhat heartier food—veal, game, roast beef, and light farinaceous dishes. The worse the symptoms are in any case, the more strict must we be in regard to diet. For drink, besides water or Seltzer water, very weak tea and non-oleaginous cocoa are good.—Shall we permit coffee? This is often a question of great interest to the patient: it must be answered according to his individual experience. Coarse bread is to be forbidden. Ordinary white bread, toasted, if it seems desirable, may be allowed in moderate amount.

Solid articles of diet must be finely cut up and well chewed before being swallowed. It is sometimes advantageous to take more than three meals a day, each one being proportionally smaller. Other patients relish their food better if the intervals between eating are prolonged.

There are other special indications to be met. As we have already seen, one of the chief factors in maintaining digestive disturbance is the abnormal detention of undigested food in the stomach. If we can only empty the viscus properly, we thereby relieve it of the abnormal processes of decomposition and fermentation, as well as of mucus which may have collected in injurious amount. This indication is best fulfilled by the mechanical treatment of chronic gastric catarrh by means of the stomach-pump. Its results are often brilliant; but rinsing out the stomach is never agreeable to the patient, and the method should not be resorted to except in severe and inveterate cases. The most suitable are those where an examination of the contents of the stomach, made as above described, shows that abnormal fermentative processes are going on, or that the ingesta stagnate in the organ. For further particulars we refer to the chapter on dilatation.

If we now turn to a consideration of the internal remedies used in chronic gastric catarrh, it is possible to decide with regard to their employment in a much more intelligent fashion than heretofore, because we are able in all severer cases to prosecute a more accurate investigation of the digestive process. If the examination of the gastric juice shows a diminution of hydrochloric acid, this may be artificially supplied. We prescribe, thirty to sixty minutes after each meal, ten to fifteen drops of dilute hydrochloric acid in half a glass of water. The benefit to be thus derived must not be overestimated. In appropriate cases, however, its influence is favorable. One may also make a trial of pepsin—pepsini Germanici solubilis, seven and a half grains (grm. 0·5) after each meal—or of pancreatin. The popular wines of pepsin are not to be recommended, because of the alcohol they contain.

On the other hand, alkalies have been long employed in chronic gastric catarrh with good effect. Their value is more easily understood to-day, because we know (see the appendix to this chapter) that many disturbances of digestion are associated with an excessive production of acid; still it can not be denied that even in other cases alkaline remedies are sometimes beneficial. This is probably because the alkalies may contribute to the neutralization of abnormally formed acids; because they promote the discharge of the gastric contents; because they tend to clear away the mucus; and because, finally, as has been experimentally shown, bicarbonate of soda, chloride of soda, and carbonic-dioxide gas exercise a stimulating influence upon the secretion of the gastric juice. As is well known, the alkalies are mainly prescribed in the form of alkaline mineral waters. The springs of Carlsbad possess the greatest reputation. Those at Ems, Kissingen, Tarasp, and Vichy also deserve to be mentioned as health resorts for patients with gastric trouble. A good portion of the benefit at these places is, to be sure, dependent upon the fact that many patients are much more apt to follow a strict regimen when they make use of a particular "cure" than when they remain at home.

If the character of the vomitus or of the gastric contents indicates abnormal processes of fermentation in the stomach, the best remedy is probably a methodical lavage of the stomach. We may also try drugs which prevent fermentation, the most important of which is again hydrochloric acid in rather large doses. Other remedies for this purpose are salicylic acid in powders of eight grains (grm. 0·5) each; creasote, two or three half-grain pills (grm. 0·03) a day; and benzine, twenty drops in milk.

The best drugs to stimulate the secretion of gastric juice are the bitters. It is this property which has earned them the name of stomachic tonics. Compound tincture of gentian, tincture of nux vomica, tinctura amara, and tinctura calami, P. G., quassia and columbo—these are the most employed. In general, however, they are not very efficient. An excellent tonic in many of these cases is cundurango bark. A decoction may be made (15 parts to 200 of water); or we may write for powders of seventy-five grains (grm. 5) each, one powder being made into one or two cups of infusion by the patient himself.

A few remedies remain to be mentioned, which are said to exert a direct beneficial influence upon the catarrh, and which many physicians extol highly. Their efficacy, however, is somewhat problematical. We refer chiefly to subnitrate of bismuth, sulphate of zinc, and nitrate of silver.

Certain symptoms may demand especial treatment, such as vomiting. It will usually yield to regular and persistent washing out of the stomach. Other remedies are small bits of ice, and minute doses of opium or chloral. Potassic bromide may also be tried.

Violent gastralgia requires narcotics, such as hydrocyanic acid and morphine. "Sour stomach" may be relieved by a pinch of bicarbonate of soda, or of calcined magnesia. Persistent anorexia may yield to the bitters mentioned above, or to small doses of quinine. If the bad taste in the mouth is annoying, the mouth should be frequently rinsed out with Seltzer water, a one-per-cent. solution of carbolic acid, or five drops of tincture of myrrh to a glass of water. For habitual constipation, enemata, or the various mineral waters, are good; also Carlsbad salts. In obstinate cases pills of rhubarb or aloes may be employed. Still, we ought never to forget that the infrequency of the stools is often merely a natural consequence of the scanty diet, and that it is therefore possible to do harm with our purgatives. Iron is often prescribed for the concomitant anæmia; but it should be employed cautiously, for it is often ill borne in gastric disease.

APPENDIX.

HYPERACIDITY AND HYPERSECRETION OF THE GASTRIC JUICE. ACID DYSPEPSIA.

It was formerly believed that in most cases of disturbed digestion there was a diminished secretion of the gastric juice, and in particular of hydrochloric acid; but the newer investigations of Reichmann, Riegel and others, have taught us that it is by no means rare to find in these cases an exaggeration of the production of acid and an increased secretion of the gastric juice. Hyperacidity is the term applied to that condition in which at the time of digestion an abnormally large amount of hydrochloric acid (0.4 to 0.6 per cent.) is found in the gastric juice. "Hypersecretion" refers to that anomaly where at other times than when digestion is taking place, when the stomach is empty, there is invariably to be found a considerable amount of strongly acid gastric juice in the stomach. Hyperacidity may exist independently of hypersecretion, while hypersecretion is usually conjoined with excessive secretion of acid. Probably, therefore, hypersecretion is merely an exaggeration of those processes which lead to hyperacidity.

Hyperacidity may usually be demonstrated merely by obtaining an unusually strong reaction for hydrochloric acid with methyl-violet in the gastric contents, but a more accurate diagnosis of course requires a quantitative estimation of the acidity. The recognition of hypersecretion is not easy. If we introduce a tube into an empty stomach—the best time being early in the morning—we shall find that the stomach is never empty, but that it invariably contains a larger or smaller amount of strongly acid liquid unmixed with particles of food, and that this liquid evinces its character as gastric juice by possessing an abundant amount of hydrochloric acid and digestive properties.

As to the causes of hyperacidity and hypersecretion, little as yet is known. They are perhaps occasioned by conditions of nervous irritation, or perhaps by special forms of catarrh. It is certain that gastric ulcer is very frequently associated with hyperacidity (see below).

As we have already mentioned, increased production of acid and of gastric juice is by no means rare, and therefore it is of great practical importance. These conditions occasion quite severe dyspepsia, which is usually termed *dyspepsia acida.* The symptoms of which such patients complain are mainly pain and extremely sour eructations, often associated with pyrosis. The pain is usually described as burning, and it is located mainly in the region of the stomach, but usually radiates toward the navel. It occurs sometimes independently of the ingestion of food (hypersecretion?), sometimes an hour or an hour and a half after eating (hyperacidity?). The extremely sour eructations take place usually at the time of digestion. Vomiting is not very frequent, but it may occur. The appetite is in many cases diminished, but in others it is not much affected. Often there is complaint of great thirst. The general nutrition in many cases remains tolerably fair. In other cases the patients become decidedly emaciated.

Although the above symptoms, and in particular the sour eructations and the burning pains in the stomach, often of themselves arouse a suspicion of acid dyspepsia, yet the absolute diagnosis requires investigation by means of the stomach-tube. The particulars relating to this have been already detailed. It remains only to add that in testing the digestive power we usually find a satisfactory digestion of albumen, but a very incomplete digestion of starchy food. This is probably in main part dependent upon the fact that the excessive acidity of the gastric juice inhibits the amylolytic action of the saliva in the stomach (Ewald and Boas), but it may also partially result from the hindrance to the propulsion of the food from the stomach, because of a spasmodic closure of the pylorus (Riegel).

The treatment of acid dyspepsia consists first in the employment of alkalies, such as bicarbonate of soda, in sufficient doses, say seventy-five grains (grm. 5), several times a day. The use of alkaline waters, such as Carlsbad, is also often attended with good results, particularly as these dilute the gastric juice and thus diminish the irritation caused by the acidity. For this reason many patients find great relief from taking warm tea at the time of the pain. If the discomfort is great, the most efficient remedy is certainly a methodical lavage of the stomach. With regard to the best diet, we may allow flesh in a sufficient amount, while carbohydrates as above mentioned must be enjoyed in moderation.

CHAPTER III.

PHLEGMONOUS GASTRITIS.

(*Purulent Inflammation of the Stomach.*)

PURULENT inflammation of the stomach is very rare, and little is yet known about it. In most cases no special causes for it have been ascertainable. It is occasionally one of the symptoms of grave pyæmic or puerperal inflammation.

Two forms are distinguished—a diffuse and a limited variety. The latter is equivalent to gastric abscess. The submucous layer is almost invariably the chief seat of suppuration. From this starting-point the process invades the muscular and serous coats on the one hand, and the mucous membrane itself on the other.

The usual symptoms are violent gastric derangement, with pain and vomiting, high fever, and the indications of constitutional infection, namely, headache, delirium, and general prostration. Sometimes the disease is quickly fatal, sometimes it runs a more chronic course. The few cases in which recovery has been reported are somewhat obscure.

The disease can never be diagnosticated with absolute certainty. Treatment must be purely symptomatic. Ice, both internally and externally, and the narcotics, are chiefly employed.

CHAPTER IV.

GASTRIC ULCER.

(Simple or Round Ulcer of the Stomach.)

Ætiology.—Since Cruveilhier gave the first accurate description of gastric ulcer, numerous explanations of its occurrence have been propounded. Even yet there is no universally accepted view. There is, however, one point about which most authors are agreed, namely, that the ulcer is due to a self-digestion of the stomach at one limited spot. Hence it is frequently termed "peptic ulcer of the stomach."

Apart from the inherent vital resisting power of the cells, the reason that the stomach is not continually being digested by its own juices is, as we all know, chiefly the alkalinity of the blood, which is constantly coursing through the mucous membrane. Wherever the circulation is in any way interfered with, we should therefore expect to find, and we do find, that the mucous membrane begins to be digested.* If, as a result of inflammation, a minute hæmorrhage occurs at any point, the little area thus deprived of its blood-supply is at once digested. This is what is called a "hæmorrhagic erosion." The experiment of occluding the arterioles of the gastric mucous membrane with emboli has been tried by Panum and Cohnheim. The resulting hæmorrhagic infarctions were transformed into ulcers. Just what the essential conditions are, in man, under which a local disturbance of the circulation and a consequent round ulcer of the stomach are produced, we are thus far confined to conjecture. Virchow assumed that most cases were the result of thrombosis or embolism of the minute blood-vessels, caused by various diseases. Klebs suggested the possibility of a local spasm in the vessel. Böttcher has succeeded in detecting numerous colonies of micrococci in the margins of gastric ulcers, and regards them as the cause. As we have already said, however, no one of these views has been universally accepted.

It may be that in many cases some local injury of the mucous membrane is the original cause of the ulceration. Such possible causes are burns or mechanical lesions, including blows over the stomach. But even then we are unable to explain why the ulcers should spread laterally and downward. For all gastric ulcers experimentally produced, whether by embolism, contusion, burning, or even cauterization (as practiced by Quincke), exhibit a decided tendency to rapid healing. It has therefore been suggested that the further extension of the ulcers, in such cases, is due to an abnormally great acidity of the gastric juice (*vide infra*).

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* After the circulation is terminated by death, this digestive process at once begins, producing the softening of the stomach, or gastromalacia, frequently found at autopsies. There has been much discussion as to the origin of this condition, but it now seems indubitable that it is in every instance a post-mortem change.

Ulcer of the stomach is commonest in young adults, between seventeen and twenty-five years of age. It is rare in children; less so in older people. The disease is noticeably more frequent in females than in males. It is more prone to attack the weakly, anæmic, and chlorotic than the vigorous.

Pathology.—The ulcer is usually of a circular shape. Its borders are sharp; the walls often slope inward, giving the ulcer the form of a shallow funnel. Its base is almost always perfectly clean. If superficial, it does not extend farther than to the muscular coat, but it may be deep enough to expose the serous membrane, or even to perforate it (*vide infra*). The size varies greatly. Some are hardly as large as a pea; others may measure ten to fifteen centimetres in their greatest diameter. As to position, most of them are found near the pylorus. They attack the posterior wall of the stomach, particularly the neighborhood of the lesser curvature, more frequently than the anterior wall. As a rule, we find but a single ulcer, although exceptions to this statement are not very rare.

If an ulcer of any size heals, a scar is formed, with radiating lines and often of considerable size. Cicatricial contraction may alter the shape of the stomach considerably. Pyloric ulcers may leave scars which eventually produce stricture of the pylorus and consequent dilatation of the stomach.

If the ulcer attacks the serous membrane, perforation may finally result, unless an adhesive peritonitis attaches the stomach, at the point threatened, to some neighboring organ. The ulcers being usually on the posterior wall of the stomach, it is oftenest the pancreas to which the stomach becomes adherent. In other instances it is the liver, transverse colon, diaphragm, or spleen. If the ulcer penetrates organs to which the stomach has become attached in this way, the inflammation extends, so as to cause empyema, for example, or hepatic abscess; or the pleura, lungs, pericardium, or transverse colon may be perforated. We shall revert to this subject under the symptomatology of gastric ulcer.

The ulcer may cause erosion of a blood-vessel, and thus give rise to one of the most important symptoms of the disease, namely, gastric hæmorrhage.

Clinical History.—There may be absolutely no symptoms. It is not a rare thing to find at autopsies a still active ulcer of the stomach, or the cicatrix left by one, in subjects who never had during life any gastric disturbances whatever. Nor is it exceptional for a person suddenly to exhibit grave symptoms, like gastric hæmorrhage, or peritonitis due to perforation, where there has been no reason previously to apprehend the existence of an ulcer.

In other instances the ulcer does, indeed, give rise to symptoms, but they are not sufficiently characteristic to point to the correct diagnosis. There are anorexia, occasional gastralgia, vomiting, and eructations—symptoms which might with equal probability be referred to a simple chronic catarrh. In fact, it seems likely that the ulcer has little share in their production, and that they are mainly due to a co-existing catarrh. In these cases, also, grave symptoms consequent upon the ulceration may suddenly arise.

In a third class of cases there are symptoms which are to a certain extent characteristic, and lead with more or less definiteness to the true diagnosis. These "symptoms of ulcer" are chiefly a peculiar epigastric pain, which is usually intermittent, and vomiting, or, what is yet more distinctive, the vomiting of blood, or hæmatemesis. These symptoms and their diagnostic value we must now consider in detail.

Pain in the stomach is one of the most frequent symptoms of round ulcer. Its forms are very diverse. Often the patient complains only of a diffuse, painful sensation of pressure referred to the entire region of the stomach. This may be uninterrupted, or it may occur only after meals, or after excessive exertion, or as the result of some other special cause. This sort of pain is the least diagnostic of any, inasmuch as exactly similar sensations may be caused by a simple chronic catarrh. More characteristic of ulcer is a decided cardialgia—that is, a very violent pain, coming on at intervals like neuralgia. It is described as "cutting," "tearing," "boring," and the like. The pain may come on irregularly at any hour; or it may occur with considerable regularity at the end of a definite time after eating, say half an hour or an hour. It is felt chiefly in the epigastrium, but not infrequently extends toward the umbilicus, backward toward the vertebræ, into the thorax, or even into the upper extremities. In many instances a marked sensation of thoracic oppression accompanies it. Another characteristic point is that a change of position may sometimes affect the severity of the pain. It is sometimes observed that the patient, when lying upon his right side, feels violent pain, which is at once relieved by changing to the left side, probably because the ulcer is located near the pylorus. An attack of cardialgia may last for a few minutes or for several hours. It is commonly thought to be due to the direct irritation of the ends of nerves lying exposed at the base of the ulcer. As an isolated symptom, this cardialgia can not be distinguished from the purely nervous variety, but, taken in connection with other symptoms, it is often a great aid to diagnosis. We should add that precisely similar attacks of cardialgia may be produced by the scars of ulcers which have already entirely healed.

A third variety of pain may be observed in gastric ulcer. The suffering may be localized in a very limited area. Such pain is thought to be due to irritation of the floor of the ulcer by food, or to its edges being pulled upon during the movements of the organ. It generally comes on after eating, and ceases if the stomach is perfectly quiet. In position, this pain is generally epigastric, but sometimes it is umbilical, or even, now and then, more toward the back. In many cases of gastric ulcer there is also tenderness on pressure in a quite sharply defined area and at one particular spot. Most authors regard the accurately localized pain as the most nearly pathognomonic; but it must be said that it is decidedly the least frequently exhibited of any. Transitional forms and combinations of the various kinds of pain are often observed.

Vomiting is a very frequent symptom of gastric ulcer, but if the vomitus is composed of nothing except food, or food mixed with mucus or bile, the symptom is not at all characteristic. In quite a large proportion of cases, however (about one third), hæmatemesis occurs either once or repeatedly. The vomiting of a considerable amount of blood is beyond doubt the most important factor in diagnosticating gastric ulcer. Often the sudden occurrence of a decided hæmatemesis enables one to diagnosticate a gastric ulcer with an approach to certainty. If there is no vomiting of blood whatever, there is much more doubt about the existence of an ulcer.

Hæmatemesis is frequently the symptom which first leads the patient to apply to a physician. Up to this time he may have felt perfectly well, or, although there may have been some gastric derangement, he has not thought anything of it. The patient suddenly becomes faint, perhaps while he is pursuing his regular occupation, or it may be at night. He feels dizzy, and it looks black before his eyes. Then he has nausea, and finally is obliged to vomit. The vomitus is either pure blood, or a mixture of blood and food. It is partly coagulated, and often has a rather dark or blackish color. This change in color, as well as the coagulation, is due to the action of the gastric juice. The amount varies greatly in different cases: there may be a quart or more. Not infrequently blood is repeatedly vomited either at short intervals or on successive days. Part of the blood escapes through the pylorus, so that, after a profuse gastric hæmorrhage, blood is sure to be found in the stools. In them it is black and tarry. Exceptionally it happens that all the blood, beyond what is absorbed from the intestinal canal, passes off *per anum*, so that none whatever is vomited. In such cases it is often a difficult matter to locate the hæmorrhage.

The consequences of gastric hæmorrhage depend, of course, chiefly on the amount of blood lost. Sometimes, although fortunately rarely, a large blood-vessel is eroded and the patient dies. This event may be sudden, or it may occur more gradually under the influence of repeated hæmorrhages and after a few days, during which all the symptoms of acute anæmia are exhibited. On the other hand, the loss of blood may be so insignificant as to produce no especial symptoms. In most instances life is not actually threatened, but yet the signs and results of a more or less marked general anæmia are clearly visible.

In such cases the patient feels extremely exhausted, and at once takes to his bed. He has also all the subjective symptoms of cerebral anæmia. There are vertigo, tinnitus aurium, specks before the eyes, frequent gaping, and sometimes headache. To assume an erect posture aggravates the disturbance. There is usually excessive thirst. Now and then a temporary amaurosis has followed an excessive hæmorrhage.

Objectively, we notice at once the excessive pallor of the skin, particularly of the face. The lips and conjunctivæ are also blanched. The pulse is rapid, and often ill sustained. For some days there may be anæmic murmurs over the heart, and there is a distinct sound to be heard in the femoral arteries. A moderate rise of temperature is very common. This is known as the anæmic fever. The urine is pale, and usually rather abundant. Its specific gravity is not infrequently relatively high, namely, 1015 to 1020. All these symptoms are directly referable to the loss of blood, and will be discussed with greater detail in the section on anæmia.

If the hæmorrhage is not repeated, the patient gradually regains his strength. To be sure, the pallor usually persists for a long while, but the disagreeable symptoms gradually abate. Where gastric discomfort has existed previously to the hæmorrhage, it often disappears entirely after it—a circumstance which is probably in part due to the excessive caution of the patient thereafter. At the end of a few weeks the patient often feels perfectly well again; and, indeed, recovery is not infrequently complete and permanent. In other cases, however, the symptoms of ulcer return, sooner or later.

Other symptoms than those already discussed are more uncertain. Anorexia, eructations, and obstinate constipation may be present, or may be wholly wanting. In cases of suspected ulcer, it is usually inadvisable to introduce the stomach-tube for diagnostic purposes, because bleeding or perforation might result. Investigations have shown that frequently there is no serious derangement of digestion. It is interesting, however, that very often the gastric juice is excessively acid (hyperacidity, Riegel). If there be imperfect digestion, it is probably due not so much to the ulcer as to a coincident gastric catarrh. The general nutrition often suffers comparatively little, unless there are persistent anorexia and vomiting.

An event which has been already mentioned under pathology—namely, perforation of the ulcer—is of great clinical importance. It would be impossible to particularize here all the possibilities incident to it. We shall confine ourselves to the two most important because most frequent varieties of perforation: (1) into the peritoneal cavity, causing peritonitis, and (2) into the left pleura, or left lung.

Perforation into the peritoneal cavity leads almost invariably to a quickly fatal peritonitis. When the ulcer has previously caused few symptoms, if any, the excruciating abdominal pain, tympanites, vomiting, collapse, and sudden death of peritonitis may abruptly supervene upon a state of apparently perfect health. It is the exception to have the peritonitis limited. This is more apt to occur if adhesions have been formed previously. An encapsulated abscess then results, which points either into the intestine or externally, and may rarely terminate in complete recovery.

Perforation into the left pleural cavity we have observed repeatedly. It causes a purulent or septic pleurisy on that side, and pulmonary gangrene may develop at the same time or later, as a result of perforation into the lung. Whenever we meet a case of apparently spontaneous, left-sided empyema, we should at any rate always think of the possibility of gastric ulcer.

The general course of round ulcer of the stomach varies, as we can see, so greatly in different cases that it can not be depicted simply. Complete recovery is by no means rare. In other cases the symptoms persist for years with varying intensity. We have already spoken of the hæmorrhage and perforation which may suddenly intervene, and of their significance. Relapses are not infrequent, even after apparent recovery. If the ulcer cicatrizes, the scar itself may give rise to persistent disturbances; there may be obstinate cardialgia, or, if the scar is at the pylorus, dilatation of the stomach (*vide infra*) may gradually be developed.

Diagnosis.—The diagnosis can be made only when the above-mentioned characteristic symptoms are present. Of these, hæmatemesis is by far the most significant, for it is with very few exceptions the result of gastric ulcer. Particularly is this true of individuals under middle age.—But how shall we determine whether the blood ejected did not come from the nose or the lungs, rather than the stomach? The answer is not always easy. If an epistaxis occurs at night, a part of the blood often flows back into the naso-pharynx, and, being swallowed, excites vomiting, so that a gastric hæmorrhage is suggested. It is also very important for the physician to remember that blood may be "vomited" in hysterical cases. If there is also nervous cardialgia, one might easily be misled to assume the existence of a gastric ulcer. Other hysterical symptoms (see the chapter on hysteria) and the character of the blood will usually guide us to a correct diagnosis. The blood probably comes in most cases not from the stomach, but from the gums or pharynx, and it is consequently of a comparatively bright color and rather watery.

The diagnosis between gastric and pulmonary hæmorrhage in doubtful cases depends on the following factors: 1. The previous condition of the patient—whether he has had cough, expectoration, and other pulmonary symptoms, or, on the other hand, gastric pain and vomiting. 2. On the character of the hæmorrhage, whether accompanied by vomiting or by cough. But there may have been both. Violent vomiting may excite a cough; and, on the other hand, blood which has been coughed up may be in part swallowed and induce vomiting. 3. On the character of the blood: if from the lungs, it is usually bright-red and frothy, containing bubbles of air, with few clots, and of alkaline reaction. In gastric hæmorrhage it is usually dark, mixed with food, partly clotted, and acid in reaction. 4. On the results of physical examination. In this, of course, we must be extremely cautious after a hæmorrhage, lest the movements of the patient excite fresh bleeding; and yet we may be able, from the general condition of the patient, or from dullness at the apices, or moist râles, to demonstrate pulmonary disease. If the blood came from the stomach, we usually detect nothing but the signs of anæmia. 5. The subsequent symptoms. In cases of pulmonary hæmorrhage there is almost sure to be an expectoration, for the next few days, either of pure blood or of matter stained with blood; and, in gastric hæmorrhage, the next dejection will almost certainly be black, from the presence of decomposed blood. In doubtful cases, the appearance of blood in the stools almost invariably settles the question in favor of gastric hæmorrhage.

In cases of gastric ulcer, unaccompanied by hæmatemesis, the diagnosis is only more or less probable, not certain. The existence of cardialgia and localized pain in the stomach must always make us consider the possibility of an ulcer, but we can not be positive about it.

The differential diagnosis between gastric ulcer and purely nervous cardialgia, and between ulcer and cancer, will be discussed later in the appropriate chapters.

Prognosis.—The dangers in ulcer of the stomach, particularly hæmorrhage and perforation, have already been spoken of. Whether these misfortunes will actually occur in any individual case, and when, we can not determine.

There can be no doubt that a large number of ulcers heal perfectly; but, as we have already said, even the resulting scar may cause trouble. This possibility must be borne in mind. Of these persistent symptoms, cardialgia is the most frequent. Dilatation is also possible. And, finally, it is probably not a very rare event that cancer eventually develops in the floor of the old ulcer. We shall revert to this matter under cancer of the stomach.

Treatment.—If the diagnosis of gastric ulcer is evident, or if the symptoms are of such a nature that there is a justifiable suspicion of an ulcer, the patient should be urgently advised to submit himself to careful and methodical treatment; for it is only by means of a sufficiently persistent and properly conducted treatment that we can hope for good therapeutic results in ulcer of the stomach.

The essential condition is, that the patient should keep his bed for the first part of the treatment, at least for two or three weeks. Complete bodily rest is certainly an important assistance in promoting healing of the ulcer. The patient should also have moist compresses, or, still better, as Leube has advised, warm poultices applied all day long to the epigastrium. These poultices give great relief to the pain. In the third place—and this is probably the important matter—the patient must keep strictly to an accurately prescribed diet. Every mechanical and chemical irritation of the floor of the ulcer must be avoided, and therefore, at first, liquid nourishment alone is to be permitted. It is best to allow the patient, for the first ten days, milk, bouillon, and at the most some eggs and thoroughly moistened bread. A very suitable article of diet is soup made out of the Leube-Rosenthal solution of meat. In the fourth and last place, we should consider the hyperacidity of the gastric juice, which, as has been already said, exists in the case of ulcer. The patient therefore should be given every day a tablespoonful of Carlsbad salts, dissolved in a pint of warm water, to be taken in three or four divided doses during the forenoon and afternoon. If treatment of this sort is strictly carried out, the discomforts of the patient, his pain and vomiting, will abate in a few days. After some ten days have passed, if the patient feels well, he may cautiously begin to eat such food as the brain of calves boiled, boiled pigeon, boiled fowl, and flour gruel. If this dietary makes the pain return, we must fall back upon the first kinds of nourishment. Usually, however, the above-named foods are well borne, so that after another period of eight or ten days the patient may be allowed some beef, such as sirloin or beefsteak, shaved raw ham, and, later, also underdone roast meat, game, veal, fish, and the like. The more severe the earlier symptoms were, the slower and more cautiously will one proceed with changes in diet. If desired, one may also employ either artificial nutriments, such as the meat peptones of Koch and of Kemmerich, and leguminose, and also various kinds of infant foods.

The above methods will usually accomplish all that we can hope to do, and it is not till they prove unavailing that, while persisting in our dietetic treatment, we should try the other remedies, whose efficiency is often extolled but has never been demonstrated. Among these, the favorite is subnitrate of bismuth in powders of five to fifteen grains (grm. 0·30–1·00) each, mixed with sugar. To this we may add a sixth of a grain (grm. 0·01) of morphine, if there is cardialgia. One powder is to be given three times a day, fifteen minutes before meals. Nitrate of silver is also frequently administered in pills of one sixth of a grain (grm. 0·01) thrice daily; or in a solution of 1 to 400, of which the dose is one or two teaspoonfuls.

Finally, there are special symptoms which may need to be relieved. Violent pain, when not yielding to regulation of the diet, demands morphine. We may also try warm or cold applications, or chloroform, externally. Gerhardt recommends three or four drops of liquor ferri chloridi in a wineglass of water, for the relief of pain.

Excessive vomiting and persistent nausea are likewise to be combated by the narcotics. Opium is the best; morphine, chloral, and bromide of potash may also be tried. If there be no improvement, we may try creasote, or three or four drops of tincture of iodine. At the first appearance of blood in the vomitus the greatest bodily quiet and most careful dieting is absolutely indispensable. For the first day or two it is best to allow nothing except ice-cold milk and bits of ice in the mouth to appease the burning thirst. The patient must lie as quietly as possible in bed. A flat ice-bag, not too heavy, should be placed on the epigastrium. In case of persistent nausea or eructations, small doses of morphine are to be prescribed. We must wait till four or five days after the hæmorrhage before we cautiously increase the amount of nourishment, which must still be liquid.

If peritonitis appears as the result of perforation, the best means to try are the outward application of ice to the epigastrium and the internal use of opium in large amounts—that is, half a grain to a grain (grm. 0·03–0·05) every one or two hours. Unfortunately, however, the cases are exceptional in which the peritonitis does not become general. Then, possibly, operative interference might be of some avail. Other measures are almost hopeless. We may try to alleviate the pain by narcotics, but it is very rarely that we can prevent a fatal issue.

[In cases of recent hæmorrhage or markedly painful digestion, the editor has pursued the following plan of treatment with apparently good results: The most powerful agent in the promotion of cicatrization is rest. Absolute rest in bed is therefore enjoined, with the secondary end in view of reducing the demands of the system. The patient is then fed exclusively by the rectum for at least one week, generally for two weeks, nothing but a little water being given by the mouth. A simple cleansing enema is given once daily; and at six-hour intervals a nutrient enema, six to eight ounces in bulk, and composed of one or two raw eggs, an ounce of expressed beef juice, and fully peptonized milk, is administered. Tolerance by the rectum is promoted by the addition to the enema of a few drops of laudanum. After one or two weeks small quantities of milk at frequent intervals are given by the mouth, and the rectal alimentation is gradually diminished as that of the stomach is increased. Pain usually ceases immediately on the cessation of gastric ingestion, and the loss of weight during a fortnight of rectal feeding is surprisingly small.]

APPENDIX.

MELÆNA NEONATORUM.

We desire here also to mention the condition known as *melæna neonatorum.* In rare instances during the first week after birth, hæmorrhages occur from the stomach (hæmatemesis), which are sometimes associated with dark, bloody stools. As a rule the hæmorrhages are repeated, so that after a few days death occurs with all the phenomena of anæmia; but, even after what seem to be the worst symptoms, recovery may exceptionally take place.

The cause of melæna neonatorum is by no means clear, but in some of the cases small ulcers are found in the mucous membrane of the stomach and intestine, as to the origin of which we have no certain information. Passive congestion, embolism, and infection have been suggested as causes.

The treatment consists in the application of ice poultices to the abdomen. Liquor ferri perchloridi has been also recommended, in doses of one drop every two hours in oatmeal-water.

CHAPTER V.

CANCER OF THE STOMACH.

Ætiology.—We can not here discuss the ætiology of carcinoma in general, and we shall therefore merely enumerate the factors which experience has shown to favor the development of cancer in the stomach.

Age has a remarkable influence. Gastric cancer is decidedly most frequent late in life, between the fortieth and sixtieth year. Still it is occasionally seen in younger persons. We have ourselves seen several cases in persons between twenty-two and twenty-five years of age.

Sex is of no importance.

Heredity has a slight but undeniable influence. The most famous example of the transmission of cancer is presented by the family of Napoleon.

The relation of gastric cancer to other antecedent diseases of the stomach is very interesting. Whether frequent errors in diet or the use of alcohol increase the liability to this disease is uncertain. On various sides attention has been called to a possible, and as it seems to us very probable, connection of gastric cancer with antecedent ulcer. Not only at the bedside, but at the post-mortem table, a relatively large number of cases have been observed where cancer had developed on the floor of an old (and usually cicatrized) ulcer. Hauser made the interesting discovery of a-typical growths of epithelium in the scars of gastric ulcers—a phenomenon which points strongly toward the relation suggested.

Pathology.—The stomach is a favorite seat for cancer. About a third of all cases of cancer are gastric. The parts of the organ most often attacked are the pyloric end and the lesser curvature. Less frequently the cardiac end and the fundus suffer.

The new growth takes the form either of a circumscribed tumor or of a diffuse infiltration, thickening the walls. The disease invariably originates in the mucous layer, extending thence into the submucous and muscular coats. The connective and muscular tissues in the neighborhood of the cancer are quite often considerably hypertrophied.

Histologically, gastric cancer is of the cylindrical-cell variety, starting from the glandular epithelium. The soft tumors are termed medullary; the firm and hard, scirrhous or fibroid. The medullary cancers are particularly apt to be quite extensively broken down on their exposed surface, thus forming what are known as cancerous ulcers. This seems to be mainly the result of the gastric juice acting on the superficial and insufficiently vascularized portion of the tumor. The base of these ulcers is usually clean, as we should expect from the mode of their production. In many cases of rather young subjects, and also in others, we find colloid cancer. This form also may appear either in nodules or as a diffuse growth, infiltrating the tissues. As to metastasis of gastric cancer, *vide infra.*

Clinical History.—Most cases of gastric cancer exhibit a combination of grave digestive disturbances with a relatively rapid loss of flesh and strength. Now and then the gastric symptoms assume less prominence. The chief sign of disease is a constantly progressive marasmus or anæmia, the true cause of which is either entirely latent or not unmistakably recognizable till late in the illness.

Some of the gastric symptoms are not very characteristic. They merely show that digestion is disordered. There is loss of appetite and distress after meals. The patient complains of a disagreeable sensation of pressure in the epigastrium, increased by food. Sometimes this amounts to actual cardialgia. Many patients are annoyed by frequent eructations. Occasionally vomiting is troublesome; in other instances there is scarcely any. The vomitus may contain nothing but mucus and the ingesta; or it may assume, from the admixture of blood, a very characteristic and somewhat pathognomonic appearance.

Free gastric hæmorrhage and consequent hæmatemesis is exceptional, or at least much less frequent than in ulcer of the stomach; but the vomitus often contains decomposed blood, and in many cases this will be for a time almost a constant appearance. Most of the ulcerated cancers bleed frequently, a little at a time. The escaping blood is broken up by the action of the gastric juice, its red hæmoglobine being transformed into the black hæmatine, which produces that "coffee-ground" or "chocolate-colored" appearance of the vomitus so significant of the disease. In such cases the presence of blood can be demonstrated conclusively by the spectroscope, or by means of the so-called hæmine reaction. To perform this, a small portion of the vomitus is heated to boiling in a watch-glass, having first been mixed with a little glacial acetic acid and a few crystals of common salt. A drop of this is then allowed to dry upon an object-glass, when the rhombic crystals of hæmine are quickly formed. These crystals have a dark-brown color, and are easily recognized (*vide* Fig. 35). We should mention that in the case of ulcerating carcinoma ventriculi the vomitus may have so foul an odor that it may even be regarded as stercoraceous.

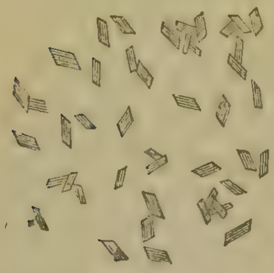
Fig. 35.—Hæmine crystals.

Immediate microscopic examination of the vomitus may reveal red blood-globules. Other characteristic constituents are rare. It is only exceptionally that bits of cancer can be demonstrated. Of course, if seen, they end all doubt. Sarcinæ are often found, just as in other gastric diseases.

Of great diagnostic importance is the investigation of the gastric juice (*vide supra*, page 378). As Von den Velden first pointed out, in most cases of gastric cancer there is no free hydrochloric acid in the gastric juice, or at least none to be detected by means of the well-known reaction with methyl-violet and the other reagents; while on the other hand lactic acid is not infrequently abundant, and may even be present for a long while after food has been ingested. The digestive power of the gastric juice in persons afflicted with carcinoma is consequently, as may be easily demonstrated by experiment, decidedly below normal. The reason of this frequent absence of free hydrochloric acid in carcinoma is not yet clear. Probably this phenomenon does not depend directly upon the carcinoma, but upon the catarrh of the rest of the gastric mucous membrane which is usually present in cases of carcinoma; and again, when the gastric mucous membrane is much atrophied, as has likewise been repeatedly shown to be the case, the production of hydrochloric acid ceases.

Physical examination of the stomach, and palpation in particular, are of the greatest value. In a large number of cases the new growth can be more or less plainly felt through the abdominal walls, as a hard, nodular tumor. The tumor is situated in the epigastrium, in a majority of cases, but it may be lower down or more to one side, according to the region attacked. It should be remembered always that a permanent tumor may essentially alter the position of the stomach to which it is attached. As an illustration, a case of pyloric cancer, which we saw, with secondary dilatation of the stomach, had resulted in such a displacement of the pylorus that the tumor was to be felt through the abdominal walls, about a hand's breadth above the symphysis pubis. Sometimes the tumor varies in position according to the fullness of the stomach. The effect of the respiratory movements upon the tumor varies. In some cases, particularly if the tumor is adherent to the liver, it can be plainly felt to move downward with each inspiration, while in other cases it remains perfectly stationary.

In a minority of the cases no tumor can be felt at any time. Under such circumstances the diagnosis can not often be definitely established. The tumor is undiscoverable, first, in most cases of diffuse cancerous infiltration of the stomach-walls. We may, indeed, notice an increased sense of resistance and hardness in the epigastrium, but can not refer this condition with certainty to a new growth. Secondly, the new growth may extend chiefly inward, toward the cavity of the organ, and may thus escape detection. And, finally, the tumor may be so concealed by the liver or the edge of the ribs that it is inaccessible to the touch. Such cancers as attack the cardiac extremity, the posterior wall, or the lesser curvature of the stomach, are particularly apt to be out of reach of palpation.

Percussion of the cancer rarely gives flatness, but instead a muffled tympanitic resonance. This is sometimes an influential factor in the differential diagnosis from cancer of the liver.

The physical examination sometimes yields secondary evidences of cancer, in addition to those which are due directly to the new growth. In most cases of pyloric cancer a resultant dilatation of the stomach can be demonstrated.

Next in importance to the gastric symptoms is the disturbance of general nutrition. Loss of flesh is not rarely the very first symptom which calls the patient's attention to his disease. This wasting is observed earliest in cases which are attended with anorexia and vomiting. The patient also gradually takes on that familiar cachectic look which is characteristic of most cases of cancer. Some patients become excessively anæmic. The skin acquires a waxy pallor, and there are all the symptoms which result from great anæmia, such as cerebral symptoms and functional cardiac murmurs. Sometimes the blood itself presents decided peculiarities in such cases. Thus we may find microcytes and poikilocytes in it. Gastric cancer and pernicious anæmia *(q. v.)* have been repeatedly confounded. The cause of this excessive anæmia is not always perfectly clear. In one such case we made the interesting discovery of extremely abundant metastatic cancer in the bones. As the bone marrow is known to have something to do with the production of the blood, it may be that the anæmia was due to this abnormal condition. At any rate, the grave anæmia which results from cancer, and sometimes also from other chronic diseases of the stomach, particularly gastric ulcer, can not be regarded in just the same light as are the loss of flesh and the cachexia. Often the anæmia is very great before the general nutrition has suffered much impairment.

When the disease is pretty well advanced there may be moderate œdema of the ankles and the back of the hands. This is to be explained, as in other cachectic and anæmic conditions, by the impaired nutrition of the vascular walls, the hydræmia, and the cardiac weakness.

Special derangements of other organs are relatively infrequent. Metastatic cancer is of importance. It attacks the liver chiefly. If the hepatic new growth is considerable it may quite overshadow the primary cancer. Secondary carcinosis of the peritonæum is also apt to cause marked symptoms, such as ascites and abdominal pain. Secondary cancer may also involve the mesenteric and retroperitoneal lymph-glands, the lungs, and other organs, but does not usually give rise to striking symptoms when so situated. It may be added that patients with cancer of the stomach sometimes present a swelling of the lymphatic glands above the clavicle, especially on the left side, and that many authors regard this fact as of some value in making a diagnosis.

Direct extension of the new growth into neighboring organs is not very frequent. We will venture to mention one case which we saw, on account of its great rarity. The new growth caused adhesion of the anterior wall of the stomach to the abdominal walls, and then, penetrating through them and the skin of the epigastrium, finally appeared as a tumor, of about the size of one's fist, projecting outward. If a cancer ulcerates, it may destroy all the layers of the stomach, and result in perforation and secondary peritonitis; or, if previous adhesions have been formed, the perforation may open up an abnormal communication between the stomach and some neighboring part of the intestine. The transverse colon is the part usually perforated; less often the small intestine.

As to the bowels, constipation is the rule. Diarrhœa is rare. The urine is usually pale and but slightly acid. Its amount is diminished, as we should expect from the slight amount of nourishment taken, and from the vomiting. Over the heart we may sometimes hear soft anæmic murmurs. The pulse is usually accelerated, although, if there be extreme marasmus, it may be slow. The temperature is normal, or even subnormal. If there is some inflammatory complication, or if the anæmia is extreme, fever may occur.

The entire duration of the disease may occupy one or two years. It is exceptional for it to last longer, except where the cancer develops in the floor of a preexisting ulcer. In this event, the symptoms of ulcer gradually give place to those of cancer. In individual cases the disease, of course, exhibits many variations and departures from the typical course. Sometimes the constitutional symptoms of weakness and emaciation are more prominent, and sometimes the distinctively gastric disorders.

The fatal termination is usually preceded by the symptoms of constantly increasing weakness. It may be hastened by complications. Now and then grave nervous symptoms appear, often quite suddenly. The patient falls into a condition resembling that of diabetic coma (*q. v.*), he is somnolent, and has a peculiar dyspnœa, with deep and labored respirations, and such an attack almost always ends fatally. Recovery from cancer of the stomach is unknown.

Diagnosis.—In addition to the ordinary symptoms common to most gastric disorders, such as pain, eructations, and vomiting, the distinctive factor in diagnosis is the discovery of a tumor connected with the stomach. The demonstration of this is almost conclusive. Other subsidiary evidence can be obtained in most instances. The patient is wasted, has a cachectic look, and is somewhat advanced in years. The most characteristic gastric symptom, as we have already said, is coffee-ground vomitus, containing blood.

It is not always easy, nor even possible, to make sure that a tumor in this region is really of gastric origin. The chief characteristics of the tumor of gastric cancer have already been mentioned. The main thing to exclude is cancer of the left lobe of the liver, or of the pancreas, omentum, or transverse colon. No general scheme for the differential diagnosis between these and the gastric disease can be given, for the circumstances and difficulties vary with almost every case. A careful consideration of all the facts, and wide personal experience at the bedside and the post-mortem table, are requisite here; and yet the most practiced diagnostician may be misled.

We may be able, however, to feel a tumor plainly, and to be sure that it is gastric, and still find it difficult, or even impossible, to determine whether it is cancerous, or whether it is not a circumscribed induration and thickening resulting from an ulcer of the stomach. This is particularly true of small tumors near the pylorus, attended with secondary dilatation. The clinical symptoms are usually valueless here, for stenosis of the pylorus must produce identically the same symptoms in either case. The age of the patient, the duration of the disease, and possibly the history of characteristic symptoms earlier in the illness, may enable us to reach a probable diagnosis. In this connection it is also to be considered that, as already mentioned, the scars of old ulcers not infrequently form the basis for the development of a cancer. The previous history of the case may present such symptoms as cardialgia and hæmatemesis, indicating that an ulcer has existed, while the objective examination, by demonstrating a tumor and the absence of free hydrochloric acid, leads us to diagnosticate the later formation of a carcinoma. In such cases, and indeed in any doubtful ones, we may test the contents of the stomach for free hydrochloric acid, and, if this is invariably absent, cancer may be inferred.

Here we should add that, even without any evidence of previous ulceration, there may be a simple non-cancerous hypertrophy of the pylorus with consequent stenosis. This occurrence we can affirm from our own experience. The tumor can not be distinguished clinically from the carcinoma of the pylorus, and not infrequently the autopsy itself will not enable us to determine at once whether there is a carcinoma or a simple ulcerative scar or hypertrophy. Absolute certainty is to be gained in such cases by a microscopic examination of the tumor, and by the discovery of metastatic growths, if there be any.

In cases of gastric carcinoma where there is no tumor to be felt, it is always very difficult to make an absolute diagnosis. In general, we should consider the possibility of a cancer in every case where a patient of advanced years complains of severe gastric symptoms without any other demonstrable cause. The more evident the constitutional symptoms of cancer, such as increasing emaciation and cachexia, the more reason is there for fearing the existence of a malignant growth. Here the investigation of the gastric contents is very important. If there is good evidence of hydrochloric acid in the gastric juice (*vide supra*), this is certainly an important argument against the assumption of a cancer. On the other hand, if there is no hydrochloric-acid reaction the suspicion of cancer will be decidedly strengthened, although by no means assured, since severe atrophic catarrh of the stomach may be associated with the complete absence of hydrochloric acid. If one finds decomposed blood in the gastric contents (coffee-ground vomiting, *vide supra*), this, as has been stated, is an almost sure sign of carcinoma.

To distinguish between ulcer and cancer of the stomach may sometimes be a difficult matter. In general, however, we shall be enabled to form a diagnosis by considering the much longer course of the disease in ulcer, as well as the usual youthfulness of the patient, the abundant presence of hydrochloric acid in the gastric juice, the characteristic vomitus, and the hæmatemesis.

Treatment.—As to treatment, we must be content if we relieve the patient's suffering. We possess no means of antagonizing the new growth. The bark of cundurango, which Friedreich recommended a few years ago, has not proved efficient. It may, however, be given with some advantage, as it seems to be a good stomachic tonic. A decoction may be made of 15 parts of the bark to 150 of water, and 10 parts of syrup of orange-peel may be added to improve the taste.

The only possibility of a cure of gastric cancer lies in the operative removal of the same, as first carried out by Billroth. Numerous attempts at operation in the last few years have many of them resulted unfortunately, but a few most favorably. We are therefore thoroughly justified, in all cases where we see any possibility of operative interference, to propose an exploratory incision. After the tumor is laid bare, we may then determine whether the case is operable or not. We can not here enter into particulars upon the subject.

The symptomatic treatment of gastric carcinoma does not differ greatly from that repeatedly described in the preceding chapters. The diet must be regulated, and the administration of hydrochloric acid is important, inasmuch as we know it to be absent from the gastric juice in this disease; otherwise we merely fulfill such indications as arise. For pain, we use the narcotics, and warm or cold applications. If there is persistent vomiting, we give small doses of opium or morphine, chloral, bits of ice, creasote, or tincture of iodine. For acid eructations, we prescribe bicarbonate of soda or magnesia. Very considerable, although unfortunately only temporary, benefit is derived from washing out the stomach. The most suitable cases for this are those of pyloric cancer with consequent dilatation. The various stomachic tonics and bitters must not be forgotten. Our chief aim must be to maintain the patient's strength as long as possible, and to do what we can, morally as well as physically, to alleviate his unhappy fate.

CHAPTER VI.

DILATATION OF THE STOMACH.

Ætiology and Pathology.—Dilatation of the stomach is, in a majority of instances, a secondary condition, the result of pyloric constriction. As we have already seen in the preceding chapters, this constriction is usually due mainly to cancer or some other new growth, or to the scars of ulcers. Narrowing produced by pressure from without is relatively infrequent. Tumors in the neighborhood may, however, thus cause dilatation; and, if Bartels be correct, a movable kidney (*q. v.*) may also compress the pylorus or the beginning of the duodenum sufficiently to produce the same result.

The manner in which the stenosis leads to dilatation is perfectly analogous with that in which stenosis of the aorta causes dilatation of the left ventricle. The propulsion of food out of the stomach into the duodenum becomes more difficult and consequently the muscular fibers of the stomach are aroused to abnormal activity, that this hindrance may be at least in part if not wholly overcome. As a physiological sequence, we find in most cases of pyloric stenosis the muscular coat hypertrophied, and particularly so in the neighborhood of the pylorus. It is not till the muscle proves inadequate to overcome the obstruction that the dilatation begins. A portion of the ingesta remains in the stomach, and the bulk of this stagnating mass gradually increases. Its weight and pressure have a direct mechanical influence in promoting the gradual expansion of the organ. In addition, the processes of decomposition usually attack the contents of the stomach, and the gases thus generated contribute largely to the mechanical dilatation. Speedily these abnormal chemical and other irritants excite catarrh. The catarrh lessens the resisting powers of the tissues, interferes with the absorption of the contents of the stomach, and in both ways tends to increase the dilatation. The united effect of all these pernicious influences may finally be to produce a dilatation up to three or four times the normal volume of the organ, the flabby fundus hanging down like a great bag into the hypogastrium.

In a smaller number of cases of dilatation we find no stenosis of the pylorus. A large dilatation of this sort is very rare. Smaller degrees of enlargement may frequently exist, but still they are so difficult to diagnosticate with certainty that we can not really say just how frequent they are. The cause of such dilatations is in many instances an impaired power of resistance, affecting chiefly the muscular layer. This condition may sometimes be due to congenital weakness of the muscular fibers, which not only makes them more yielding, but also favors the retention and stagnation of food in the stomach. In other cases the walls are weakened by disease. Thus a persistent chronic catarrh may lead to moderate dilatation; or constitutional weakness resulting from anæmia or severe illness sometimes renders the gastric muscular fibers so weak as to permit dilatation. In all such cases muscular insufficiency is the chief factor, because it favors the accumulation of ingesta within the stomach. In chronic gastric catarrh, it is probable that the muscular fibers become not merely weak but paretic, just as the muscles of the larynx frequently do in laryngeal catarrh.

One factor of gastric dilatation remains to be mentioned. It is the habitual overloading of the stomach with ingesta. That gluttons and drunkards are liable to dilatation of the stomach has long been a familiar fact. It also occurs among the indigent, who are obliged to make up for the poor quality of their nutriment (e. g., potatoes) by taking very large amounts of it. Such a condition may well be compared to cardiac dilatation from excessive arterial tension, and may be termed "overstraining of the stomach." The condition does not become strictly pathological till compensation begins to be insufficient, so that even the hypertrophied muscles are no longer equal to the task of propelling the food properly into the duodenum. In diabetes, excessive ingestion and deficient nutrition combine to produce dilatation, and it has been repeatedly observed in this disease.

Symptoms and Diagnosis.—The gastric symptoms are only in part due to the dilatation, being also due to the original lesion or to other attendant circumstances. Most patients complain of loss of appetite, frequent or constant pressure in the region of the stomach, heartburn, eructations,* and vomiting. The vomiting is frequently to a certain extent characteristic. It occurs at rather long intervals, and then a very considerable amount is vomited at once. There may be several quarts ejected. The vomitus not infrequently contains bits of food eaten several days previously. Usually vomiting affords the patient temporary relief.

We must have recourse, however, to physical examination in order to be certain about our diagnosis. In many cases, although by no means in all, inspection reveals the contours of a distended stomach through the abdominal walls. The fundus and the greater curvature are most prominent. Sometimes we can observe the peristaltic movements of the stomach, which if not present may perhaps be started up by the mechanical irritation of sudden and repeated palpation. If we administer to the patient a half-drachm each of sodic bicarbonate and tartaric acid, one after the other, as suggested by Frerichs, the consequent distention of the stomach with carbonic-dioxide gas will often render its dimensions much more evident, and we shall also be able to ascertain if the prominence already noticed is really gastric.

By palpation it is often possible to make out the greater curvature and the fundus still better than by inspection, particularly if the muscular coat happens to be contracted. A splashing sound may sometimes be evoked by giving quick, short blows with either hand alternately upon the walls of the stomach. We can hear and feel the contents of the stomach rushing to and fro with great distinctness. This is very characteristic, though not pathognomonic.

To determine the size of the stomach by percussion is so difficult, that percussion is seldom as reliable as inspection and palpation, but now and then it does aid us. The patient must be examined in both the erect and the recumbent posture, and both when the stomach is full and when it is empty. If we introduce about a quart of water into the empty stomach and then find a line of dullness below the navel which was not there before, we have good reason to believe that the organ is dilated. This is Penzoldt's test. Sometimes we are unable to define the limits of the stomach by percussion until we have dilated it with carbonic dioxide; but of course we are to bear in mind that the stomach is in this case expanded beyond its usual limits.

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* In some instances the regurgitated gases have proved inflammable.

The use of the bougie is valuable. In health the instrument will only penetrate about sixty centimetres, measured from the mouth, while in dilatation it can often be introduced as far as seventy centimetres. Occasionally we may be able to feel the end of the bougie through the lax abdominal walls, as Leube first remarked. In such a case the nearer the point is to a horizontal line joining the two anterior superior spinous processes of the ilium, the greater is the certainty that dilatation exists. Under ordinary conditions the bougie probably does not extend any lower than the level of the umbilicus, if as far as that.

A consideration of the symptoms already enumerated, combined with the above methods of physical examination, will in many cases enable us to diagnosticate with certainty any considerable dilatation of the stomach; and yet it must be confessed that sometimes quite extensive dilatation may escape observation. In such instances there may be either a partial or complete absence of symptoms suggestive of any serious gastric derangement, so that no careful examination of the organ is made, or the methods of physical examination already mentioned, when employed, yield a negative or ambiguous result. It is true that other methods have been suggested by various authorities to determine the size of the stomach and test the functional capacity of its muscular tissue; but none of these have as yet been generally adopted. Thus Schreiber introduced an India-rubber bag on the end of a catheter, and tried by blowing it up to gain information as to the size and position of the organ. With the same end in view, Rosenbach practiced auscultation of the râles caused by blowing into a catheter, the opening of which was placed at the level of the fluid contained in the stomach.

Other symptoms are mostly analogous to those seen in other severe gastric disorders. The general nutrition becomes gradually so impaired, particularly if there is much vomiting, that the patient may seem like a skeleton. Kussmaul has sometimes observed painful cramps in the flexors of the arms, the calves, and the muscles of the abdomen. These he refers to the abnormal dryness of the muscular tissue. The bowels are almost always very much constipated, mainly as a result of the diminished amount of ingesta which reaches the intestine. The urine is small in amount, and often neutral or alkaline in reaction. The alkaline reaction Quincke believes is more apt to be present while the stomach is being washed out, because in this way the system is deprived of a relatively large amount of acid.

Course of the Disease and Prognosis.—The course and duration of the disease are chiefly dependent upon the nature of the original lesion from which the dilatation proceeds. If there is cancerous stenosis of the pylorus, of course the case is hopeless. Cicatricial stenosis with secondary dilatation allows a more favorable prognosis. With proper treatment and a sensible mode of life, the patient may be tolerably comfortable for years; but finally nutrition becomes more and more impaired, and death ensues.

The general course of the disease presents all sorts of vicissitudes. As long as the hypertrophied muscles are able to overcome the abnormal obstruction, there may be no symptoms of importance. Just as in cardiac cases, it is only when compensation becomes disturbed that the results of dilatation are observable. If we can tone up the muscles again, or reduce the work they are called upon to perform to an amount which they are capable of accomplishing, marked relief follows.

Those cases of dilatation which are not due to pyloric stricture have, on the whole, the best prognosis. In milder cases of this kind there may be permanent recovery, provided proper mechanical and dietetic treatment be promptly and persistently employed.

Treatment.—Our first aim in treatment should be to relieve the dilated organ of the great amount of material it contains and to avoid new accumulations. If this double effort is successful, we have removed the injurious influences, both mechanical and chemical, which we have seen to maintain and persistently to aggravate the dilatation.

The indication is best met by the "mechanical treatment of dilatation," for which we are so deeply indebted to Kussmaul. This method is beneficial to the chronic catarrh which accompanies the dilatation, or which may even have produced it. We have no direct means of treating stenosis of the pylorus, if it exists, unless possibly by surgical operation. The operative removal of cicatricial stenosis has already been repeatedly performed with success, and it is to be expected that it will come to be practiced more and more.

For emptying and rinsing out the stomach the best means is simple siphonage. A long, soft Nélaton's catheter, supplied with sufficiently large openings on its sides, near its extremity, is joined to a rubber tube of about one metre in length, and to the end of this is fitted a large glass funnel (*vide* Fig. 36). The stomach-tube being introduced and the funnel raised, the entire tube is filled with water and thereupon lowered. Thus siphonage is produced and the contents of the stomach flows out. By alternate raising and lowering of the funnel, combined, of course, with the pouring in of water or soda solution, and the escape of the same again, the stomach may be thoroughly cleansed. It is still more convenient to have a Y-tube of glass fitted to the upper end of the catheter connecting respectively with a reservoir of water and with a slop-jar by rubber tubes, the alternate opening and closing of which will permit water to run into the stomach from the reservoir and then out again into the jar.

The siphon apparatus and the necessary manipulations are so simple that we have had several patients with gastric dilatation who daily washed out their own stomachs. This simple tube has almost completely superseded the regulation stomach-pump. The latter consists of a catheter attached to a syringe which has two openings at its end, one to aspirate and one to allow the escape of liquid. More than once, when this instrument has been employed, a piece of the lining membrane of the stomach has been sucked into the hole at the end of the stomach-tube and torn off. The assumption that the pump empties the stomach more completely than does the siphon is seldom borne out by facts.

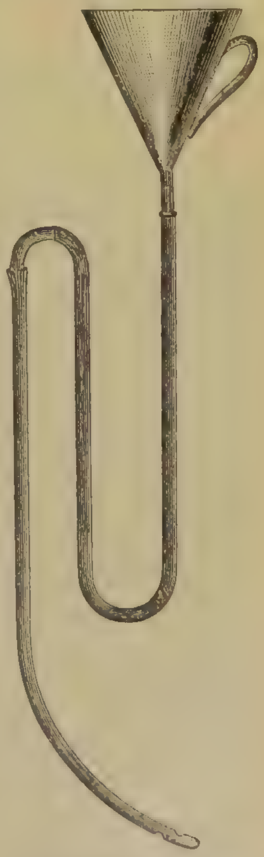

FIG. 36.—Stomach tube, with Hegar's funnel.

We may use pure water for lavage, or, as already mentioned, a one- or two-per-cent. solution of soda or of Carlsbad salts, which may more effectually clear away the mucus. If there is reason to believe that fermentation is particularly active, we may employ a one-per-cent. solution of salicylic acid or a two-per-cent. solution of resorcin. The best time for washing out the stomach is shortly before meals. It is done once or twice a day.

The patient's diet should be carefully regulated. The food should be nourishing, easily digestible, and of the least possible bulk. We should therefore try Leube's meat solution, fine shavings of raw beef or ham, soft-boiled eggs, and milk in small amounts, but should avoid vegetables, coarse bread, and the like. In this way we can usually succeed in improving the patient's nutrition decidedly, and the vomiting and sense of gastric oppression as well as other disagreeable symptoms will cease. Whether the improvement will be permanent depends, as has been said, upon the nature of the dilatation and the causal lesion.

In conclusion, we should mention that the attempt has been made to tone up the muscular fibers of the stomach and stimulate them to more vigorous contraction. Energetic faradization and galvanization of the stomach have been recommended. The application may be made through the abdominal walls, or by means of an electrode introduced through a stomach-tube. Nux vomica has also been employed to meet the same indication. The symptomatic treatment of gastric dilatation may employ also all the other remedies mentioned in discussing catarrh of the stomach. It is advantageous to have the patient wear an elastic bandage firmly applied around the abdomen.

CHAPTER VII.

NERVOUS DISORDERS OF THE STOMACH.

(*Nervous Dyspepsia. Gastric Neurasthenia.*)

It very often happens that persons complain of gastric symptoms for which we can assign no objective lesion of the stomach. There is neither any certain indication of any important pathological change, such as dilatation, cancer, or ulcer, nor any history to be obtained of any of the causes of chronic gastric catarrh. On closer study of the cases we find, however, that the stomach symptoms are most strikingly dependent on certain nervous and chiefly psychical conditions. We are therefore justified in describing such affections as nervous diseases of the stomach, and entirely distinct from other affections of that organ.

If we seek to give a more accurate account of the precise nature of these nervous disorders, we must admit, in the first place, that there may be in some cases actual disease of the nerves of the stomach. Thus, for example, cases of pure "nervous cardialgia" occur, the foundation of which is probably a neuralgia of the nerves of the stomach, precisely corresponding to the neuralgias of other sensory nerves. Again, motor derangements are seen which are evidently caused by abnormal excitement of the motor nerves of the stomach, or else of the corresponding nerve-centres. To this class belongs Kussmaul's "peristaltic restlessness of the stomach," characterized by a marked exaggeration of peristalsis, which is both seen and felt through the abdominal walls, is sometimes accompanied by all sorts of gurgling noises, and of which the patient himself is often painfully conscious. Peculiar attacks of nervous vomiting belong in the same category. In many persons they occur from time to time without special cause and with great violence. They may last for hours, or even days, and are often associated with cardialgia, general prostration, and other nervous phenomena. Leyden observed such cases, and described them under the name of "periodical vomiting with paroxysms of gastralgia." The cause of this state is far from clear. Except at the time of the attack there are no symptoms whatever of gastric disease or any other, and we are therefore obliged to assume a "nervous origin" for it. The paroxysm reminds one decidedly of the gastric crisis of tabes dorsalis (*vide* p. 643). It may also be related to migraine (*q. v.*).

If in these cases the vomitus is abnormally sour, we have a condition which Rossbach has named nervous gastroxynsis. Gastroxynsis seems to be associated, therefore, with secretory disturbances, and recalls the above-described acid dyspepsia (*vide* p. 382). There is also a form of persistent, purely nervous vomiting. We have seen it with particular frequency in school children of both sexes, in whom about the only symptom was that they would vomit after every meal.

The varieties already enumerated are not, however, nearly so common as others in which the nervous condition is not so pronounced. The patient complains almost all the time of a train of nervous and dyspeptic sensations, all of which occur mainly after eating, during digestion, and which may therefore be properly classed as nervous dyspepsia. Such a patient will say that he has a sensation of pressure and pain in the stomach after every meal, and that after a few mouthfuls the organ feels full and distended. As a consequence, there are often thoracic oppression and palpitation. The patient frequently suffers also from eructations, which may increase to actual vomiting. All these symptoms may occur in the same way where there is actual gastric disease, particularly gastric catarrh, but in the present instance they are usually associated with other phenomena which indicate their nervous character. In the first place, we may notice that there is often a striking changeableness in their severity. The same patient who to-day complains of severe gastric oppression after a few spoonfuls of soup, will to-morrow, when happy and excited, enjoy a grand dinner without being in the slightest degree reminded of his stomach. This and many other circumstances show most plainly the influence of emotion upon the state of the stomach. We all know how appetite may at once vanish under the influence of violent anger or keen anxiety or lively hope, or any strong emotion, whether pleasurable or the reverse, and that great excitement not infrequently causes vomiting or the like. In the same way, sensitive persons are affected by the slightest psychical influences. And of all such, the most harmful is disquietude with regard to their own bodily condition. The fear that a dish they have eaten may harm them, the constant dread that a serious disease of the stomach is in process of development —such mental disquietude is it which is best calculated to maintain the unhealthy state and gradually to aggravate it. In this way a peculiar subjective hyperæsthesia is developed which feels exquisite "pain" in the stomach when there is really nothing more than the ordinary normal sensation. And, in conclusion, there are developed in the same way certain half-unconscious, half-voluntary movements which produce eructations, vomiting, and the like.

What we desire, therefore, especially to emphasize, is our own conviction that in the large majority of the cases of so-called nervous dyspepsia there is no functional derangement of the nerves of the stomach themselves, but a diseased "psychogenous" excitation of the nerve-centres, the consequences of which are expressed mainly in the domain of the digestive functions. Nervous dyspepsia is only a particular example of that great class of nervous diseases which owe their origin to hypochondriacal conditions of the mind, and which may occur in the most diverse organs of the body. This explains why the gastric symptoms are frequently attended by other nervous phenomena, among which may be mentioned symptoms of increased psychical irritability, headache, pressure in the head, vertigo, and abnormal sensations in the extremities of pain or cold or numbness. There are almost always also some intestinal symptoms. The patient complains of distention of the abdomen, and particularly of irregular and constipated bowels.

Nervous dyspepsia may become a rather serious matter, if the patient's anxiety to avoid errors in diet, and the anorexia occasioned by his uneasy sensations, prevent his taking sufficient nourishment. In this way there may be gradually produced a considerable or even excessive emaciation, with corresponding debility and weakness. Such a patient may become completely bedridden, and give one at first the impression of severe illness.

In such cases the diagnosis is not always easy, although it can usually be formed upon careful consideration of the whole course of the disease and the character of the patient. Physical examination in nervous dyspepsia is, of course, invariably negative in its results. The epigastrium is often very tender on pressure, and the vertebræ painful. The skin over the stomach is often noticeably insensible to pin-pricks. This fact is a connecting link between nervous dyspepsia and the allied hysterical affections of the stomach. If it is possible to examine the contents of the stomach with the aid of the stomach-tube, the processes of digestion and the secretion of gastric juice are found to be undisturbed. This discovery often has, of course, great diagnostic value, and it holds true at least for most of the cases which rest upon a purely psychical, hypochondriacal basis. If, on the other hand, the patient has not only well-marked nervous symptoms, but also a diminution of the motor power of the stomach, and diminished or (as is not infrequent) increased secretion of acid in the stomach, the diagnosis is usually obscure. These changes may, indeed, depend on purely nervous causes; but still in the individual case it is generally difficult or even impossible to exclude with certainty the simultaneous existence of an actual catarrh or ulcer of the stomach.

In many severe cases it requires long-continued observation of the patient to render a correct diagnosis possible. We may be aided by learning what remedies help and what hurt the patient. This is particularly true where the diagnosis lies between ulcer and nervous dyspepsia, where it may be very difficult, and often, despite careful balancing of all the symptoms, scarcely possible to reach a sure conclusion. A patient with nervous dyspepsia subjected to the strict regimen suitable for ulcer often grows worse and worse, while an opposite course of treatment (*vide infra*) may achieve the most surprising success.

The prognosis depends mainly on the outward circumstances of the patient. If the harmful mental influences or other ætiological factors are persistently active, actual and permanent recovery is hardly to be expected; but if the cause can be removed, complete recovery is not rare, even in what seem to be grave cases. A liability to relapses is, of course, almost always left behind.

Treatment.—If nervous dyspepsia has once been diagnosticated, the proper aim of our therapeutic efforts becomes perfectly definite. We must, in the first place, convince the patient that he has no incurable or even dangerous gastric disease, but that, on the contrary, his stomach is perfectly capable of performing its functions in a normal manner. Nothing could be more harmful to a sufferer from nervous dyspepsia than to have his physician manifest great anxiety about treatment, and prescribe a very strict diet. The patient must rather be gradually led to use an abundance of nutritious food. It is in this way alone that he regains a confidence in himself, when he sees that the hearty food does him no harm, that he is gaining flesh, and that the bowels are becoming regular.

Internal remedies are best omitted altogether if the patient has already taken a good deal of medicine. We should particularly warn him against the abuse of purgatives. If we must prescribe something, a bitter tonic is very good, such as tincture of nux vomica. Anæmic patients may also take iron. For nervous vomiting and regurgitation the best remedy is bromide of potash. Antipyrin also is sometimes useful.

Those methods of treatment are of great value which are directed to the toning up of the body in general and the nervous system in particular. The patient may go into the country or to the mountains or the seashore. Methodical treatment with cold water is good; in particular, sponging, combined with rubbing of the trunk and the whole body, almost always gives good results. On the other hand, those health resorts which often do good in the case of organic gastric disease are but seldom beneficial in nervous dyspepsia. Thus, we have frequently seen such patients, who had been sent by their physicians to Carlsbad, return worse rather than better. We have repeatedly found electricity valuable, although we are not prepared to deny that its subjective effect may be of chief importance. Galvanism is applied along the spinal column, and also through the stomach horizontally, one large electrode being placed upon the epigastrium and the other on the back. It is well to reverse the current frequently. Faradization of the abdominal walls is indicated, especially when there is constipation.

We need hardly add that the ætiological factors must not be overlooked. The patient must be warned against useless overexertion, emotional excitement, etc. (Compare the chapter on neurasthenia.)

The main point is to aim at a methodical moral training of the patient. He should learn to feel and to behave like a healthy person. He should regain his self-control, and not allow himself to be upset by every slight psychical shock, whether subjective or coming from without. It is self-evident that this goal is to be attained not by baths and prescriptions, but by correct moral guidance. It is therefore true of nervous dyspepsia, as of all similar neurasthenic conditions, that it is to be cured not by physic but by the physician.

SECTION V.

DISEASES OF THE INTESTINES.

CHAPTER I.

INTESTINAL CATARRH.

(*Catarrhal Enteritis.*)

Ætiology.—The majority of cases of intestinal catarrh, like gastric catarrh, are due to an abnormal irritation of the mucous membrane of the intestine by its contents. In most cases the irritants are of a mechanical or a chemical nature, and depend upon the quantity and quality of the food taken, which explains why catarrh of the stomach and of the intestine are so often combined with each other. Noxious substances, taken into the system by the ingestion of spoiled food, like spoiled meat, fish, beer, etc., very often play a part in the origin of intestinal catarrh.

To the intestinal catarrhs caused by improper food we may add the toxic catarrhs which are produced by the direct ingestion of poisonous substances into the digestive tract. Severe inflammations of the intestinal mucous membrane arise from poisoning by mineral acids and corrosive alkalies, arsenic, corrosive sublimate, etc. Intestinal catarrh may also arise from the imprudent use of certain drugs, especially active cathartics.

A great many cases of intestinal catarrh are due to infectious influences, including most of the apparently spontaneous catarrhs, and also many, if not all, of the catarrhs attributed to taking cold or getting wet, and, finally, those affections which often develop epidemically or endemically in hot weather, and which we term summer complaint, cholera morbus, etc. Cholera morbus is an especially severe form, and will be described more fully later on. We must also mention here that intestinal catarrh is very often one symptom of other general infectious diseases, like typhoid, dysentery, septic diseases, or severe malaria.

In a final class of cases intestinal catarrh develops from disturbances of the circulation, which cause a passive hyperæmia of the intestinal mucous membrane. Diseases of the liver and portal vein, and also chronic diseases of the heart and lungs, are the chief affections which produce a stasis in the portal system, and thus an intestinal catarrh; but here the stasis is probably, in most cases, only a predisposing factor in the development of the catarrh, since the action of all other irritants is made easier by the disturbance of the circulation.

The great frequency of intestinal catarrh in both sexes, and at every age, is well known. Children, above all, have a pronounced tendency to diseases of the intestine, so that, by a probable estimate, almost one third of the illnesses of children are to be referred to the intestinal canal. We will give a special description of intestinal catarrh in children on account of this fact.

Pathological Anatomy.—The pathological changes in catarrhal inflammation of the intestines are essentially the same as are met with in the inflammation of any other mucous membrane. Redness and swelling of the mucous coat, increased secretion of mucus, and in severe cases purulent products on the surface of the membrane, and a cellular infiltration of the tissue itself, are the well-known processes characteristic of all catarrhal inflammations. The solitary and agminated follicles often swell in follicular catarrh, and may finally become the seat of superficial follicular ulcers. We often find superficial erosions on the rest of the mucous membrane, and in severe cases the so-called catarrhal ulcers.

If the catarrh has lasted a long time, we sometimes find quite a considerable thickening of the mucous membrane, which is due to a hyperplasia of the connective tissue, and which gives an uneven, puffy appearance to the internal surface of the intestine. Circumscribed hyperplasia of the connective tissue may actually lead to the formation of polypi. If the orifices of Lieberkühn's follicles are stopped, we have a cystic degeneration of the follicles from the retention of the intestinal juice.

We very often find, however, a considerable atrophy of the mucous membrane, especially in the chronic intestinal catarrh of children. This atrophy, which has lately been carefully investigated, especially by Nothnagel, affects chiefly the glandular layer of the mucous coat. In place of the glands, which in many parts may wholly disappear, we find connective tissue more or less rich in cells. The atrophy is usually most pronounced in the colon and the lower part of the ileum. The muscular coat may also take part in the atrophy.

Certain peculiarities of catarrh affecting single portions of the intestine will be mentioned later on.

Symptomatology.—The symptom by which we chiefly determine an affection of the intestinal canal, and which in the milder cases is often almost the only sign of an intestinal catarrh, is diarrhœa—that is, abnormally frequent stools of a looser consistency than usual; yet, strictly speaking, we should not attribute every diarrhœa to a catarrh of the intestinal mucous membrane, since a large number of influences may directly produce an increased peristalsis and a consequent diarrhœa. Thus, for instance, it is a well-known fact that sudden terror or great anxiety may sometimes cause an obstinate diarrhœa in a very short time. In general nervous and neurasthenic conditions, we sometimes have a chronic diarrhœa which can be due only to abnormal processes of innervation—" nervous diarrhœa." The diarrhœa which may arise immediately after taking cold is also merely the result of abnormally great peristaltic movements excited in a reflex manner. Probably a number of chemical and infectious irritants may also stimulate the movements of the intestines, and thus set up a diarrhœa, without causing at the same time a catarrh of the mucous membrane. Practically, however, we can not make a sharp distinction between diarrhœa and intestinal catarrh; and, in most of the diarrhœas which have lasted for some time, we are certainly right in supposing that there are actual anatomical lesions of the intestine, as well as functional disturbances.

There are two chief factors which cause diarrhœa in intestinal catarrh. In the first place, as has already been intimated, the same injurious substances which cause the catarrh also excite peristalsis. The many products of the abnormal processes of decomposition in the intestine also exert a like influence. Besides the abnormal irritants, however, we ought also to consider an abnormally great irritability of the intestinal walls in catarrh. Thus it happens that the fluid contents of the intestine are expelled by the vigorous peristaltic movements (which the patient himself often feels as a "rumbling in the abdomen"), before the normal consolidation of the fæces is completed by the absorption of water. The food, under normal conditions, passes through the small intestine in two or three hours, and thus the consolidation of the fæces takes place, as is well known, almost exclusively in the colon. We see, therefore, why the diarrhœa owes its origin chiefly to the increased peristalsis of the large intestine; although in many cases the peristaltic action of the small intestine is also increased.

Besides increased peristalsis, another circumstance may perhaps contribute to the diarrhœa, viz., the greater fluidity of the contents of the intestine due to the increased secretion of mucus and the exudation caused by the catarrhal inflammation.

In the intestinal catarrh from passive congestion we must consider still another factor, to explain the thin and watery stools—namely, the diminished absorption of water by the intestine from disturbance of the circulation. In other catarrhs this factor is quite subordinate to increased peristalsis.

The diarrhœal dejections show a considerable difference in regard to their minor characteristics. Their number varies very much. There are sometimes two or three, and sometimes ten or more, evacuations in the twenty-four hours. The consistency of the stools is pap-like, or almost wholly watery. This is due to the abnormal amount of water in them, amounting to ninety or ninety-five per cent., while the amount in normal stools is about seventy-five per cent. The color of the thin stools in intestinal catarrh is usually bright yellow, but they are sometimes greenish from the admixture of bile pigment, and sometimes slimy (*vide infra*).

In only a part of the cases does microscopic examination give us information as to the extent and intensity of the catarrh. We usually find the remains of the food, muscular fibers, starch-granules, and fat, and also countless bacteria, and often triple phosphates, occasional pus-corpuscles, and cylindrical epithelium—chiefly the constituents which are found in normal stools. Further peculiarities will be mentioned below.

Besides the diarrhœa, there is often, but by no means always, abdominal pain in intestinal catarrh, either continuous, or having the character of the paroxysmal, so-called colicky pains. In catarrh of the rectum there is that constant painful desire to go to stool which we term tenesmus.

Physical examination of the abdomen gives, on the whole, few important results. Sometimes the abdomen is flat, and sometimes there is meteorism. Marked peristaltic action of the intestines often causes gurgling and rumbling noises—*borborygmi*. On palpation, the abdomen is often somewhat sensitive. The peculiar colicky pains, however, are, as a rule, alleviated by external pressure. In rare cases we may detect a fluctuation on palpation, if the intestine contains much fluid. The results of percussion depend largely upon the fullness of the intestines.

In many cases of simple diarrhœa the general health is practically unaffected, but in other cases of acute intestinal catarrh, especially in the severe infectious forms, the disturbance of the general health may be quite considerable. The patient feels so dull and weak that he stays in bed. We often see a moderate rise of temperature, between 100° and 102° (38°-39° C.). There are very often gastric symptoms also, especially loss of appetite, and vomiting. Other organs are quite rarely affected, except in duodenal catarrh, when the liver is involved (*vide infra*). In acute infectious intestinal catarrhs, there is sometimes an eruption of herpes on the lips. We have repeatedly seen, in severe cases of acute enteritis, marked muscular and articular pains, and even slight but manifest swelling of the joints.

Different Forms of Intestinal Catarrh.—Since the intestine is an organ which is only slightly accessible to physical examination during life, and since we can only rarely make a post-mortem examination in the mild diseases of the intestine, our knowledge as to the different forms of enteritis is defective in many respects. In practice we content ourselves in most cases with diagnosticating an intestinal catarrh simply from the existence of diarrhœa, without laying much stress upon the special variety; but in many cases some points can be obtained which give information as to the more accurate seat of the catarrh. The distinction between acute and chronic intestinal catarrh is also of practical significance.

Duodenal catarrh can be diagnosticated only if it is combined with jaundice. The details regarding it may be found in the chapter on catarrhal jaundice.

Isolated catarrh of the small intestines, of the jejunum and ileum, is probably only of rare occurrence, except when the upper portions of the colon are involved. We can very rarely diagnosticate it with certainty, but there are a number of factors which permit us to decide that the small intestine is chiefly affected, or at least that it is involved in the disease. In the first place, we may assume an affection of the small intestine, from obvious reasons, in all those cases in which there are also gastric disturbances. It is evident that, in the frequent combination of gastric and intestinal catarrh, the portions of the intestine nearest the stomach will be chiefly affected. Physical examination of the abdomen also gives some indications, since the slight sensitiveness and swelling of the abdomen, as well as the visible abnormal peristaltic action, chiefly affect the middle and lower portions of the abdomen in catarrh of the small intestines, while the analogous symptoms in catarrh of the large intestine affect the lateral and upper portions of the abdomen, corresponding to the anatomical course of the colon. We can not make a sharp distinction, however, in this respect. The results which auscultation and percussion over the abdomen give in regard to the point of origin of the gurgling sounds and the fullness of the loops of intestine are very rarely unequivocal, and hence are of little value in diagnosis.

Careful examination of the stools gives us more information. As has already been said, we need have no diarrhœa in a catarrh confined to the small intestines, since diarrhœa is due only to the increased peristalsis of the large intestine; hence diarrhœa is absent, for example, in most cases of duodenal catarrh (catarrhal jaundice). In extensive isolated catarrh of the small intestines the firm stools passed may, however, be regarded as pathological, because, on microscopic examination, they appear intimately mixed with little lumps of hyaline mucus (Nothnagel). As a rule, of course, catarrh of the small intestines is combined with a catarrh of the upper portion of the large intestine. Then we have a diarrhœa, but the thin stools show some peculiarities which point to an implication of the small intestines. As a result of the increased peristalsis of the small intestines, we find certain constituents in the stools which are normally contained in the small intestines, but which under normal conditions are no longer to be met with in the fæces in the large intestine. We find here, in the first place, undigested constituents of the food, large masses of muscular fiber, or even fragments of meat which may be recognized by the naked eye, and also starch and fat. Of course the opposite hypothesis does not hold good, that, if we find a large amount of the undigested portions of the food in the stools, it must necessarily always point to a catarrh of the small intestines, since the digestion may be impaired by other circumstances, like fever or anæmia, and increased peristalsis of the intestines, from any cause, must result in the same symptoms. A diarrhœa, where the thin stools contain a very large amount of undigested particles of food which can be recognized by the naked eye, was formerly called lientery, and the term is still occasionally used.

If the stools contain bile in addition to some portions of the food, it is to a certain degree characteristic of catarrh of the small intestines. Under normal conditions the contents of the small intestines alone show Gmelin's test for bile-pigment, while the contents of the large intestine, and also the normal stools, do not. In intestinal catarrh, with increased peristalsis of the small and large intestines, there is, however, often quite a large admixture of still undecomposed bile-pigment. The green stools which are so often seen in the diarrhœa of children, and more rarely in that of adults, are also well known. Such stools usually show a marked color reaction with nitric acid. In other cases we find only certain constituents of the stools stained with bile—a fact to which Nothnagel has called special attention. Yellow pigmented bits of mucus and cylindrical epithelium, and round cells stained with bile, are especially characteristic of the diarrhœa of catarrh of the small intestines.

Catarrh of the large intestine is probably present in every diarrhœa, as has been repeatedly stated, inasmuch as the thin stools can be explained only by an increased peristalsis of the large intestine; but in a number of cases we have symptoms which point especially to a disease of the large intestine, particularly of its lower portion.

Physical examination of the abdomen should show changes, like swelling, sensitiveness to pressure, etc., chiefly in the lateral portions, corresponding to the course of the colon; but this is rather a theoretical hypothesis than a sign of practical value. We can not definitely affirm, either, that "colicky pains" are characteristic of catarrh of the large intestine alone. The condition of the stools, however, is of importance. In the first place, we may note that, if the stools contain many masses of mucus which may be recognized by the naked eye, it is of diagnostic significance. As we have seen above, the stools in catarrh of the small intestines also contain mucus, but it is intimately mixed with the other constituents of the fæces, and hence it can usually be recognized only by the microscope. In catarrh of the large intestine, however, the mucus rather adheres to the outside of the other constituents, and is often present in large masses visible to the naked eye. If the catarrh affects the lower part of the large intestine chiefly, it may be that the intestinal contents are already formed into firm lumps, which may sometimes be wholly or partly inclosed in a layer of mucus. In acute catarrh of the lowest part of the large intestine the evacuations are sometimes composed chiefly of pure mucus, with a greater or less admixture of pus, as is seen chiefly in the "catarrhal flux" (see the chapter on dysentery). The more the rectum is involved in the inflammation, the worse is that painful feeling of tension and pressure at the anus during and after the evacuation, which we term tenesmus.

Isolated inflammation of the rectum (proctitis) is, at least in part, directly accessible to examination by the finger or by the speculum. Painful tenesmus and an admixture of mucus, and especially of pus in the stools, are the chief symptoms of the disease. In most cases, however, we have to do, not with a primary disease, but with a secondary catarrh of the rectal mucous membrane, as a result of different morbid conditions in the vicinity of the rectum, or of new growths, syphilitic processes, etc., in the rectum itself. Periproctitis (ischio-rectal abscess) belongs to the domain of surgery, and can not be described here.

Intestinal catarrh is divided into an acute and a chronic form.

In the acute intestinal catarrhs, excluding the toxic inflammations, we class simple diarrhœa, which usually passes off in a few days, and the severe enteritis, which is probably usually infectious, and which is attended by a marked disturbance of the general health, by fever, and sometimes by gastric symptoms also, as well as by herpes, by occasional slight albuminuria, by articular pains, etc. It lasts from three to ten days. Cholera morbus (*vide infra*) is to be regarded as a special form of acute infectious inflammation of the gastric and intestinal mucous membranes.

Chronic intestinal catarrh either comes from an acute disease of the intestinal mucous membrane, or it gradually develops independently. In adults it is by no means a frequent disease, at least as regards pronounced cases, and is much rarer, for example, than chronic gastric catarrh; but we have already mentioned that it is very common in practice among children.

[Chronic intestinal catarrh is much more common in the southern than in the northern portions of our country. It was the great scourge of camps during our late civil war, and there are many men alive to-day, pensioned and unpensioned, who have never recovered from the diarrhœa contracted during their army life.]

In regard to the ætiology and symptomatology, much the same may be said of chronic catarrh which we have learned to recognize in considering acute catarrh. In regard to ætiology we must note, in adults, chiefly the intestinal affections remaining after an attack of an acute disease, like dysentery, severe malaria, or typhoid. Among the most prominent symptoms are the abnormal evacuations, usually alternating between diarrhœa and constipation, but sometimes showing persistent constipation, due chiefly to the atrophy of the muscular coat and the disturbance of the nervous apparatus. We must also mention, as a prominent symptom, the secondary disturbances of the general nutrition, like emaciation and anæmia. In regard to peculiarities in the character of the stools, we must refer to what has been said above. As chronic catarrh of the large intestine is far commoner than chronic catarrh of the small intestines, we very often find large amounts of mucus in the stools. Special mention must here be made of a particular form of chronic catarrh of the large intestine, in which large pieces of membrane and complete mucous casts of the intestinal canal are evacuated.

This peculiar condition, of which we have seen several cases, affects women most frequently, but it is also met with in men. The disease is almost always associated with obstinate constipation. Either at the same time with hard scybala, or quite independently, large masses of this membrane are from time to time evacuated, and the passage of them is often associated with quite severe colic. These masses, as the microscope shows, consist of mucus, and often contain also much cylindrical epithelium, and more rarely a few round cells and occasional crystals of cholesterine and triple phosphate. The general nutrition sometimes suffers but little, but in other cases quite a good deal. We very often see in women, at the same time, all sorts of hysterical and nervous symptoms. The disease, which is termed membranous enteritis, desquamative catarrh of the large intestine, or mucous colic (*colica mucosa*), may last for years. No thorough examination has yet been made into the ætiology and pathology of the disease, but it is most probable that the intestinal mucus collects into the membranous formations above described in the bottom of the longitudinal folds of the spasmodically contracted large intestine (Marchand).

Treatment.—Most of the milder cases of acute intestinal catarrh need only a dietetic treatment. If the patient avoids all injurious substances for a few days, he recovers completely. The different gruels, like barley and oatmeal gruel, and also weak broths and thoroughly toasted bread, or the German *zwieback*, are generally regarded as the most suitable food. The coarser vegetables and fruits, fat meat and brown bread, are to be avoided as much as possible. In other respects we may refer to the dietetic rules laid down under the treatment of chronic gastric catarrh.

It is also an important rule, confirmed by much experience, to keep the abdomen warm. Children should always stay in bed, and adults should do so, at least in all severe cases. It is a good plan, particularly in children, to protect the abdomen from cold by a flannel band.

In many of the mild cases it is scarcely necessary to use internal remedies. Gum mixture (P. G.) or almond mixture is a good prescription if there is no special indication, but in severe cases further medication may be proper. If we have reason to suspect some irritating ingesta or a collection of fæces as a cause of the intestinal catarrh, a cathartic acts favorably at the beginning of the treatment, in spite of the existence of diarrhœa. Our best cathartic in such cases is castor-oil or calomel. In all those cases where many thin dejections point to a greatly increased peristalsis of the intestine, we use astringents, especially opium, which we give in the form of the simple tincture or the wine in doses of ten or fifteen drops, one to three times a day; or as a powder, half a grain to a grain (grm. 0·03 to 0·05) of opium with a grain (grm. 0·05) of sugar, two or three times a day. It is also recommended to combine the opium with some mucilaginous vehicle, as 2 parts of laudanum to 150 of gum mixture or decoction of salep (P. G.), a teaspoonful every two or three hours.

Besides opium, the different astringents are used in the treatment of intestinal catarrh, especially tannic acid, acetate of lead, logwood, columbo, catechu, and many others. These remedies are often given combined with opium, as in the following prescriptions:

℞ Opii .... gr. ss. (grm. 0·03);
Acidi tannici .... gr. j. (" 0·05);
Sacchari albi .... gr. j. (" 0·05). M.
S. As a powder two or three times a day.
℞ Decoct. radicis calumbæ .... (1–15) ℥ v. (grm. 150·0);
Extract. opii .... gr. j. (" 0·05);
Syrup. aurantii .... ℥ ss. (" 15·0). M.
S. Tablespoonful every two hours.

Good results, particularly in chronic cases, are sometimes obtained from the administration of bismuth. Either the subnitrate or the salicylate may be given in powders containing seven or eight grains (0·4–0·5).

If there is severe colic, opium, or, under some circumstances, an injection of morphine is the best remedy. In milder cases it is sufficient to apply warmth to the abdomen, by warm poultices or hot towels. The colic, however, often depends upon the presence of old fæcal masses in the intestine, when it is necessary to prescribe a cathartic, like castor-oil.

In all cases where the symptoms point to a more intense disease of the large intestine, local treatment may be employed. This is chiefly of importance in the treatment of chronic intestinal catarrh which is situated mainly in the large intestine. We irrigate the large intestine daily with weak astringents, and sometimes with disinfectants. The necessary apparatus is very simple. It consists of an ordinary irrigator, to which a rubber tube, about half a metre long and with a proper tip, is attached. Instead of the irrigator we can use an ordinary glass funnel, a "Hegar's funnel." We may very well use, for an end-piece to be introduced into the rectum, a long, soft, elastic œsophageal tube, which can easily be pushed quite high up. The fluids used for irrigation must always be warmed to about 85° (30° C.), and should be allowed to run in gradually and slowly. The amount of fluid used for one irrigation should be two or three pints (1–1½ litre), or sometimes more. The patient keeps on his back during the irrigation. The knee-elbow position, which is much more uncomfortable than the dorsal, is only occasionally necessary. The fluids most used are a one- or two-per-cent. solution of salicylic acid, solutions of salicylic and boracic acids combined, a one-per-cent. tannin solution, or a solution of acetate of lead (1 to 1,000).

If there is painful tenesmus, it is usually relieved by suppositories of cocoa-butter and extract of opium.

In chronic intestinal catarrh a careful regulation of the diet is of the greatest importance. Besides local treatment we must consider internal remedies, chiefly the astringents mentioned above, to which we may add alum, guarana, and kino. Furthermore, the preparations of strychnine, and, among newer remedies, salicylate of bismuth and naphthaline, in powders of two to five grains (0·10–0·30), deserve mention. Baths, like Carlsbad, Kissingen, Marienbad, or Tarasp, often give good results, especially in cases where there is at times constipation.

[Saratoga, Bedford (Pa.), and the Virginia Springs are those in chief repute in this country. The change of scene and the regular life, to which patients will more readily submit in these and similar places than at home, have probably more effect than the water itself.

I have known the Rockbridge alum-water, internally, render good service. A sea voyage is sometimes curative, and, in general, a sufferer from chronic diarrhœa should shun malarial regions.

Treatment must often be patient and prolonged; relapses are liable to follow imprudence of any kind. Time is, of course, required for the restoration of the intestine to its normal structure and function, and perseverance is not infrequently rewarded in full measure. A decided change of climate will sometimes be found to render service greater than that of diet and drugs.]

The symptoms which simulate a chronic intestinal catarrh often depend, as we have said, on abnormal processes of innervation. These are the cases which are associated with general nervous and nervous-dyspeptic symptoms. Internal remedies in such cases are of but little assistance, but judicious general treatment, cold-water cures, electricity, and massage may be attended with very good results. (See the chapters on nervous dyspepsia and habitual constipation.)

CHAPTER II.

CHOLERA MORBUS.

(*Cholera Nostras. Cholera Infantum.*)

By the name "cholera morbus" we mean an acute disease of the stomach and intestinal canal, of a definite form, whose symptoms in severe cases greatly resemble those of genuine Asiatic cholera. It is in the highest degree probable, from the whole course of the disease, that cholera morbus also depends upon an acute infection of the body by a specific, organized producer of disease, but this has not yet been definitely discovered. The statement of Finkler and Prior, that bacilli are found in the intestinal contents in cholera morbus which can not be distinguished from the comma-bacilli of genuine Asiatic cholera (*q. v.*), has not proved correct. Finkler's bacilli seem to have no pathogenic significance in cholera morbus.

Cholera morbus comes on usually as an epidemic, and almost exclusively in the hot summer months—June to August. Hence it is often termed summer cholera. Children in the first two years of life are chiefly attacked, especially those who are artificially fed or who have recently been weaned. The disease also attacks older children and adults, but much more rarely.

[Special opportunities are afforded in this country for the study of cholera infantum. The causative conditions are, briefly, unsuitable food, a high temperature, and bad hygiene—conditions which are all combined and attain their maximum intensity in large cities. That a high thermometric range alone is not sufficient is shown by the comparative immunity of all country districts. Those who live in the country, or have never been busied among the city poor, have no idea of the atmosphere breathed by the children of the poorer classes, especially during the heated term, nor of the extreme difficulty—impossibility we can almost say—of getting a really good milk. Even if the milk was good at the start and has not been tampered with, the time which necessarily elapses after it is drawn from the cow and before it reaches the consumer permits marked fermentative changes during hot weather. And milk is and must remain the main article of diet for children under two years of age.]

The symptoms of cholera morbus are those of a severe acute gastro-enteritis. The disease begins suddenly, or after some slight warning, with violent vomiting and severe diarrhœa. In some cases one of these symptoms predominates, and in others the other. The vomitus consists partly of the food taken, and partly of a slimy, watery substance. The stools at first retain their fæcal character, but they soon became more colorless and more watery, so that they sometimes approach the well-known rice-water appearance of the stools in genuine cholera. Abdominal pain is usually absent, but a feeling of pressure and constraint in the epigastrium is often present. The diminished secretion of urine and the frequent muscular pains cause the whole type of the disease to resemble genuine cholera still more closely.

The severe disturbance of the general condition is especially characteristic. The patient becomes extremely dull and has a wasted look, the voice is weak and hoarse, an unquenchable thirst sets in, the pulse is very small, the skin of the face and the extremities is cool and livid; in short, we have the pronounced picture of a general collapse. The body heat also falls, although at the first stage of the disease there is often a rise of temperature.

[The temperature is always high, even during the stage of collapse, when the skin and extremities are cool to the touch; if the thermometer is introduced into the rectum—generally the best place, by the way, to take the temperature in young children—it will rise to 101° to 102°, and is more apt to reach 104° to 107°. This shows that inflammation plays a large part in the pathology of the disease.]

The picture of a severe general disease is especially prominent in cholera infantum. In severe cases of this form of the disease the general restlessness, which at first exists, rapidly passes into somnolence. The child lies with sunken, half-closed eyes, the conjunctivæ are slightly injected, the corneæ are cloudy, the face is pale and cyanotic, the fontanelles are depressed, the skin is cool, and the pulse is small and frequent and can scarcely be counted. Amid these symptoms, which are usually termed "hydrocephaloid" by specialists in children's diseases

[Marshall Hall], death comes on in coma or with slight convulsions. There can scarcely remain room for doubt that these severe cases of gastro-enteritis are of infectious origin, and that the constitutional symptoms are the result of toxic matters generated in the intestine under the influence of the micro-organisms (*vide* page 373).

The mortality of children with cholera infantum is very marked, especially in large cities, and among the poorer classes of society. Severe cases usually end fatally in a few days, but, on the other hand, many cases recover, either because the course of the disease from the first is not so severe, or because cases apparently hopeless take a favorable turn. In adults it is extremely rare to see cholera morbus terminate unfavorably. Patients also recover quite rapidly from apparently severe conditions, although the stomach and intestines often remain rather sensitive for a long time.

The anatomical appearances in children who die of cholera infantum contrast, from their insignificance, with the severe symptoms observed during life. The catarrhal affection of the gastric and intestinal mucous membranes is not at all prominent in the cadaver, and the solitary follicles and Peyer's patches show only a slight swelling. The other lesions which are most frequently seen are lobular atelectases in the lungs, and venous hyperæmia and œdema of the pia mater.

The diagnosis of cholera morbus presents no difficulty if the characteristic symptoms of the disease are present. The distinction between it and genuine Asiatic cholera used to be occasionally quite difficult, and was rendered possible only by considering the ætiological factors, and the evident connection between the individual case and other cases of undoubted cholera. By Koch's discovery of the comma-bacilli in Asiatic cholera the distinction between the two diseases has now become absolutely certain. In all suspicious cases, therefore, we must examine the dejections for comma-bacilli, and upon the result of this examination depends the determination of the proper means of prophylaxis.

The treatment of cholera morbus in adults must be first to take special care to limit the diet. The food should be only gruels, or at most broth, soft-boiled eggs, and milk. It is a good plan to give the milk iced, and in small amounts. The distressing thirst is best relieved by cracked ice. Wine (iced champagne) is to be given if the general weakness becomes marked.

Among drugs, opium is the most effective remedy, and, whether in powder, as the extract, or in liquid form, as laudanum, is the first thing to use to relieve the diarrhœa and vomiting. All other remedies which are recommended in cholera morbus in adults, like nitrate of silver, or calomel, are quite subordinate to opium. In other respects we may refer to the treatment of acute gastric and intestinal catarrh.

We are more cautious in prescribing opiates for children, although here small doses of opium, one or two drops of laudanum according to the age of the child, may often be indispensable. In fresh cases calomel has obtained a great reputation, a sixth of a grain (grm. 0·01) two or three times a day. Ice-cold cow's milk, given in teaspoonfuls, serves best as food, if the child can not be fed naturally by breast-milk. As soon as the signs of marked collapse appear, we must use hot baths of chamomile or mustard, hot packs, and also stimulants, small amounts of wine and injections of camphor. If the child's stupor increases, we may under some circumstances use cool packs and shower-baths.

We will pass over the many other remedies recommended for cholera infantum, like quinine, salicylic acid, and creosote, since in severe cases they unfortunately almost always leave us in the lurch. In practice, of course, we are often obliged to try one or another of these remedies.

[Treatment must be prompt and judicious; many a child is thus saved which would otherwise die.

Calomel is less used in this country than in Germany, and I think rightly. It is not very often that a preliminary purgation is required; the bowels have usually been quite sufficiently unloaded before the physician arrives, and the indication he has to meet is to check the vomiting and diarrhœa. Opium, either in the form of Dover's powder and combined with bismuth, or as paregoric, is nearly always in place before the stage of collapse; precise directions should be given to omit the opium or diminish the dose if stupor comes on. Opium can also be given by enema if necessary. One should not wait till the symptoms are desperate before giving brandy, a good way to administer which is by dropping it on a teaspoonful of shaved ice. Improvement is shown by decrease or cessation of the vomiting, and greater consistency with less frequency of the dejections. While the symptoms are at their height there is but little use in trying to give nourishment, which can not be digested even if it is retained.

A warm mustard bath aids in the re-establishment of the cutaneous circulation, and thus tends to relieve the gastro-intestinal tract.

The diet is essentially the same as in the more chronic inflammatory and in the non-inflammatory diarrhœas of young children.

Details to complete the above brief sketch must be sought in works devoted to the diseases of children. It should, however, be added that removal to the pure air of the sea-shore in non-malarial regions will sometimes turn the scale in cases which are apparently hopeless. Pure country air is to be sought if the seaside can not be reached; but the latter is the better of the two.]

CHAPTER III.

INTESTINAL CATARRH OF CHILDREN.

(*Chronic Dyspepsia of Children. Pedatrophy.*)

THE great frequency and the practical importance of the "dyspeptic conditions" in children in the first years of life, which conditions are associated with severe disturbances of nutrition, justify a short description of them, but we must refer to the special manuals on children's diseases for a detailed account.

That diseases of the digestive organs play so large a part in children's troubles is owing, on the one hand, to the great sensitiveness which the digestive apparatus in children shows to the irritants which are brought in contact with it, and, on the other, to the too common foolishness and carelessness which the child's parents and nurses show in its feeding. Of course it is not always ignorance and neglect, but often, unfortunately, poverty and want which cause children to suffer, and explain the terrible mortality in the first years of life.

The simple fact that by far the larger number of children who suffer from dyspeptic and atrophic conditions are fed artificially, leads us to the belief that the cause of most of the intestinal diseases in children is to be found in faulty and injudicious feeding. The food, which is not suited to the child's digestive powers, is only imperfectly absorbed; it undergoes many processes of decomposition, whose products irritate the intestinal mucous membrane and give rise to increased peristaltic action. Thus the imperfect digestion, or "dyspepsia," excites a catarrh of the gastric and intestinal mucous membrane, by which again, in a vicious circle, the digestive power is still further reduced. Hence the boundary between "dyspepsia" and catarrh can be drawn only artificially.

The anatomical changes of the intestinal mucous membrane in children who die of "chronic intestinal catarrh" are, as a rule, only slightly marked, and contrast, in their apparent insignificance, with the severe intestinal symptoms observed during life. Probably here also the toxic influences of the matters abnormally generated in the intestine, play a more important part than do the anatomical changes. We must remember, however, that most catarrhal conditions are hard to recognize in the cadaver because of the disappearance of the hyperæmia. Sometimes the swelling of the follicles is especially marked—follicular catarrh. Follicular ulcers are also seen. In other cases the atrophy of the mucous membrane, which is often seen after chronic catarrhs, is the chief lesion. Chronic thickening and swelling of the mucous membrane is of rarer occurrence. In most of the severe cases the large intestine, and also the lower portion of the ileum, are the chief seat of the changes. We often find a swelling of the mesenteric lymph-glands, and also a fatty liver. In the lungs extensive atelectases or nodules of catarrhal pneumonia often develop as a result of the imperfect respiration.

The symptoms of chronic intestinal catarrh are, in the first place, those due directly to the intestinal trouble, and, secondly, the quite rapid disturbance of the child's general nutrition.

The condition of the stools is the most important intestinal symptom. The normal dejection in children until they are weaned is of the color of the yolk of an egg, of a rather pasty consistency, and of a faintly sour smell. In intestinal catarrh the stools are more frequent, six or seven, and even more, a day. They are thinner, more watery, contain large flakes and lumps of undigested bits of caseine and other remains of food, and smell badly. They very often have a green color, or acquire it on standing. According to recent French investigations (Lesage), this green coloring matter is generated by a specific kind of bacilli, which are also to be regarded as the pathogenic cause of intestinal catarrhs with green evacuations. We may find admixtures of mucus, sometimes in the form of the so-called "sago grains," especially in catarrh of the large intestine. In the severe forms we often find pus-corpuscles and epithelium under the microscope, besides remains of the food. The stools may contain small amounts of blood.

There is no definite distinction in regard to the dejections in catarrh of the large and of the small intestines. On the whole, the rule holds that, in catarrh of the small intestines chiefly, the stools are larger, they are passed with more wind or gas, and show a more uniform consistency; while in catarrh of the large intestine they are smaller but more frequent, ten or twenty a day, are passed noiselessly, are associated with tenesmus, and show a different consistency in their various parts, partly normal, partly thin, partly slimy, etc. Examination of the abdomen is in so far of importance that, in general, in catarrh of the small intestines the abdomen is much swollen, while in catarrh of the large intestine it is often deeply sunken.

We often find disturbances in the stomach, vomiting, eructations, etc., as well as trouble in the intestines. There may be thrush in the mouth, or the development of aphthous ulcers.

In almost all long-continued cases, however, the general disturbance of nutrition, the atrophy (athrepsia) of the child, takes the first place in the picture of the disease. The muscles become shriveled and flabby, and the whole body finally becomes so much emaciated that the pale, dry skin hangs in broad folds and wrinkles about the bones, whose prominences are everywhere visible. The face is sharp, and has an aged expression from the many little folds of the skin. The eyes are dull, lusterless, and wide open; the voice is merely a low, hoarse whimper. The abdomen is deeply sunken, or in some cases it is swollen by meteorism, in peculiar contrast to the emaciation elsewhere; and its surface is traversed by bluish veins.

From this sad picture, unfortunately so frequently seen in practice among children, we can usually recognize the condition of things at the first glance, for by far the larger part of the cases called "pedatrophy" are due to chronic digestive disturbances. Very often it is combined with rachitic changes in the bones, of whose occurrence we shall speak further in the description of rachitis. Tubercular changes, too, are often found in the cadaver, especially in the lungs and the bronchial and mesenteric lymph-glands. In such cases, of course, the tuberculosis is usually to be regarded as the main disease, upon which the intestinal affection, which may be simple or even tubercular, has developed secondarily. During life tuberculosis in little atrophic children may very easily be overlooked.

If we would give a full account of the treatment of the atrophic conditions in children due to digestive disturbances, we must include in our consideration the entire hygiene and care of children in health and disease, for all children's physicians are united in the opinion that, as the cause of most intestinal diseases in children is to be found in improper feeding, so recovery from existing digestive disturbances can take place primarily only by a corresponding proper and judicious feeding. In what follows we can refer only to the most important principles and general points which are here to be considered.

The only proper and natural food for a child in its first year is breast-milk. All dyspeptic conditions are much rarer in children who are nursed than in bottle-fed children, and, when they do occur in children at the breast, they often are only of brief duration. They are then to be referred usually to certain disturbances in the mother, such as disease, insufficient diet, or severe mental excitement. The return of menstruation or a new pregnancy has sometimes an unfavorable influence on the character of the milk. Finally, we may mention that in spite of the best of milk, if the breast is given irregularly and too frequently, it may cause anomalies of digestion in nursing children.

Most of these slight disturbances may easily be readjusted, but sometimes it happens that, without any discoverable reason, the milk of a wet-nurse "does not agree" with the child. Then we must change the nurse. The atrophic conditions which develop and progress in children, in spite of plenty of normal food, are usually due, not to simple digestive disturbances, but to deep-seated, general, constitutional diseases like tuberculosis, or syphilis.

The great majority of cases of chronic intestinal catarrh and chronic atrophy are found, as we have said, in bottle-fed children. The first question which every physician should ask a mother who brings him such a child for treatment, must therefore refer to the sort of feeding which the child has. If the mother, for any reason, can not nurse it herself, and if the bottle-fed child has digestive disturbances, we must invariably consider, in the first place, the possibility of procuring a wet-nurse. Feeding by the milk of a wet-nurse is a remedy which, at least in many cases, by saving the child's life, repays the many annoyances and quite large expense which a wet-nurse causes. We must tell the parents this, and represent to them, without reserve, the great dangers which threaten the life of every bottle-fed baby. Complete, and sometimes even quite rapid, recovery may be obtained through a wet-nurse, even in cases of severe chronic intestinal catarrh, where atrophy and weakness are already very far advanced.

Often, however, it is impossible to hire a wet-nurse, especially in the poorer classes of society. We must continue bottle-feeding, and these are the cases where chronic intestinal catarrh demands the greatest number of victims; yet even here the physician can always do much good by instructing the parents.

The best substitute for mother's milk is cow's milk. This must be as fresh as possible, and is given boiled. The Soxhlet apparatus for preparing the milk is rather troublesome, but very appropriate and advantageous. By its means the milk is completely sterilized. One part of the milk must be diluted, according to its quality, with two or three parts of boiled water in the first months, in children from four to six months old with equal parts of water, and in older children with about half as much water. From nine to twelve months, the child may have undiluted milk. In general we give the milk warmed to about 85° (28° C.), but children with gastro-intestinal catarrh often bear cold milk, given in small amounts, better than warm. Among the special additions to the milk, by which physicians have tried to make cow's milk more like human milk, we may mention as occasionally advantageous sugar of milk, as much as will go on the point of a small knife, added to each portion of milk given, and soda, a tablespoonful of a one- or two-per-cent. solution to a pint (half a litre) of milk. The much-practiced dilution of milk with salep, oat-meal, and barley waters is always injudicious, and it should be a principle, especially with children under three months, to avoid entirely any starchy food. It is not a bad plan to add veal-broth to the milk, as it is sometimes well borne even by weak children.

Cow's milk, properly diluted, is better for children with chronic intestinal catarrh, in many cases, than any other food. In acute digestive disturbances, however, it is sometimes advisable to omit the milk entirely for a few days, and give instead of it only a little mucilaginous drink like decoction of salep. In chronic dyspepsia we must first try good cow's milk. If the milk is not well borne, if the diarrhœa increases, and if the child becomes still more emaciated, we may try to get milk from another and better source; but it often happens that either we can not procure good milk, or that the child can not bear even the best cow's milk. We are then obliged to have recourse to one of the many "artificial foods" and "substitutes for mothers' milk" in the market. We can not here go into particulars concerning these. Each of these preparations has occasional good results to show, but none of them has an uncontested pre-eminence over the rest. We will mention the preparations most in use at present, of whose value in individual cases we have convinced ourselves: Swiss condensed milk, Nestlé's and Frerich's infants' foods, Biedert's cream mixture, and Liebig's soup. Almost every physician has his special favorite preparation, which in his own personal experience has done him relatively the best service.

If we keep fast to the principle that every intestinal catarrh in children is to be treated in the first place by a judicious regulation of the diet, in many cases we shall not have to use any drugs. These may be of service only when we have also carried out the dietetic measures which are specially necessary.

Calomel has obtained the greatest reputation in the treatment of intestinal catarrh in children. It deserves to be tried in fresh cases, in doses of $\frac{1}{12}$ to $\frac{1}{6}$ of a grain (grm. 0·005–0·01) in powder. If the diarrhœa lasts a long time, we may very well use opiates, although with great caution. The combination of calomel and opium often does good service.

℞ Calomel ....gr. $\frac{1}{6}$ (grm. 0·01);
Extracti opii ....gr. $\frac{1}{30}$ (" 0·002);
Pulv. acaciæ.........................gr. ss (" 0·03).
M. et ft. pulv.
Sig.: One such powder, three or four times a day.

With little children we may put two to four drops of laudanum in three ounces (grm. 100) of liquid, like gum mixture, salep decoction, muriatic-acid mixture, etc., and give a dessertspoonful of this every two or three hours.

Many attempts have been made to check the abnormal processes of decomposition in the intestine by prescribing remedies which possess antiseptic and antizymotic properties. Creasote has been warmly recommended by many, four to six drops in two ounces (grm. 50) of water with half an ounce (grm. 15) of syrup, a teaspoonful every two hours. Other drugs used for the same purpose are naphthalin, one-half- to one-per-cent. solution of dilute muriatic acid in water, and one-per-cent. solution of chloral in water. A prescription for naphthalin is as follows:

℞ Naphthalin....................................0·5 to 1·0;
Mucilag. acaciæ,
Aquæ destillat.....................................āā 40·0;
Ol. menthæ pip...gt. j.
M. Sig.: Shake. Teaspoonful every two hours.

If the stools have a green color, it is said that lactic acid is especially beneficial. A teaspoonful of a two-per-cent. solution may be given fifteen minutes after every meal.

A number of other remedies, "astringents," are given to act directly on the diseased mucous membrane. Those most to be recommended in chronic diarrhœa are subnitrate of bismuth, one or two grains (grm. 0·05–0·1) four to six times a day, which may be combined with opium, nitrate of silver (1 to 2,000 solution), alum (1 to 200 solution), guarana, five to fifteen grains (grm. 0·3–1·0), three times a day, and many others.

If a large amount of mucus in the stools points to a catarrh of the large intestine, we may sometimes employ irrigation of the colon with excellent results. We inject the fluid, one-per-cent. solution of tannin or alum, or solution of acetate of lead (1 to 3 to 1,000), once or twice a day. The amount of fluid to be introduced at once, by a Hegar's funnel, with a gum-elastic catheter, may reach one or two pints (half a litre to a litre).

In conclusion, we must mention the advantage of daily warm baths in atrophic children. We often order some special "strengthening" additions to the bath-water, such as salt, iron, or sweet-flag.

[In view of the great importance of this malady, it seems desirable to remark on one or two points.

In the first place, prophylactic measures are deducible directly from the ætiology. No child should be kept in a large city in summer, if it can be provided for at the sea-shore or in the country. The vast number who must perforce remain are to be kept under the best general hygienic conditions possible. Mothers should be encouraged to take their children to the relatively pure air of the public parks as much as they can. The relation between diet and the diarrhœa of children should be dwelt upon whenever there is opportunity.

The establishment of boards of health has done much, and will do more, to check the ravages of summer diarrhœa.

Great advances have been made in the artificial digestion of cow's milk within a few years. By the aid of the preparations of Fairchild Bros. & Foster the caseine is digested in part, and the remaining portions coagulate in light flocculi; at the same time no appreciable taste is imparted to the milk, provided that a moderate amount of care is exercised. The importance of preventing the formation of large, firm curds has long been recognized, and an older means of attaining this end was mechanically to separate the curd by adding to the milk a barley or other similar water-gruel.

Mellin's, Horlick's, and Ridge's foods also deserve mention in this connection.

The quantities of food, as well as the frequency with which it is given, must be determined in each case.

The system of milk inspection which has come into vogue of late years in many places does much to improve the milk supply, as does also an improved method of transportation. But still, especially during the summer heat, a really fresh and pure article is nearly impossible of attainment by the poorer classes in large cities, and it is these classes which swell the mortality from infantile diarrhœa. For them, in particular, the modern simple and inexpensive methods of sterilizing milk may be made of the greatest service. The seashore homes for poor children which have been established near the large cities on the Atlantic seaboard have fully justified themselves.

It seems to the editor that the author has insisted more strongly than is necessary on the vital importance of a wet-nurse, in view of the very great advances which have been made within a few years in the preparation of artificial foods for infants. For full details as to these processes, the reader must be referred to modern treatises on the diseases of children.

The drugs most worthy of confidence are, in the opinion of the editor, opium and bismuth; in mild cases the former is often not required; the dose varies greatly with circumstances ; the latter can safely be given in ten-grain doses, every three hours, to a child of six months. Calomel does not now enjoy the reputation in this country that it does in Germany, though it has its advocates here. For more minute details see works on children's diseases.]

CHAPTER IV.

TYPHLITIS AND PERITYPHLITIS.

(*Typhlitis Stercoralis. Inflammation of the Cæcum.*)

Ætiology and Pathological Anatomy.—Inflammation of the cæcum and its vicinity has a special place among the diseases of single portions of the intestines. The reason why circumscribed inflammation so often develops here is to be found in the peculiar anatomical arrangement of the cæcum and its appendix, the vermiform process. This arrangement explains why fæcal masses or foreign bodies are easily retained in the cæcum, and give rise to an inflammation of it.

Inflammation of the cæcum is due, in most cases, to an accumulation of fæces in it, and hence it is usually termed *typhlitis stercoralis.* Since the causes producing the inflammation are usually permanent in their action, the anatomical inflammatory changes are generally much more intense in perityphlitis than in the other forms of intestinal catarrh. The inflammation attacks the whole intestinal wall, and sometimes even invades the surrounding connective tissue, being then called perityphlitis.

The great majority of severe cases of perityphlitis do not start from the cæcum itself, but from the vermiform appendix. This rudimentary portion of the intestine, so unimportant physiologically, plays a great part in pathology. Small fæcal masses from the cæcum often enter the vermiform appendix, and, under some circumstances, may remain there. The fluid in them is absorbed, they are very often incrusted with lime-salts, and thus the little so-called "fæcal calculi" are formed. In many cases the return of fæcal masses into the cæcum is probably hindered by the valve at the orifice of the vermiform appendix, Gerlach's valve. Foreign bodies, like little seeds of fruit, often enter the vermiform appendix and give rise to the formation of a fæcal calculus. These calculi often have such a rounded shape that they were formerly considered, very erroneously, to be retained cherry-stones.

In many cases fæcal calculi may remain in the vermiform appendix for a long time without producing any further injurious results, but as a rule they cause a mechanical irritation of the mucous membrane which leads to inflammation, and often, in some circumscribed spots, to a pressure necrosis, and later to ulceration of the vermiform appendix. If the ulcer does not cicatrize—it always may—the ulceration gradually deepens. When there is, finally, perforation of the vermiform appendix, we have either a circumscribed or a general purulent peritonitis, according as adhesions have or have not formed in the vicinity. Although general peritonitis almost always ends fatally, the circumscribed purulent perityphlitis may, at least in a number of cases, finally recover (*vide infra*).

Clinical History.—The symptoms of simple typhlitis stercoralis sometimes develop quite rapidly, but in other cases they are preceded by somewhat protracted prodromata. The latter are chiefly constipation, which may at times be interrupted by diarrhœa, and occasional dull pain in the ileo-cæcal region. These symptoms gradually or suddenly increase. The pain in the ileo-cæcal region, in particular, becomes more intense, and prevents the patient from making any considerable movement. Sometimes there is complete stoppage, but in other cases small amounts of fæces are still passed. The patient frequently vomits one or more times. The general health is also much disturbed. The patient, as a rule, is dull, has no appetite, and has a moderately high fever, somewhere between 101° and 104° (38·5°–39·8° C.), the course of which has nothing characteristic.

The most important signs for diagnosis come from the physical examination of the abdomen. The abdomen as a whole is often moderately swollen by meteorism. The collection of gas probably takes place chiefly in the ileum, above the part of the intestine which is narrowed by the accumulation of fæces. The meteorism often is entirely absent or is only slight, and then we may notice by mere inspection a marked prominence in the region of the cæcum. If we examine more closely we perhaps meet with very considerable sensitiveness and tenderness on pressure in the same region. We also feel a resistance, which is either diffuse or well-defined, exactly like a tumor, and which gives flatness or a dull tympanitic resonance on percussion. This characteristic ileo-cæcal tumor, which usually confirms the diagnosis, is caused in part by the collection of masses of fæces, which often seem quite compressible, and permit us to determine approximately the portion of intestine affected, and in part by the materially thickened intestinal walls, and eventually by the inflammatory exudation in the vicinity. In their clinical relations there is no sharp distinction between typhlitis and perityphlitis, and inflammatory processes which rise from the vermiform appendix. The further course only can make the distinction possible.

Most cases of simple typhlitis stercoralis take a favorable course. If the patient has timely care and proper treatment the pain and fever gradually disappear. Large dejections follow, and after ten days to three weeks complete convalescence sets in. The abnormal resistance in the region of the cæcum due to the thickened intestinal walls may often be felt, of course, for a much longer time. A tendency to constipation is also apt to be present for a long time. Relapses are not uncommon, and it not infrequently happens that people who have once had typhlitis may be again attacked later on by the same affection.

Typhlitis takes a more severe course in the happily quite rare cases where the intestinal stenosis as a result of retention of fæces is well marked. The meteorism is greater, the vomiting is more frequent, and finally assumes a decidedly stercoraceous character. The constitutional symptoms are much more severe. The patient is extremely dull, the skin is cool and livid, the pulse is small and frequent. In such cases death may ensue with all the signs of general collapse, if we do not finally succeed in causing the evacuation of fæces and hence removing the obstruction.

The symptoms of perityphlitis are essentially the same as those of typhlitis, but they are usually of a higher degree of severity. The resistance to be felt in the region of the cæcum is less sharply circumscribed and is deeper seated. As a rule there is less meteorism where perityphlitis predominates than in typhlitis. The pain is usually very severe, and often shoots into the right leg, where there may be numbness and formication; but, on the other hand, there are cases in which quite extensive perityphlitic processes cause very slight subjective symptoms for a long time.

[It is to be remembered that the relation of the appendix to the cæcum is not a constant one, and that the seat of the tumor is therefore not precisely the same in all cases. The induration or resistance may extend well up toward the liver or backward into the flank. Especially in those cases in which the appendix lies backward near or over the brim of the pelvis, the tumor may be distinctly felt in the rectum or vagina, the digital exploration of which should never be neglected in cases attended with any uncertainty.]

The course of perityphlitis is always tedious, but in favorable cases we may see complete absorption of the inflammatory products and recovery. Severe cases often end by forming abscesses, which may be ichorous. The local symptoms do not disappear, the fever continues and assumes a septic character. Finally, if the abscess tends to break externally, the swelling in the ileo-cæcal region becomes more prominent and more sharply defined, the skin becomes thin and red, there is fluctuation, and the abscess breaks spontaneously, if it has not been opened previously. Besides perforation outward, we also meet with perforation into the abdominal cavity, with a consecutive general peritonitis, and sometimes perforation into the ascending colon, with evacuation of pus into the bowels and final recovery. [Occasionally the pus is discharged into the bladder.]

An unfavorable complication repeatedly observed in perityphlitis is an extension of the inflammation to the ileo-cæcal vein, which results in a purulent phlebitis of this and also of the portal vein. A general pyæmic condition develops, with chills and marked elevation of temperature. This almost always ends fatally, and at the autopsy we usually find many metastatic abscesses in the liver.

The **diagnosis** of typhlitis and perityphlitis may easily be made in most cases from the peculiar localization of the swelling and tenderness, and by paying attention to the general course of the disease. During life we can merely conjecture whether the special origin of the inflammation is in the cæcum or in the vermiform appendix, but we can never determine this with certainty, since in both cases the type of the disease, as we have said, is almost precisely the same. The further course of the disease is the only means of deciding whether the inflammation has remained circumscribed, or has invaded the adjacent parts in the manner mentioned above.

Chronic cases may be confused with new growths, especially with cancer, arising from the cæcum or the vermiform appendix. Tumors of the right kidney, or the right ovary, and also psoas abscess, after caries of the vertebræ, have in some cases given rise to false diagnoses. In such doubtful cases we are usually able to form a definite opinion only after long and careful observation.

We may mention here in addition that in rare cases there may be a closure in some part of the vermiform appendix. The portion behind this closure is then gradually more and more distended by the secretion of the mucous membrane, and thus the so-called dropsy of the vermiform appendix is developed, which may give rise to a tumor which can be felt in the ileo-cæcal region.

[In brief the early signs and symptoms are those of a local peritonitis, the seat of which is generally highly suggestive of its origin in the appendix. In some cases the symptoms are not indicative of serious trouble, and patients not infrequently walk into the outpatient departments of hospitals for advice. It is unnecessary to say that such should be put at absolute rest in bed with the utmost promptitude.

In women the diagnosis is somewhat complicated by the possibility of acute inflammation about the uterus or its appendages. In both sexes the possibility of the serious error of mistaking appendicitis for typhoid fever is to be constantly borne in mind. Acute intestinal obstruction affords another, though practically less serious, possibility of error.

The tumor may require two or three days for its development.]

The **prognosis** in every case of typhlitis and perityphlitis is to be given with some reserve, since we can not foresee the further course of the disease. A favorable termination, however, is by far the most frequent, and the rule is that the cases are mild and the inflammation is confined to the cæcum. In severe cases of perityphlitis, which end in suppuration, all depends upon whether a general peritonitis ensues or not, and then, if the inflammation be limited, whether the patient's strength is sufficient or not to sustain him until the abscess finally heals. If his strength holds out, he may sometimes recover after the disease has lasted for months.

The **treatment** of typhlitis has two tasks to accomplish: first, to remove the accumulation of fæces, which in most cases has caused and which still keeps up the inflammation, and, in the second place, to prevent, if possible, the further extension of the inflammation, when it has once appeared. Unfortunately, the accomplishment of these two tasks are opposed to each other, and thus, in a given case, it is often hard to decide whether to satisfy the first indication by prescribing cathartics, or the second by prescribing opium. In general, as we believe, the fear of the harm which cathartics may do by tearing any adhesions formed, etc., need not be carried too far. In fresh cases of simple typhlitis stercoralis, which come on with manifest stoppage and a palpable fæcal tumor in the region of the cæcum, we may always prescribe cathartics like castor-oil or rhubarb, if only we exercise due caution about it. When there are copious dejections after a few spoonfuls of castor-oil, the pain and fever usually speedily disappear. If we do not wish to be imprudent in cases where marked tenderness leads us to suspect that the peritoneum is already involved, we may order large enemata of water instead of cathartics, and these often act well. In cases, too, where there are signs of intestinal stenosis, we may hope for the best results from large enemata repeated three or four times a day.

If the inflammation has extended to the parts adjacent to the cæcum, and if, accordingly, we have a perityphlitis, cathartics are no longer proper, and are even sometimes injurious. The treatment then is chiefly to give opium, half a grain to a grain (grm. 0·03–0·06) of the extract every hour or two, according to the severity of the case. By prescribing opium we soonest attain a cessation of the pain and a limitation of the inflammation. If we suspect a large accumulation of fæces besides the perityphlitis, or if symptoms of intestinal stenosis appear, the opium treatment can be combined very well with large enemata.

The local treatment of typhlitis and perityphlitis is often of great service. In most cases an ice-bag on the ileo-cæcal region is well borne and relieves the pain. If there is very much tenderness, we can employ local blood-letting, eight to fifteen leeches, in robust persons, with very good results.

If the signs of a local abscess-formation appear, we may replace the ice-bags by warm compresses or poultices. We do not take the useless trouble of reducing the fever by quinine, but we try to keep up the patient's strength as much as possible. If there is fluctuation externally, we should open the abscess and treat it antiseptically. We must refer to the text-books on surgery for all the details.

[Tait and his followers deprecate the use of opium in peritonitis of this as well as of other forms, and advocate saline laxatives as a means of reducing congestion. The profession at large, however, in this country and in England, do not adopt his views. Absolute rest in bed is to be secured as early as possible, and an exclusively liquid diet is to be enforced, the nourishment being given in such quantities and at such intervals as the condition of the stomach and other features of the case seem to indicate; but four to six ounces every two hours should rarely be exceeded. It should be remembered that grave cases of this affection are of short duration, and that death from simple exhaustion is not to be dreaded. The editor has of late followed a plan of treatment suggested to him by Dr. Sabine, of Brookline, Mass., whose experience with it has been fairly large. No food whatever is given until the temperature is normal, even if this period extend to a fortnight. Water is freely allowed, and small quantities of stimulants may be administered if desirable. Morphine is given in small doses, not exceeding one eighth of a grain, if there is pain or restlessness, however slight. Peristalsis is thus discouraged, the metamorphosis of tissue is delayed, bodily and mental rest are secured. No attempt is made to move the bowels. The favorite local application with us is a hot poultice.

In fulminant cases, with perforation and general peritonitis at the start, the results of surgical and medical treatment are alike bad. In the ordinary run of cases the peritonitis is at first local, though it may at any time become general from rupture of the limiting adhesions, and herein lies one of the main dangers and arises the necessity of the greatest watchfulness. Abscess formation probably does not occur unless perforation takes place; but we have at present no certain criterion, at least during the first three or four days, by which we can determine the existence of perforation. Hence springs the doubt as to whether to operate, and how soon. If the tumor consists only of fæces and simple inflammatory exudation, it will be passed along and absorbed unless perforation takes place. If an abscess has formed, spontaneous cure is highly improbable unless the pus be discharged into the intestine, bladder, or vagina—avenues which are far less desirable than that afforded by an external incision. Fitz advocates waiting for three or four days at least, and then being guided by the development of the general and local symptoms as to whether to operate or not. It may be stated as a general rule that in cases where the existence of perforation is doubtful, but the symptoms as a whole are grave and increasing, it is wiser not to postpone incision, the consequences of an unnecessary but properly performed operation being less serious than those of overmuch delay.

In mild cases, and those in which delay is decided on, opium in some form should be given in sufficient amount to relieve pain and allay restlessness; but anything like narcotism is to be avoided, lest the effects of the opium lead the attendant into a feeling of false security. In recurrent cases it is probably wiser to excise the appendix between attacks, unless the patient is so situated that he can put himself under skilled treatment on the first threatenings of an attack. The necessity of so doing should be carefully explained to him. As to methods of operation, the reader is referred to works on surgery.]

CHAPTER V.

PERFORATING ULCER OF THE DUODENUM.

There is a form of ulcer of the duodenum, especially of its upper, horizontal portion, which is precisely analogous to the round gastric ulcer in regard to ætiology, pathological anatomy, and, very largely, symptomatology. The ulcer is probably also due in most cases to the action of the acid gastric juice on the duodenal mucous membrane, under conditions which have been mentioned more fully in the ætiology of gastric ulcer. We must mention here the noteworthy fact that, after extensive burns of the external skin, ulceration of the duodenum, rarely of the stomach also, has been repeatedly observed. This is probably due to the plugging of a duodenal vessel by an embolus from decomposed masses of blood.

Ulcer of the duodenum is much rarer than the round gastric ulcer, and, in distinction from the latter, it has been found decidedly oftener in men than in women.

Many cases of ulcer of the duodenum run their course entirely without symptoms, or they cause symptoms when sudden hæmorrhage appears, from erosion of the pancreatico-duodenalis, gastro-duodenalis, etc., with hæmatemesis and bloody stools, or the sudden signs of peritonitis from perforation. In many cases a type of disease exists for a long time whose symptoms, as we have said, are so like the clinical symptoms of gastric ulcer that we can very rarely distinguish the two forms with certainty during life. We notice especially continuous or neuralgic pain, which, in ulcer of the duodenum, has its chief seat in the right hypochondrium. Severe gastric symptoms, especially vomiting, are not as common as in gastric ulcer. The general health and general nutrition may remain quite undisturbed for a long time.

Ulcer of the duodenum ends by cicatrization and recovery, or by cicatrization and the formation of stenosis, with secondary dilatation of the upper portion of the duodenum and of the stomach. In regard to the different adhesions and perforations of the ulcer into neighboring organs, we may refer to what has been said of gastric ulcer.

The treatment must be governed by the same principles which were laid down in the treatment of gastric ulcer, especially as the diagnosis is usually doubtful.

CHAPTER VI.

TUBERCULOSIS OF THE INTESTINES.

Tuberculosis of the intestines is in most cases a secondary disease, and is one symptom of a more extensive general tuberculosis. It develops most frequently in the course of chronic pulmonary tuberculosis, and depends here, as we have seen, upon an infection of the intestines by the tubercular sputum that has been swallowed.

Intestinal tuberculosis, however, may also be a primary disease, and the source of further extension of tuberculosis over the body. "Tuberculosis of the abdominal organs," which usually comes from the intestines, has a clinical significance, especially in children. It is not improbable that in such cases the first infection of the intestine comes from without, and that the tubercular poison is taken into the body with the food. Here we must suspect especially the milk from cows with pearly distemper—that is, with tubercular disease.

The anatomical changes in intestinal tuberculosis are precisely analogous to the tubercular changes in other mucous membranes. The tubercular new growth has its origin usually in the lymph-apparatus of the intestine, in the solitary follicles, and in Peyer's patches. The first miliary tubercles form beneath the epithelium, and soon fuse with one another into a diffuse infiltration. In its further course the infiltration on one side extends deeper into the surrounding tissue, so that it attacks the submucous and muscular coats even to the serous coat, and on the other side, by the destruction of the new growth which begins at the surface and constantly spreads, tubercular ulcers are formed. We can often make out with the naked eye single miliary tubercles or groups of them at the base or in the infiltrated edges of the ulcer. This is especially plain in deep-seated ulcers on the corresponding portion of the serous coat. The form of the larger tubercular ulcers is often irregular. In many cases the long diameter of the ulcer is parallel to the circumference of the intestine, so that the girdle-like ulcers, which are especially characteristic of tuberculosis, are formed.

Tubercular ulcers are situated both in the large and in the small intestines. They are usually most marked in the vicinity of the ileo-cæcal valve. Tubercular ulcers in the stomach are extremely rare. Besides the intestinal tuberculosis there is very often tuberculosis of the mesenteric lymph-glands, and also frequently tuberculosis of the peritoneum.

The symptoms of intestinal tuberculosis are usually quite subordinate to the symptoms caused by other co-existing tubercular affections. There may often be quite extensive tubercular ulcers without any marked symptoms, but, as a rule, the onset of diarrhœa turns the attention to the intestinal complication (see the chapter on pulmonary tuberculosis).

Primary tuberculosis of the abdominal organs sometimes presents quite a characteristic type of disease, especially in children. This was termed by the older physicians *tabes mesenterica*. The chief feature of this type of disease consists in a progressive general emaciation and anæmia, which is usually associated with a persistent hectic fever, which obstinately resists all the remedies employed. The abdomen is usually swollen by meteorism, but it is sometimes flat or sunken. In some cases, but less frequently than was formerly believed, we can feel the swollen mesenteric lymph-glands through the abdominal wall during life. The liver may be enlarged and its lower border can often be felt. The bowels are irregular, and there is usually a moderate diarrhœa, persisting in spite of all remedies. The invariably fatal termination is due to an increase of the general marasmus, or to a final acute tubercular affection, like miliary tuberculosis or tubercular meningitis. The autopsy shows tuberculosis of the intestines, peritoneum, lymph-glands, liver, etc., to a greater or less extent. The lungs may be quite free from tuberculosis. We will return to this affection in the description of tuberculosis of the peritoneum.

The treatment of intestinal tuberculosis can be only symptomatic. Besides the general dietetic treatment which seeks to keep up the patient's strength as far as possible, medical interference is demanded by the abdominal pain and diarrhœa. The chief remedy is opium, which, alone or in combination with tannin, acetate of lead, etc., acts most rapidly in relieving the intestinal symptoms. Warm poultices and wet compresses give the best service for local applications.

In other respects the treatment coincides with the general treatment of tuberculosis (*vide supra*).

CHAPTER VII.

SYPHILIS OF THE RECTUM.

In not very rare cases we see in the rectum, especially in its lower portions, extensive syphilitic ulcerations, which produce a severe and practically important type of disease. The more intimate relation between syphilis of the rectum and the general syphilitic process is not perfectly clear. According to quite a widespread opinion, the infection of the rectum comes from the secretion trickling down from the ulcers of the genitals. The facts seem to support this view, since syphilis of the rectum is seen much more frequently in women than in men. Some authors have even asserted that all the so-called "syphilitic" ulcers in the rectum have no connection at all with genuine syphilis, but are chancroids. It is in fact striking, even if it by no means proves such a hypothesis, that, at the autopsy of people who have died of "syphilis of the rectum," we rarely find definite syphilitic changes in other internal organs—a fact which we also can confirm.

The most characteristic mark of syphilitic ulcers in the rectum is their tendency to form cicatrices and stenoses. This result of the ulcer is also important in its clinical relations, since the chief symptoms of the disease usually begin with the development of the stenosis. The seat of the stenosis is usually so low down that we can conveniently reach it with the finger, on a digital examination of the rectum during the patient's life. The rectum narrows like a funnel upward, and we can feel the quite sharp edge of the ring-like cicatrix with the point of the finger. This funnel-shaped stenosis of the rectum is so characteristic of syphilis of that organ that, in almost all cases, we can make the diagnosis with perfect certainty from this alone.

The rectum and the descending colon are usually dilated above the stenosis, and here extensive, irregular ulcerations, with undermined edges, are usually found in the mucous membrane. These are partly of a specific nature, and partly diphtheritic ulcers caused by the pressure of the accumulated fæcal masses.

The symptoms of syphilis of the rectum usually develop quite gradually. At first the bowels are irregular, and there are disturbances of defecation which stubbornly resist the ordinary remedies employed. There are sometimes, in the first stage of the disease, frequent and severe hæmorrhages with the dejections, as we have seen, and for a long time these may falsely be considered to be "bleeding from hæmorrhoids." The symptoms become more marked as cicatrization of the ulcer increases and as stenosis of the rectum develops. There is usually a decided catarrh of the rectum, so that the thin stools contain a large admixture of mucus and pus. The patient's condition is extremely distressing, from the pains with the frequent but always scanty dejections, and from the severe tenesmus. Nodular thickenings and prolapse of the mucous membrane, and sometimes true hæmorrhoids, form about the anus. The patient's strength constantly diminishes from the pain and the continual diarrhœa. He finally becomes emaciated, looks very pale and wretched, and has fever toward night. Death ensues from increasing general weakness, or rarely from a terminal peritonitis due to perforation, after the whole disease has lasted one and a half to two and a half years.

This unfavorable termination unfortunately seems to be the rule in all the cases described; hence the prognosis is to be regarded as very serious in all cases of syphilis of the rectum. Improvement worthy of mention, or even perhaps recovery, is possible only when the disease is recognized at the outset and properly treated.

At the outset of the disease the treatment, of course, must consist chiefly in an energetic general attack upon the syphilis by mercurial inunction and iodide of potassium; but, as soon as the characteristic funnel-shaped stenosis of the rectum has formed, we can not expect much from anti-syphilitic medication, since this can no longer exert any influence on the cicatrices and their results. Improvement is now to be obtained only by mechanical dilatation of the stenosis by bougies, or, if this is not enough, by a surgical operation. A suitable local treatment by irrigation is also of benefit to the catarrh and to the ulcers that still exist in the rectum. Internally, we may continue to use iodide of potassium.

CHAPTER VIII.

CANCER OF THE INTESTINES.

THE development of cancer is far more rare in the intestine than in the stomach. Carcinoma is seen with any frequency only at the lower end of the intestine, in the rectum. In other parts the points of election for the development of carcinoma are the colon, especially at its bends, the cæcum and vermiform appendix, and the small intestine, especially in the region of the papilla duodenalis.

Most cancers of the intestine appear in the form of ring-like swellings that take in the whole circumference of the intestine. More rarely we find a more diffuse papillary proliferation, extending over a larger surface of the intestine. There is often quite an extensive destruction of the new growth on the surface of the cancer, from which deep ulcerations arise. We sometimes find metastases in other organs; for example, the lymph-glands, the abdominal cavity, or the liver. In its histological structure, cancer of the intestine is to be regarded as invariably a cylindrical-celled carcinoma, which sometimes shows a plainly glandular structure—adeno-carcinoma—and sometimes that of the other forms of cancer—scirrhus, medullary, or colloid.

Cancer of the intestines, like all cancers, occurs chiefly, if not invariably, in advanced life.

The clinical symptoms of cancer of the intestines are only in a part of the cases so pronounced that we can make a sure diagnosis of the disease. Cancer of the rectum, however, presents a characteristic picture.

Cancer of the rectum begins usually with distress at stool and pain in the rectum, which at first comes only with defecation, but later becomes almost continuous. The pains often shoot into the neighboring parts—the thighs, the genitals, etc. The local symptoms gradually increase, the stools often contain some mucus and blood, and diarrhœa alternates with obstinate constipation. The patient also becomes emaciated, and constantly grows weaker and more miserable. Finally, we often find a complete paralysis of the sphincter ani, so that a mucous, bloody fluid constantly comes from the half-open anus. The diagnosis can almost always be easily and surely made by digital examination of the rectum. We feel the firm, nodulated proliferations of cancer, and we can usually make out with approximate accuracy its extent and its invasion of neighboring organs, like the vagina and bladder. Examination with the rectal speculum makes the diagnosis more accurate. In some cases the destruction of the new growth may cause perforation into the organs mentioned, and we can easily understand the results of this, such as cystitis, purulent discharges from the vagina, etc. We may also have peritonitis from perforation. Secondary cancer appears with especial frequency in the liver, also in the peritoneum and elsewhere.

Carcinoma of the colon causes, as a rule, only very indefinite symptoms, which for a long time are hard to interpret. These symptoms consist chiefly of distress at stool, obstinate constipation, dull pains in the abdomen, and the signs of slowly increasing general weakness and emaciation. In many cases the stools consist of peculiar, flat, compressed little lumps, which have a certain resemblance to sheep's dung. A similar appearance may also be seen in cancer of the small intestines. In many cases examination of the abdomen gives a negative result, but we can sometimes feel the new growth as an evident tumor through the abdominal wall. In such cases, however, it is almost always difficult to decide upon the seat of the tumor with certainty. It may be very easy to confuse this form with cancers arising from the stomach, the omentum, the mesenteric lymph-glands, etc. We may also be deceived as to the location of the tumor by the fact that sometimes the tumor which is felt in intestinal cancer is not the new growth itself, but corresponds to the fæcal masses collected above it. Carcinoma of the cæcum can often not be distinguished for a long time from the tumors caused by chronic typhlitis and perityphlitis. The patient's age, the tedious course and increasing severity of the disease, and sometimes the swelling of the inguinal glands, are the only things that make us think of a cancer. In a case seen at the surgical clinique in Leipsic, a cancer arising from the vermiform appendix ruptured externally through the skin. The rare instances of cancer of the small intestines cause still greater difficulties in diagnosis. In cases where the tumor can be felt externally we can sometimes make out a marked mobility of the swelling, corresponding to the different positions of the affected loop of intestine. Cancer of the duodenum in many of its relations resembles cancer of the stomach, especially of the pylorus. It also leads finally to dilatation of the stomach with its well-known results, as well as to dilatation of the part of the duodenum above the new growth. Cancers situated in the region of the papilla duodenalis usually cause intense and protracted jaundice.

The prognosis of all intestinal carcinomata is absolutely unfavorable. The disease may sometimes last quite a long time, some two or three years, but in some cases the duration of the special symptoms is brief, a few months or even weeks, apparently because the tumor has previously existed for a long time without symptoms. Intestinal cancer terminates with the signs of increasing loss of strength, or it perforates, causing a terminal purulent peritonitis. Extensive ichorous processes in the surrounding connective tissue, phlebitis, and pyæmia, may follow intestinal cancer. A number of cases die with the signs of a slowly or rapidly developing intestinal stenosis (*vide infra*), but in some cases, upon ulceration of the cancer, the already pronounced symptoms of stenosis may disappear for a time.

The treatment must be confined to relieving the patient's symptoms as far as possible. We must provide for easy dejections by a suitable diet and cathartics. Pain, if it is present, must be lessened by narcotics. Surgical treatment of intestinal cancer has shown good results as yet only in cancer of the rectum. The palliation which lasts for a long time after scraping out the rectum is often quite marked, even in advanced cases. In early cases, permanent relief may be given by extirpation of the rectum. If the cancer has advanced too far for this, scraping out the rectum with a sharp spoon may lessen the discomfort at least for a time. All the details are to be found in the text-books on surgery.

CHAPTER IX.

HÆMORRHOIDS.

By the name "hæmorrhoids" we mean diffuse or varicose dilatations of the hæmorrhoidal veins, especially of the venous plexuses at the lower end of the rectum. Hæmorrhoids are single large varices, which usually rise from the submucous layer, and push the mucous membrane out before them. If they are situated outside of the sphincter ani we speak of external hæmorrhoids, in distinction from internal hæmorrhoids, which lie above the sphincter. The size of the nodules varies with the fullness of the dilated veins; but hæmorrhoids, as a rule, do not consist exclusively of dilated vessels, for we often find, at the same time, quite a considerable thickening of the surrounding connective tissue, so that the whole mucous membrane has a swollen appearance, with a polypoid proliferation in parts. The hæmorrhoids usually present themselves as bluish tumors, from the size of a pea to that of a walnut, which surround the anus like a garland. Many of them have a broad base, while others are pedunculated.

The cause of hæmorrhoids is, chiefly, frequently-repeated stasis in the veins affected. The hindrance to the venous blood return has sometimes a purely local cause. Thus hæmorrhoids quite frequently develop in persons with habitual constipation, and hence in those who lead a sedentary life. Hæmorrhoids also occur as a result of stasis in the portal system, in cirrhosis of the liver, etc., and finally in general disturbance of the circulation, as in diseases of the heart and lungs. Quite often, however, we can discover no sufficient cause for the development of the disease, and we are then forced to the hypothesis of a local disease of the affected venous plexus, which is probably often connected with an individual, and apparently sometimes hereditary, predisposition. We most frequently see hæmorrhoids in men in middle life.

Hæmorrhoids sometimes cause only slight symptoms, or none at all, but in other cases they are a tedious, burdensome, and even distressing evil. The chief symptom is pain, which is felt as a constant burning at the anus, but which increases to great severity at each dejection. There is much pain when the hæmorrhoids and the surrounding tissue gradually get into an inflamed condition. In the skin about the anus, erythema, excoriations, and sometimes little but very painful fissures are formed. The mucous membrane at the lower end of the rectum is often found in a catarrhal state, which gives rise to the presence of pus and mucus in the dejections—"mucous hæmorrhoids." Sometimes there is a genuine phlebitis in individual hæmorrhoids, which ends by forming an abscess. There is very severe pain if an internal hæmorrhoid is pushed out by the strain and pressure at stool and is strangulated by the sphincter. Since all the conditions mentioned—marked temporary filling, inflammation, and strangulation of a hæmorrhoid—must at times give rise to a great increase of the disturbance, we can understand why we often hear such troubles spoken of as "attacks of hæmorrhoids."

Hæmorrhoidal bleeding is a frequent and familiar symptom, which rises from a rupture of the dilated veins, and usually comes on at stool. The hæmorrhage is seldom very large, so that the loss of blood is in itself very rarely dangerous. The swelling of the varices after the hæmorrhages have ceased explains why the hæmorrhoidal symptoms are apt to be less marked as long as there are hæmorrhages than when there are none. Hence the old term of the "golden vein" for hæmorrhoidal bleeding.

Besides the local symptoms mentioned, in the anus, there are sometimes other symptoms which are due to an implication of the neighboring venous plexuses, the vesical, prostatic, and sacral plexuses. There is often pain in the sacral region, difficulty in micturition, and sometimes even blood in the urine—"vesical hæmorrhoids"—and in women vaginal catarrh, anomalies of menstruation, etc. Since the symptoms of some primary disease in the liver, or heart, or of other co-existing morbid conditions, like abnormal corpulency, or chronic gastro-intestinal catarrh, may be added to the general picture, we can comprehend why medical superstition has found in hæmorrhoids an excuse for the strangest ideas, like that of "transposed hæmorrhoids"!

The treatment of hæmorrhoids is usually no easy task, since the disease often has causal factors which can not be removed. In all cases where there are large hæmorrhoids causing severe symptoms, there is only one radical cure—removal by operation—which is not dangerous and not hard to perform. It is best done by squeezing the nodule with a clamp and burning it with the thermo-cautery. Details in regard to it may be found in the text-books of surgery.

If single nodules are inflamed, we may apply ice locally, or under some circumstances local blood-letting is to be preferred. If an abscess forms it must be opened. We try to replace strangulated hæmorrhoids carefully and slowly with the oiled finger.

The treatment of chronic hæmorrhoidal symptoms consists chiefly in looking out for regular and easy movements of the bowels, because the local discomfort can thus be best relieved. We must also attend to any underlying trouble, like disease of the liver or heart. The food to be prescribed depends upon the patient's physical condition. It is usually wise to limit the supply of meat, and to recommend instead fruit, vegetables, light farinaceous food, and rice. It is well to prescribe sufficient physical exercise, cool baths, under some circumstances sitz-baths, and rubbing. We must also consider cathartics, especially the bitter waters, the springs at Marienbad and Kissingen, and the regular use of cold enemata, rhubarb, aloes, etc. Sulphur is a remedy very often used in the treatment of hæmorrhoids, the chief ingredient of most of the "pile powders" for internal use. It may be ordered thus:

℞ Sulphuris loti,
Potassii bitartratis, } .... āā ℥ ss. (grm. 15);
Sacchari albi,
Elæosacchari citri,* } .... āā ʒ ijss. (grm. 10). M.

Hæmorrhoidal bleedings, as has been said, are only exceptionally so severe as to require interference. We may use ice, chloride of iron, or tamponing the rectum.

[Persistent and intelligent treatment will often bring about very great relief, or even complete cure, in chronic cases. There are many local applications, each of which has its warm advocates—so many that it is not possible here to go into details. Suffice it to say that an astringent with or without an anodyne may be used as an enema, in suppository or in ointment. The fluid extract of hamamelis, glycerine, and some other remedies given by the mouth, are reported to have afforded good results.]

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* Powdered sugar, ʒ j; volatile oil, gtt. ij. M.

CHAPTER X.

HABITUAL CONSTIPATION.

A PERSISTENT tendency to constipation is a frequent symptom in many different diseases, where it is almost always due to a diminution of the normal peristaltic movements of the intestines. In many conditions this diminished energy of peristalsis is only one symptom of general weakness of the body. Thus we see in all possible forms of chronic disease, which are associated with loss of flesh and strength, that the intestinal movements become sluggish, and hence the dejections are delayed; yet many other causes have a similar action. The small amount of food taken, and its quality, as it is often composed largely of fluids and "non-irritating" substances, and also the rest in bed, or at least the slight amount of physical exercise taken—all these conditions play a part in the constipation frequently seen in patients with chronic disease.

In other cases we have to do with disease of the intestine itself, as the cause of the habitual constipation. In chronic primary and secondary intestinal catarrh we often observe a persistent tendency to constipation, which is only occasionally interrupted by diarrhœa. Many factors also act here simultaneously. The chronically inflamed membrane, which is often covered with mucus, is less irritable, and hence it is more difficult to excite the intestinal movements by reflex action than if the intestinal mucous membrane is normal. The muscular coat itself often takes part in the morbid changes, and atrophy of it has been repeatedly observed as a result of chronic intestinal catarrh. The habitual constipation in chronic affections of the peritoneum is explained in a similar way, as the muscular coat is also directly affected by collateral œdema, etc. We must mention here the constipation in all forms of chronic jaundice, which depends in part at least upon the absence of the irritation which the bile normally exerts upon the intestinal wall.

We very often see chronic constipation in the different diseases of the nervous system, especially of the brain and spinal cord. Here we have to do with abnormal inhibition or direct disturbance of the nervous stimulus, which is necessary to cause the intestinal movements. Abnormal mental conditions are also of great influence. In many psychoses, especially in hypochondriasis, melancholia, and many forms of hysteria and neurasthenia, we very often see habitual constipation.

While constipation, in the diseases thus far described, is a symptom which is more or less subordinate to the other symptoms of the disease, there is a form of habitual constipation, extremely important practically, where the constipation is the chief or almost the sole symptom, and accordingly it may be regarded to a certain extent as a disease *sui generis*. Patients very often come to the physician who look perfectly well, and are entirely able to attend to their business, but who are constantly troubled because they can not have a movement of the bowels every day, like other people, but only every three or four days, or even less frequently. In some cases of this sort the patient's complaint is limited to the delay in the stools, but more often a number of other abnormal subjective sensations and disturbances are added to the habitual constipation, and these are regarded as a result of the constipation by the patient himself, and are usually observed with great care and excessive accuracy. These are the cases which may lead to the highest degree of hypochondriasis. The patient's whole thought and consciousness are busied almost exclusively with his own morbid condition, through which he has lost all energy and joy in life. He seeks aid from different physicians and from quacks, usually without any real confidence, and without having the perseverance to follow out the directions given. Besides the trouble with the bowels, such patients complain chiefly of dizziness, of pressure, coldness, and other abnormal sensations in the extremities, very often of cold sweaty hands, of a feeling of oppression in the chest, of disturbed sleep, etc.

It is not always easy to interpret these cases correctly. The nervous affection, like hypochondriasis or neurasthenia, is often the primary disease, which is followed by constipation, while in other cases the habitual constipation leads secondarily to the nervous depression. The two conditions usually form a vicious circle, since each of them is apt to keep up and to increase the other. The cause of primary habitual constipation can not usually be discovered. Probably we often have to do with a congenital weakness of the muscles or of the innervation of the intestines, since many cases date from early youth.

The treatment of habitual constipation is a difficult and often a thankless task, and it demands patience and professional tact. It goes without saying that we must first of all look for the causal factors. If we succeed in improving the underlying disease—as, for instance, the chronic gastro-intestinal catarrh, the chronic affections of the heart or lungs, the anæmic conditions, or certain nervous troubles —a regulation of the bowels often follows of itself. In ordinary habitual constipation we must first attend to the patient's diet. Since most of these patients also suffer from symptoms of nervous dyspepsia, they are usually very careful in their diet, and take only a little, easily digestible, and chiefly liquid food. It is no wonder that no good dejections follow such food. Improvement can be obtained only by eating plenty of food which can mechanically stimulate the intestine. Hence we must try to bring the patient back to ordinary " household fare "—to take, besides plenty of meat, a sufficient amount of bread, vegetables, etc. It is a very good thing to recommend especially certain kinds of bread, like Graham-bread or rye-bread, and also larger amounts of butter, besides fruit, prunes, grapes, dates, figs, almonds, nuts, and honey. The well-known remedy of drinking a glass of cold water in the morning before breakfast is deservedly popular. We should be very guarded in giving special cathartics, since the patient easily gets accustomed to them, and then must take them in larger doses. Of the milder laxatives, the different bitter waters like Friedrichshall, do the best service. We usually prescribe one or two wine-glassfuls. The other cathartics, like tamarinds, rhubarb, aloes, gamboge, or jalap, which may sometimes be used regularly for a long time, are prescribed in various combinations, as pills and powders. We may often have to change our remedies and our doses a number of times until we find the right ones.

In the treatment of habitual constipation associated with hypochondriasis, the first rule is to treat the patient's mental condition. We should not make merry over his trouble, nor should we rudely let him feel that we do not consider his complaints so important as he himself imagines. The patient does not deserve to be scoffed at, since his subjective symptoms are to him of the most urgent nature; but it is extremely important to divert his thoughts from his trouble. As in many other reflex processes, so in defecation, the voluntary attention abnormally directed to it has an inhibitory action. Hence we admonish the patient to think of his trouble as little as possible and to begin his regular activities again, and we try to convince him of the groundlessness of his fears. The cathartics, which most patients have already taken freely without the desired action, are usually of no advantage at all. On the contrary, it is almost always necessary to forbid the patient to use cathartics at all. Except by a proper diet (*vide supra*), we try to regulate the peristalsis by external remedies alone. Methodical massage of the abdomen is most employed for this, and also electrical treatment, faradization of the abdominal walls, and faradization and galvanization transversely through the abdomen. To be sure, a good part of the benefit of these methods depends upon their moral effect upon the patient. We must not omit a proper general treatment: cold sponging, baths, a country residence, and sufficient physical exercise. Finally, it is often very useful to cause the patient to go to the closet at a fixed hour daily, even if there be no special desire for a stool, and to try to have a movement of the bowels. Thus a sort of training and education of the bowels is achieved, even where the patient at first believes it impossible.

By these means only do we succeed in giving the patient renewed courage, and sometimes finally in attaining recovery even in severe and persistent cases. (Compare the chapters on nervous dyspepsia and on neurasthenia.)

CHAPTER XI.

STRICTURE AND OBSTRUCTION OF THE INTESTINES.

Ætiology and Pathological Anatomy.—Different pathological processes may lead to stricture or complete obstruction of the intestinal tube in different parts. Since in this affection the purely mechanical effect of the intestinal stenosis is the chief cause of the clinical symptoms, the type of the disease is very similar in all the cases of this class, in spite of the manifold anatomical causes. Hence, after enumerating the individual affections which may lead to stricture of the intestines, we can describe their symptoms in common.

The anatomical causes of stricture or obstruction of the intestines are as follows:

1. *Congenital closure* of the intestines is found at the anus, *atresia ani*, and much less frequently in the colon or small intestines. The form first mentioned is the only one of clinical interest, since it may be relieved, at least in some cases, by operation. All the other forms of congenital closure of the intestines are incompatible with a long duration of life.

2. *Tumors and Cicatricial Strictures.*—Cancer of the intestine is the only tumor that has any clinical significance. We have already described its most important anatomical relations and the possibility of intestinal stenosis as a result of it.

We see cicatricial strictures most frequently in the large intestine after recovery from dysenteric ulcers. The syphilitic stenosis of the rectum, which we have already described, is also of practical importance. Typhoid ulcers very rarely lead to cicatricial stenosis. Strictures as a result of tubercular ulcers of the intestines are also extremely rare. Stenosis of the duodenum after the healing of a duodenal ulcer (*vide supra*) resembles, in its clinical symptoms, stenosis of the pylorus, and not stenosis of the intestines.

3. *Intestinal Obstruction.*—The most frequent form of intestinal obstruction comes from the impaction of fæces. From the different conditions which cause enfeeblement of the peristaltic movements, an accumulation of fæces (*coprostasis*) may arise, especially in the colon, and gradually increase, and finally lead to the fully developed symptoms of intestinal stenosis. Since in such cases a paralysis of the muscular coat has been supposed to be the first cause of the constipation, the occasional stercoraceous vomiting which finally sets in has been termed "*ileus paralyticus.*" We must also mention that, in intestinal stenosis from other anatomical causes, coprostasis is often an important factor in increasing the stenosis.

We see obstruction of the intestines from other causes much less frequently than from impaction of fæces. In some cases impacted gall-stones have been found, especially in the lower part of the ileum, almost completely stopping up the lumen of the intestine. Genuine intestinal calculi may exceptionally lead to obstruction. We must also mention here the very rare cases where a large foreign body has been swallowed and wedged itself into some part of the intestine. Such a thing has been seen, especially in children and among the insane.

4. *Intestinal Constriction.*—Although the mechanism of intestinal constriction in external herniæ lies in the domain of surgery, we must mention here the chief causes of the so-called internal intestinal constriction, internal incarceration, or strangulation. In the abdominal cavity itself pouches and diverticula are found, either as normal or abnormal formations, in which single loops of intestine may be caught and constricted. The duodeno-jejunal hernia—the so-called Treitz's retro-peritoneal hernia—is worthy of special mention, and comes from the entrance of a loop of intestine into the duodeno-jejunal fossa. This hernia may become very large. It is sometimes found by accident in the cadaver, not having caused any symptoms during life, but in rare cases it may be the cause of acute internal constriction. We must also mention the hernia of the omental bursa—where a loop of intestine passes through the foramen of Winslow—the intersigmoid hernia, the subcæcal hernia, etc. Diaphragmatic hernia is of greater practical significance because it is somewhat commoner. By this name we designate both genuine protrusions into the diaphragm, and also the passage of abdominal viscera through congenital or acquired (traumatic) defects in the diaphragm. These herniæ may exist without symptoms, or at least without causing any signs of severe disease, but in some cases they cause obstruction by constricting or twisting a dislocated loop of intestine.

Those cases in which abnormal slits and holes in the omentum or mesentery give rise to internal constriction are to be added to the list of the internal herniæ.

Finally, abnormal fibers, membranes, and false ligaments in the abdominal cavity are a comparatively frequent cause of internal constriction. Such cords and bands are sometimes left as the results of a former peritonitis, and may cause actual constriction or bending of single loops of intestine. One such false ligament, which must be specially mentioned, and which may cause intestinal constriction, is found as a prolongation of Meckel's diverticulum. By this we mean that diverticulum which must be regarded as the remains of the omphalomesenteric duct, still persisting, which has its seat, corresponding to the duct, from half a metre to a metre above the ileo-cæcal valve. A firm cord sometimes arises from the free end of this diverticulum, the obliterated omphalo-mesenteric vein, which adheres to some part of the internal abdominal wall and may cause constriction of the intestine. Adhesion of the free end of the vermiform appendix has been the cause of internal constriction in some cases.

5. *Twists* (*volvulus*) *and Knots of the Intestine.*—Twists about the mesenteric axis, and complete constriction of a portion of intestine from this cause, are seen most frequently in the sigmoid flexure, especially if the mesentery of the flexure is unusually narrow congenitally. The spontaneous correction of this abnormal condition is hindered by the weight of the loops of intestine filled with gas and masses of fæces, and by other portions of intestine lying on the place of twisting. Sometimes other portions of intestine wind themselves several times about the pedicle of the twisted loop so as to form a regular knot. Such twistings have been seen especially between the sigmoid flexure and a portion of the ileum. External injury sometimes gives rise to the formation of a knot. In some cases abnormally great peristalsis, severe diarrhœa, precedes the appearance of obstruction. We have ourselves seen a case of volvulus in the upper part of the small intestines, as a result of very severe vomiting caused by a remedy for tape worm given by a quack!

6. *Invagination of the Intestine* (*Intussusception*).—If a portion of intestine is pushed into the lumen of the portion that lies next below, we term the process

invagination. The cause of this is probably to be looked for in a diminution or a complete absence of peristalsis in a circumscribed portion of intestine. If now there are energetic movements in the portion immediately above, they push this into the paralyzed portion. In other cases we may, perhaps, suspect a spasmodic state of the muscular coat. We find invagination of the ileum most frequently in the bodies of atrophic children. In such cases it is an ante-mortem symptom, due to the different periods of cessation of peristalsis in the different parts of the intestines.

Besides this invagination, which has only an anatomical interest, sudden intussusceptions are seen, especially often in children up to ten years of age, for which we can usually discover no certain cause, and which in a short time lead to the severest symptoms of intestinal stenosis. Such intussusceptions, which often involve quite long portions of the intestine, may have their seat in almost any part of the intestine. The invagination of the cæcum and a portion of the lower part of the ileum into the colon (ileo-cæcal invagination) is relatively the most frequent. These intussusceptions may sometimes reach such an extent in children that the invaginated portion of the ileum may finally reach the rectum, and sometimes even project externally. Inflammation and adhesions usually appear in the part invaginated. Gangrene of the internal portion, from the constriction of the afferent vessels, is also common. The necrotic portion may be cast off and passed with the dejections, a process which, in some cases, has led to a spontaneous healing of the intussusception and to a cure of the intestinal obstruction caused by it.

We must mention intestinal polypi as a special cause of intussusception, as they gradually pull that portion of the intestine in which they are situated into the neighboring portion next below by their weight. This has been repeatedly observed.

7. *Compression of the Intestine from without*, by tumors of the uterus, ovarian cysts, pelvic abscesses, omental tumors, etc., has been met with in rare cases as a cause of intestinal stenosis. The symptoms of stricture in such cases develop either very gradually or sometimes rather suddenly.

We must now mention certain pathological changes which may follow every obstruction, from whatever causes it may arise.

The further changes in the intestine deserve the chief attention. Above the constricted point it is usually greatly swollen from gas and the accumulation of fæces. The whole intestinal wall is found in an inflamed condition, which is due in part to mechanical action, and in part to the inflammatory germs which are abundantly developed in the abnormally situated intestinal contents. A severe diphtheritic process often develops in the intestine, with ulceration, above the stenosis. In the inflamed, softened intestinal wall, thin from its abnormal distention, a little tear easily occurs in some spot, or more rarely a genuine perforation following an ulcer. Some of the putrefying contents of the intestine thus enter the abdominal cavity, and an intense purulent or ichorous peritonitis is unavoidable. This is why acute peritonitis is so frequent a lesion in persons who die of intestinal obstruction. If the intestinal stenosis has lasted a long time, we usually find in the upper portion of the intestine, besides the signs of inflammation, a manifest hypertrophy of the muscular coat, the result of the abnormally active peristalsis by which the muscle has tried to overcome the obstacle. The intestine below the constriction, in contrast to the part just described, appears narrow, contracted, and empty.

The changes in the other organs correspond to the general inanition. The frequent development of inhalation-pneumonia is easily explained, if severe vomiting has preceded (*vide infra*).

Clinical History.—In regard to the clinical symptoms we must distinguish the cases with a rapid, complete obstruction of the intestine from those in which the condition develops gradually, and where there is, therefore, merely a constriction of the intestine, at least for a time.

The first symptom of the partial **intestinal constrictions,** which arise from cicatricial strictures and new growths, from partial blocking up of the lumen, from intussusceptions, etc., is usually a disturbance in defecation. The bowels are costive, they move only at long intervals, and their motion is often associated with pain and tenesmus. In the description of cancer of the intestines we have already mentioned that the fæces passed sometimes have a peculiar, flat, compressed, or scybalous form. Blood and mucus are often mixed with the dejections and are due to the character of the primary disease. In some cases there is no constipation, and there may be even constant diarrhœa. We can easily understand from the physiological conditions that in stenosis of the small intestines, whose contents have an approximately fluid consistency, disturbances of defecation are less apt to take place than in stenosis of the large intestine, where the fæcal masses have already assumed a more firm consistency.

Physical examination of the abdomen often gives important and valuable information. The abdomen is usually swollen by meteorism, which arises from the accumulation of gas above the constricted portion. The intensity of the meteorism varies very much in different cases and at different times in the same patient. Meteorism is sometimes absent, especially in stenosis at the beginning of the intestine. There may then be gastrectasis. The marked peristaltic movements, plainly visible through the abdominal walls, are very characteristic of most intestinal constrictions. The contour of single loops of intestine is often marked, at times quite sharply, and then we can sometimes feel the thickened intestinal walls through the lax abdominal wall. We may often decide upon the seat of the stenosis from the location and course of the visible peristaltic movements. In general, it is true that peristalsis is much more noticeable when the stenosis is in the small intestine than when it is in the colon. We must finally state that we have been repeatedly struck by the great extent and strength with which we could feel the pulsation of the aorta through the swollen loops of intestine. If we put our ear to the anterior abdominal wall we can often hear many gurgling and splashing noises, which sometimes have a distinct metallic quality. Eructations are frequent, and in some cases there is occasional vomiting.

The duration of all these symptoms differs with the form of the primary disease. Either gradually or sometimes quite suddenly the symptoms of intestinal constriction pass into those of obstruction. Then the same type of disease develops as is seen in all acute internal strangulations.

The symptoms of **intestinal obstruction** form one of the severest and most frightful conditions known to pathology. The patient's general condition in a short time undergoes a threatening change for the worse. The signs of collapse rapidly develop; the face sinks in and assumes a wasted and sharp expression, the extremities become cool and livid, the pulse is frequent and can scarcely be felt, the voice is weak and obscure. The temperature usually falls, but it occasionally rises. The abdomen is much swollen from meteorism, and is usually very tender on pressure from beginning peritonitis. The passage of fæces and the escape of flatus cease entirely. We often see the peristaltic motions of the intestines above the obstruction through the abdominal walls, but in some cases the muscular coat is so paretic that it is no longer capable of marked peristalsis.

The most characteristic symptom of intestinal obstruction is the vomiting of feculent-smelling masses, the so-called stercoraceous vomiting (*ileus*, *miserere*). There is often frequent eructation at the beginning of the attack, which alternates with real vomiting. The vomitus at first is of the usual character, but it soon acquires a manifestly putrid, fæcal odor. The old opinion is false that in this vomiting real fæcal masses were forced backward from the large intestine into the stomach by an antiperistaltic action of the intestine. Stercoraceous vomiting occurs, not only when the obstruction is in the large intestine, but also in obstruction of the small intestines. In this case we have to do with a putrid decomposition of the contents of the intestine stagnating above the obstruction. Part of this putrid mass reaches the stomach during vomiting, since the pylorus gradually yields to the increasing swelling of the small intestines. The vomiting itself is probably caused in large part by the pulling on the peritoneum, and perhaps by the irritation of the abnormal matter which has entered the stomach.

We must mention, finally, certain facts observed in the different forms of intestinal stenosis, which are of theoretical interest and also of diagnostic importance. In the contents of the intestine stagnating above the stenosis, large amounts of indol and phenol are formed, chiefly from the decomposition of the albuminous substances, and also from the other products of putrefaction. These are in part absorbed and excreted with the urine. Hence, in stenosis of the small intestines we often find that the urine contains an increased amount of indican* (Jaffé) and phenol (Brieger), but in stenosis of the large intestine the amount of indican in the urine is not increased, because the albuminous substances capable of decomposition are no longer present in the contents of the large intestine to cause it.

The course of intestinal obstruction differs according to the anatomical causes which exist in different cases. In many cases of acute internal strangulation the severe type of general disease above described develops in a very short time, and may lead to death in a day or two, but usually the course is somewhat longer and lasts a week. In intestinal obstruction which develops gradually from intestinal constriction, the disease may go on longer and show many variations in its intensity. In mere intestinal constriction we can make fewer definite statements as to the duration and course of the affection, since the symptoms of the disease depend entirely upon the form of the primary disorder.

In a great majority of cases intestinal obstruction terminates unfavorably. Death results either from increasing collapse or from secondary peritonitis (*vide supra*), or in rare cases from further complications, like pyæmic conditions or pneumonia. Recovery may occur even after the severest symptoms, but it is very rare. The obstructions from impactions are most hopeful for recovery. Impacted gall-stones, fæcal accumulations, etc., may be evacuated, after which the severe symptoms disappear. The possibility of recovery in intussusception, by throwing off the gangrenous internal portion of intestine, has been mentioned above. We can not wholly deny that internal strangulations are capable of restoration, although the prognosis must almost always remain doubtful on account of the uncertainty of the diagnosis in any individual case.

In the partial intestinal constrictions, too, the nature of the trouble causes an unfavorable termination in most cases, either from the primary disease itself or from the complete obstruction that finally follows, but the possibility of recovery can not be wholly excluded in certain conditions, like impaction or external compression.

To go into details as to the clinical symptoms of the separate forms of intestinal constriction and obstruction would lead merely to repetitions. In most of the acute and many chronic cases the diagnosis can generally be made only as to the presence of a mechanical obstacle in the intestine, but the nature of the obstruction can at best be merely surmised. Individual factors in reference to this, and in regard to the question of the seat of the stenosis, are contained in the account given above of the ætiology and symptomatology.

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* The indican test is performed in the following way: We mix equal volumes of urine and officinal hydrochloric acid (P. G.), and then add, drop by drop, a concentrated solution of chloride of lime, shaking it after each drop. If now chloroform is added, upon shaking again, the chloroform will take on a striking blue color, if the urine contains any considerable amount of indican.

[The diagnosis of the nature of an intestinal obstruction is so difficult in many cases, and yet so important with reference to treatment, that the editor ventures to introduce tables of differential diagnosis of the more common forms of the condition. These tables are based upon the masterly prize-essay of Treves, of London.

ACUTE INTESTINAL OBSTRUCTION.

CHIEF COMMON SYMPTOMS.—Sudden *pain*, intermittent or constant, with exacerbations ; tends to become constant with time. *Vomiting*, early, severe, becoming feculent. *Constipation*, more or less absolute. Abdominal *distention*. *Shock*.

	STRANGULATION BY BANDS OR THROUGH APERTURES (25 *per cent. of all cases of acute obstruction*).	VOLVULUS OF COLON.	ACUTE INTUSSUSCEPTION.
AGE AND SEX	Young adults; rare after 40.	Males as 4 : 1 ; 40 to 60.	More than 50 per cent. under 10 years.
HISTORY	Previous peritonitis in 68 per cent.; previous attacks of obstruction in 12 per cent.	Previous constipation.	Usually negative.
ONSET	Sudden in 70 per cent.	Sudden.	Sudden in 75 per cent.
PAIN	Early, severe, continuous, with exacerbations.	Early, less severe, intermittent at first, becoming constant with exacerbations.	Early and severe ; increasing and later subsiding; at first paroxysmal.
LOCAL TENDERNESS	Absent at first, appears later.	Early over distended coil, and constant.	Common about a tumor.
VOMITING	Early, marked; in 60 per cent. becomes feculent ; affords no relief.	Less early, severe, and constant ; often affords relief.	Still less early and severe ; in 25 per cent. becomes feculent.
CONSTIPATION	Continuous and absolute; no blood.	Early and absolute ; no blood.	Absolute rare ; diarrhœa not uncommon ; blood in 80 per cent.
PROSTRATION	Marked.	Rather less marked; may be dyspnœa.	Marked.
TENESMUS	Absent.	In 15 per cent.	In 55 per cent., and often early.
ABDOMINAL WALL	Flaccid unless peritonitis.	Rigid from early peritonitis.	Flaccid unless peritonitis.
TUMOR	Very rare.	Absent.	In 50 per cent.; invagination sometimes felt in rectum.
METEORISM	Slight, appears about third day.	Early, rapid, increases, and is extreme.	Rare, unless marked constipation.

N. B.—No trustworthy conclusions can be drawn from the seat of the pain as to the seat of the obstruction unless local peritonitis comes on. The pain is usually referred in all forms to the region of the navel. In complete obstruction the pain is constant, though with exacerbations ; intermittent pain shows that the obstruction is partial. Coils of intestine are not visible through the abdominal wall in acute cases.

CHRONIC INTESTINAL OBSTRUCTION.

	STRICTURE OF THE SMALL GUT.	STRICTURE OF THE LARGE GUT.	FÆCAL ACCUMULATION.
AGE AND SEX	Adults.	Adults.	Adults ; more common in females; the hysterical, lunatics, hypochondriacs.
HISTORY	Cancer, trauma, tuberculosis; disordered, imperfect, irregular action of bowels from time to time, with intervals of comparative ease.	Cancer, trauma, tuberculosis, dysentery; disordered, imperfect, irregular action of bowels from time to time, with intervals of comparative ease.	Previous constipation.
ONSET	Gradual.	Gradual.	Gradual.
PAIN	Intermittent.	Intermittent.	Less prominent.
VOMITING	Late, scanty, feculent only toward end of acute attack ; may be provoked by food.	Less prominent, rarely feculent or provoked by food.	Late, scanty, rarely feculent, often absent.
CONSTIPATION	May alternate with diarrhœa; blood points to cancer.	Form of fæces may be altered; blood points to cancer.	Gradually increasing; may be spurious diarrhœa; no blood.
TENESMUS	Absent.	Often present.	Absent.
METEORISM	Not marked, unless acute attack.	Often marked.	Late; generally increases with obstruction.
TUMOR	Only in cancer, and then in 30 per cent.	Only in cancer, and then in 40 per cent.; may be felt in rectum.	Common and distinctive; most easily felt in cæcum ; little or no tenderness ; sometimes movable, and can be changed in shape.
COILS OF INTESTINE	Marked in proportion to emaciation.	Marked in proportion to emaciation.	Rarely seen.

N. B.—In any form of chronic obstruction, the symptoms of acute occlusion may suddenly supervene.]

We would here make special brief mention of only one frequent form of intestinal obstruction, on account of its practical importance. We mean that form which is caused by the accumulation of large masses of old fæces in the rectum. We sometimes find monstrous accumulations of fæces in the rectum, especially in old women who have previously suffered from habitual constipation, or in whom constipation is due to some other affection. Severe symptoms usually come on quite suddenly, after long-continued mild prodromal symptoms, and these severe symptoms are much like the picture of internal strangulation—severe, sometimes colicky, abdominal pain, great tenderness of the abdomen, which is usually swollen, marked general collapse, loss of strength, a small pulse, an outbreak of cold sweat, vomiting, etc. If we try to give an enema in such cases, very little fluid runs into the rectum. On introducing the finger, it usually strikes solidly on old, hard, fæcal masses above the sphincter, and there is often nothing left but to undertake the dirty task of removing at least a part of the scybala with our own hands. We may then succeed, by repeated enemata and by giving cathartics internally, in removing sometimes quite an incredible amount of accumulated fæces, and in obtaining thus a rapid recovery from the condition.

Treatment.—As soon as the dangerous signs of intestinal obstruction are recognized by the physician, he must decide in the first place whether the stenosis is not accessible to direct treatment. Hence we first examine in the most careful manner the external points for hernia. that we may not overlook a strangulated hernia. Then we make a digital examination of the rectum in order to decide whether the stenosis may not have its seat here, as from coprostasis, rectal tumors, or perceptible intussusception. Besides that, we of course examine the rest of the abdomen, as far as the patient's condition permits, in order by this, and by any facts in the history, to decide as to the form of the stenosis, from obliteration or compression.

Definite therapeutic measures sometimes follow from the conditions thus indicated. Strangulated external herniæ require operative treatment as taught by surgery. We may obtain aid, in some cases of stenosis from impaction, by the prudent use of cathartics. The treatment of fæcal impaction is of special importance. We have described the most frequent form of this in detail above. As has already been said, it is usually necessary to remove at least a part of the fæces with the fingers, or some instrument like dressing-forceps or a spoon. In the second place, we may use large enemata of pure water or soap-suds, which must often be repeated four or five times a day, until they have a satisfactory result. These are best given by a funnel and an œsophageal tube ("intestinal tube") introduced as high as possible into the intestine. Cathartics administered internally serve as aids, especially castor-oil and rhubarb.

In stenosis of the rectum from cicatrices and new growths we can also sometimes employ a local surgical treatment, like dilatation or scraping. The treatment of fæcal accumulations usually plays an important part here. Finally, the cases of ileo-cæcal invagination, in which the lower end of the invaginated ileum reaches the rectum, may yield to local treatment. We may try a partial replacement by a "sponge-sound" (an elastic œsophageal tube to the end of which a sponge is fastened). Blowing in air by the bellows was also recommended for this purpose by the old physicians. As a rule, however, we use here large enemata of warm water, which sometimes seem to exert a favorable mechanical action.

Very often we can not decide with certainty as to the anatomical cause and the seat of the obstruction at the bedside. In these cases nothing but a symptomatic treatment is left for the physician to employ. With constipation we usually try first cathartics, first the weaker, then the stronger, and finally, as a "last resort," reguline quicksilver, pure mercury in doses of five to ten ounces (grm. 150–300), which is sometimes claimed to act mechanically in "doubtful cases" by its weight. Except among some defenders of mercury, the present opinion among physicians tends far more to the belief that cathartics are usually of no service, but are often directly injurious by increasing the resistance. Hence we have at present gone over to the treatment of severe internal incarcerations with large doses of opium. Opium acts favorably on the patient's pain, the vomiting is diminished, and, by quieting the peristalsis, the danger of increasing the stenosis and tearing the intestine is also lessened. In fact, the opium treatment has some favorable results on record. Sometimes the first dejection appears during the administration of opium.

Since, therefore, there are many objections against the internal use of cathartics, we may try large enemata in those cases where the seat of the stenosis is not known to be in the large intestine. They must be given with caution, but persistently, and they must often be repeated; then they sometimes give good results, even in severe cases. In numerous instances of fæcal vomiting great benefit is also derived from methodical washing of the stomach as recommended by Kussmaul, Cohn, and others. Large amounts of feculent liquid are frequently removed through the tube from the stomach; and indeed it is easy to see that freeing the stomach of its accumulations may favor a more vigorous peristalsis. Even when the nature of the intestinal obstruction precludes definitive recovery, lavage usually gives no inconsiderable relief.

We need not enter into details as to the general treatment. It goes without saying that the patient's strength must be kept up as much as possible, and that in severe states of collapse all possible stimulants must be used, like hot, strong coffee, camphor, and ether. Local applications to the abdomen are usually ill borne on account of the tenderness, still we may try ice poultices or wet compresses. Opium is the best remedy for pain and vomiting, but it must often be replaced by subcutaneous injections of morphine. In cases of extreme gaseous distention the intestinal coils may be punctured with the needle of a subcutaneous syringe and the gas in part evacuated. This sometimes proves very beneficial.

Finally, it must be noted that in severe cases we are justified in proposing laparotomy. Sometimes the existing obstruction may be thus detected and removed. (For particulars, see text-books on surgery.)

[The safety with which laparotomy is now performed has stimulated the study of all affections on which the operation has any bearing. Internal strangulations and invaginations may be relieved, and the portion of intestine containing a non-cancerous stricture can be excised. An early operation offers much better chances, of course. In these days persons should not be allowed to die directly from intestinal occlusion without an attempt being made to restore the permeability of the canal by surgical means.]

CHAPTER XII.

INTESTINAL PARASITES.

(*Helminthiasis.*)

1. Tape-worms.

Natural History of the Tape-worm.—Three of the tape-worms (*cestodes*) which are found in the intestines have a clinical significance: the *tænia solium*, the *tænia mediocanellata*, and the *bothriocephalus latus*.

1. The *tænia solium* is, when fully developed, two or three metres long. Its head (Figs. 37 and 38) is about the size of that of a pin, and has four projecting cup-like suckers, and in front a beak with about twenty-six hooks. The top of the head is, as a rule, plainly pigmented. A small neck, about an inch long, is attached to the head, and then follow the single "joints" (*proglottides*) of the tape-worm, of which the youngest, lying near the head, are still very small and short. They gradually increase in length and breadth, and at about a metre from the head they have an approximately quadrilateral shape. The segments which lie farther down, and which have already reached puberty, have the form of pumpkin seeds, and are nine or ten millimetres long and six or seven wide. The matrix or uterus runs through the middle of each mature segment (see Fig. 39), and from it, on each side, go seven or eight side branches, which ramify like a tree. On one side, a little below the middle, lies the sexual orifice (Fig. 39, *a*). The male sexual organs consist of a number of little clear vesicles in the anterior portion of the segments. The thick-shelled eggs (Fig. 40, 3) develop in the uterus, and contain an embryo with six hooklets.

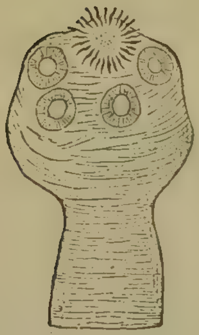


FIG. 37.—(From HELLER.) Head of tænia solium.

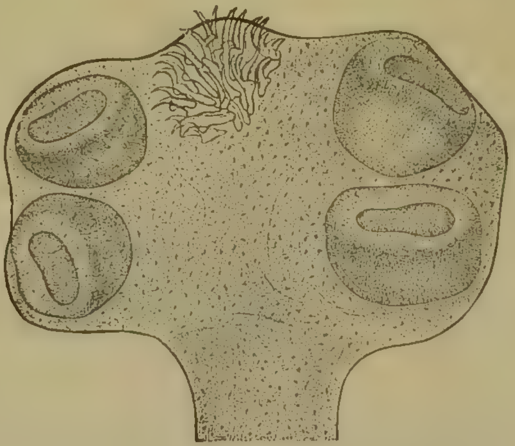


FIG. 38.—(From HELLER.) Head of cysticercus of the brain.

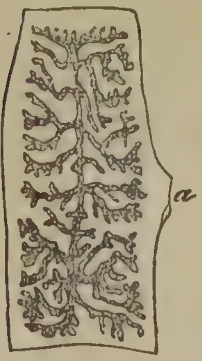


FIG. 39.—(From HELLER.) Tænia solium. Mature segment.

The tænia solium inhabits the small intestines of man. Its head clings to the mucous membrane so tightly, usually at some point in the upper third of the small intestine, that the neck is often torn off in trying to loosen the worm from the intestinal wall. The rest of the worm, which is in part in many coils, extends to the lower part of the ileum, but only exceptionally into the cæcum. From the lower end long chains, or single mature segments, are often detached, mix with the contents of the intestine, and are passed with the fæces, together with some of the eggs from the uterus.

The further development of the eggs of the tænia solium takes place in another "host," almost always in the hog. Hogs are infected by eating fæces, offal, etc., containing tænia eggs. The thick shell of the eggs is dissolved in the hog's stomach, and the free embryos pierce the walls of the stomach and intestines and travel with the blood-current, or through the tissues, into the different organs, especially into the muscles. Here they develop, in two or three months, into cysts something larger than a pea, from whose walls a newly developed tænia-head arises, a so-called *scolex* (nurse). These cysts are termed worm-cysts, measles, or *cysticerci cellulosæ.* They live from three to six years; then they die and become calcified. If a cysticercus gets into a man's stomach from his eating raw or imperfectly cooked ham or pork, a new and complete tænia sprouts from the scolex, which forms mature segments in three or four months.

We usually find only one tape-worm in a man, but several specimens have been seen at the same time in the same intestine. The length of a tape-worm's life is not certainly known, but it has happened that some persons have lodged the same tape-worm for ten or fifteen years.

Although the fully developed tænia solium is seen only in man, as we have said, the cysticercus cellulosæ has been found, in rare cases, in dogs, rats, and monkeys, as well as in hogs. It is a particularly important fact that the cysticercus cellulosæ itself may also occur, as such, in man. If tape-worms or mature segments get into a man's stomach in any way, probably by auto-infection, by the finger, etc., the embryos travel into other organs. Cysticerci are often found in men, singly or in groups, especially in the skin, the brain, the eye, and the muscles. There is a special form of cysticercus of the brain, in which we find a whole chain of cysts, like a cluster of grapes, but sterile, the so-called *cysticercus racemosus.*

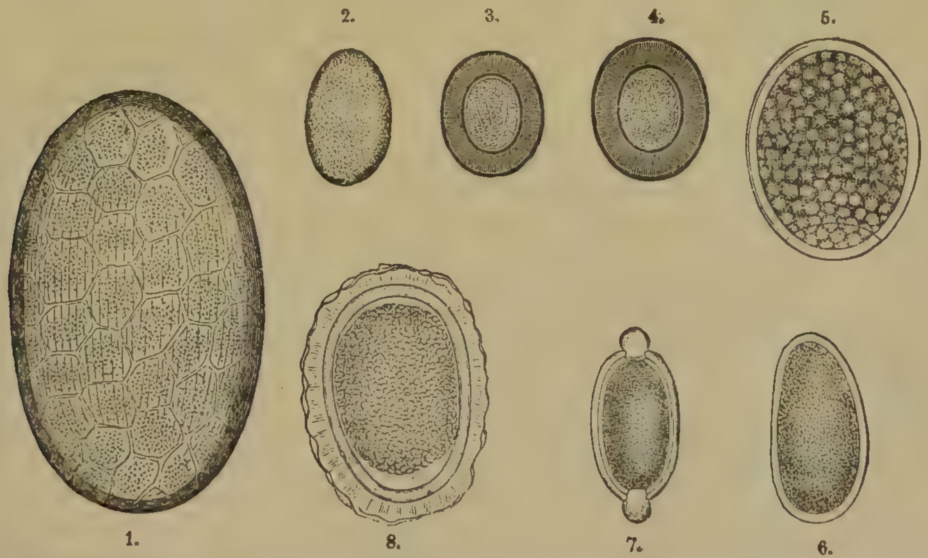


FIG. 40.—Comparative view of the eggs of some of the commoner intestinal parasites. 1. Egg of distoma hepaticum. 2 Distomum lanceolatum. 3. Tænia solium. 4. Tænia mediocanellata. 5. Bothriocephalus latus. 6. Oxyuris vermicularis. 7. Trichocephalus dispar. 8. Ascaris lumbricoides.

2. The *tænia mediocanellata*, or *tænia saginata* (from *saginare*, to fatten), is even more common than the tænia solium in many parts of Germany. It is longer than the tænia solium, being about three or four metres long, and its individual

joints are, on the whole, broader and thicker. The head (Fig. 41) has also four prominent cup-like suckers, but it has no crown of hooklets. The mature segments differ from the proglottides of tænia solium, in that the central uterus sends off many more (twenty to thirty) side branches, which divide dichotomously, and not like a tree. The sexual opening is also on the side (Fig. 42, *a*).

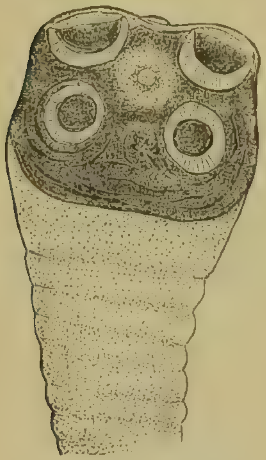


FIG. 41.—(From HELLER). Head of tænia mediocanellata.

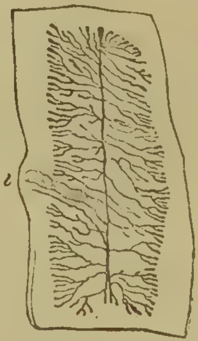
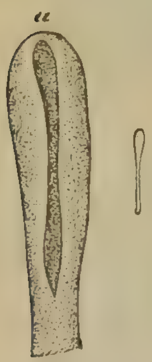
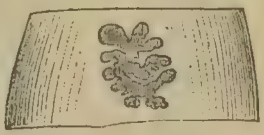


FIG. 42.—(From HELLER.) Tænia mediocanellata. Mature segment.

The life-history of the tænia mediocanellata is, on the whole, like that of the tænia solium. The tænia mediocanellata, however, throws off single mature segments much more frequently than the tænia solium. These segments are found in the fæces, and here they often exhibit a crawling motion. The cysticercus of tænia mediocanellata does not inhabit pork, but beef, so that the infection of man by this tape-worm comes from eating raw beef. In man the cysticercus of tænia mediocanellata, which is somewhat smaller than the cysticercus cellulosæ, has never yet been observed.

3. The *bothriocephalus latus* occurs in Holland, Switzerland (Geneva), Pomerania, East Prussia, Hamburg, and Russia (the German Baltic provinces). It has not yet been observed in middle Germany. It is the largest tape-worm; it may be six or eight metres long, and sometimes has over four thousand joints. The head of the bothriocephalus (Fig. 43) consists of a little club-shaped swelling, with two slit-like depressed suckers on the sides. A long, thread-like neck joins the head to the youngest segments. The full-grown segments (Fig. 44) are short, but are distinguished by their great breadth. The largest segments measure in length about three or four millimetres, and in breadth ten or twelve, but the last joints are longer and are not so broad, so that they have an approximately quadrilateral form. The uterus consists of a very tortuous canal in the center. The sexual orifice does not lie on one side, as in the tænia, but in the middle of the abdominal surface, nearer the anterior border of the segment than the posterior. The eggs (*vide supra*, Fig. 40, 5) are of an oval form, and have a hood-shaped lid at one end. They are to be found in almost every dejection of persons affected with a bothriocephalus. Single joints of the tape-worm are not passed with the stools, but portions of the worm, several feet long, come away from time to time, especially in the spring and autumn.





FIGS. 43 and 44.—(From HELLER.)
FIG. 43.—Head of bothriocephalus latus. *a*, Lateral view, enlarged. *b*. Natural size.
FIG. 44.—Bothriocephalus latus. Mature segment.

The noteworthy life-history of the bothriocephalus has been made perfectly clear by Braun, of Dorpat. The eggs develop only in fresh water. The embryo (Fig. 45), which is formed in them in a few months, and is provided with six hooklets and with vibrating cilia, is swallowed by fishes, especially by pike and eel-pouts, and develops in their muscles and internal organs into cysticerci. The infection of man with bothriocephalus comes from eating such fish containing cysticerci.

Symptoms and Diagnosis.—In many cases tape-worms are lodged in the intestines without causing any morbid symptoms. We can recognize their presence only by occasionally finding the joints in the dejections.

In other cases, however, tape-worms cause a list of disturbances which are often exaggerated by anxious, hypochondriacal, and nervous persons, but which ought not to be too little regarded. The symptoms are referred chiefly to the intestinal canal. Sometimes there is quite severe abdominal pain, which may assume a colicky character. The patient also frequently complains of irregularity of the bowels, and of occasional diarrhœa, which alternates with constipation. Many general symptoms are also added to those mentioned—loss of appetite, or at times marked voracity, general languor, disinclination to work, mental disturbance, depression, etc. We can see that, under such circumstances, the general health may suffer considerably.

We must also mention a number of symptoms which probably owe their origin to abnormal reflex processes. Among these we sometimes see marked salivation, tickling in the nose, dilatation of the pupils, palpitation, vomiting, headache (migraine), etc. In some cases even severe spasms, and choreic conditions, have been referred to the presence of tape-worms in the intestinal canal, but it is hard to decide how far such a supposed connection can really be regarded as justified.

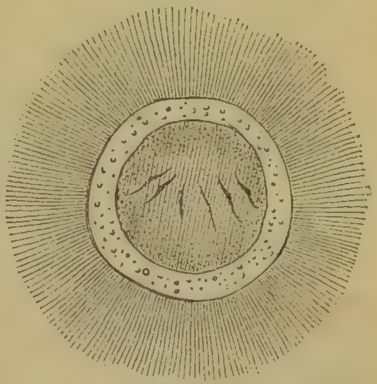


Fig. 45.—Embryo of bothriocephalus latus, with its ciliated coat. (Leuckart.)

Although many of the symptoms mentioned may arouse suspicion as to the presence of a tape-worm, the diagnosis can be made only by finding the joints or eggs of the tape-worm in the dejections. In many cases the patient himself brings some of the segments found by him in the dejections to the physician, but in judging of them a certain caution is always necessary, since shreds of mucus, remains of food, etc., are quite frequently presented to the physician, under the idea that they are segments of tape-worm. If possible, we should take pains to decide definitely, from the segments laid before us, the species of tape-worm, which is usually not a difficult task if we follow the anatomical description given above. If we spread out the pieces of tape-worm on a microscope slide, the thicker, fatter segments of the tænia mediocanellata, with its many-branched uterus, may usually be distinguished without difficulty from the more tender and more translucent segments of the tænia solium, with a smaller number of lateral branches to its sexual apparatus. The statement of many patients that single segments of tape-worm come from them at other times than when at stool, and that they find them on their underclothing, almost always points to the presence of a tænia mediocanellata in the intestine.

If we suspect a tape-worm, without having secured the certain evidence of segments in the dejections, it is a good plan to give the patient a mild cathartic, like castor-oil, or a dose of boiled pumpkin-seeds, since after this, if the intestine harbors a tape-worm, single portions of it almost always come away.

Treatment.—The "tape-worm cures," which are recommended in so great a number that we can by no means mention all of them here, but only the most important and the most serviceable, aim at killing or benumbing the worm, and then at removing it from the intestine *in toto* by cathartics.

We usually begin with a so-called "preparatory treatment." This is to cleanse the intestine, especially the large intestine, from old fæcal masses, in order to prepare as free a passage as possible for the worm. For this purpose we give the patient a mild laxative, or, better still, a large enema of cold water. We also forbid for a day or two the use of vegetables, black bread, etc., and prescribe instead a limited diet of white bread, some meat, milk, and coffee. It is a wide-spread practice to take during the preparatory treatment certain articles of food to "make the worm ill." Among these a salad of finely chopped and very salt herring with onions and garlic is especially recommended. A similar action is also ascribed to strawberries, cranberries, and bilberries. Hence, on the day, and especially on the afternoon, before treatment, we have the patient take a large amount of the articles of food mentioned, such as herring salad.

On the next morning, after everything has been prepared, after the bowels have moved the night before, etc., the patient takes no breakfast, or only some strong sweet *café noir*. Then he takes the special anthelmintic, and in one or two hours, if he feels a great pressure in the abdomen, he also takes a few spoonfuls of castor-oil or rhubarb.

The number of tænicides recommended is, as we have said, very great. At present the following are most in use:

The bark of pomegranate-root (*cortex radicis Punicæ granati*) is one of the most efficient remedies. We usually prescribe it in combination with the ethereal extract of male fern, in the following prescription:

℞ Granati radicis corticis................ ℥ iv–v (grm. 120–150);
Aquæ................................ O ij (grm. 1,000).
Macerate for twenty-four hours, and boil until it is reduced to ℥ v (grm. 150).
Add: Oleoresinæ filicis................ gr. lxxv (grm. 5).

The whole amount is to be taken in three or four doses as near together as possible. In order to obviate the bad taste of the remedy and to increase the action by introducing a larger amount at once, it has been recommended to introduce the whole amount of a still stronger decoction of pomegranate-root at once into the stomach by means of an œsophageal tube. As a rule, it is well to avoid this procedure.—To be recommended is also the tannate of pelletierine, prepared from the pomegranate-root. It is almost tasteless, and in doses of 8–25 grains (grm. 0·5–1·5) is said to be a very certain tænicide. Usually one obtains excellent results with male fern. Two to two and a half drachms (grm. 8–10) of the extractum filicis (P. G.) must be given [or one and a half to two fluidrachms of the oleoresin]. It may be put in gelatine capsules on account of its bad taste.

Another remedy, which has often proved successful, is kousso-flowers. We give three or four powders, each containing seventy-five grains (grm. 5) of powdered kousso-flowers, in white wine, giving a glass of wine containing one powder about every half-hour. Rosenthal's "kousso tablets" are more agreeable to take and are very good, but they are more expensive. Twenty of these, of fifteen grains each (grm. 1), may be taken without danger within an hour with *café noir* or lemonade. During the period of treatment the patient must lie as quiet as possible in order to avoid vomiting. Up to the present time we have not had sufficient experience of the koussine or kosseine, prepared from the alcoholic extract of kousso-leaves, which is said to be very efficient in doses of thirty to forty-five grains (grm. 2–3).

Of the other remedies we may mention kamala, one to three drachms (grm. 5–10) of the powder in wine or water, and oil of turpentine, one or two ounces (grm. 40–60) in two doses in milk—an efficient but rather dangerous remedy in these doses—and picro-nitrate of potassium. We may also prescribe male fern in powders of a drachm (grm. 4) each, taking three or four powders within an hour.

The treatment is to be regarded as absolutely successful only when we find the head of the tape-worm, as well as its joints, in the patient's dejections. We may best search for the head in the fæces by diluting the dejection with water, stirring it repeatedly, and pouring off the water. The tape-worm then remains at the bottom of the vessel.

Every tape-worm treatment is rather drastic, and hence it is well, after the treatment is over, to recommend the patient to be prudent in his diet, and to be careful about his digestive tract for some time. In persons who are very weak, or who have some other disease, we do not willingly undertake to remove a tape-worm without urgent reasons; but in people who are otherwise healthy it is always well to get rid of a tape-worm, even if it causes no severe symptoms. Of course only the tænia solium is attended with the serious danger of the cysticerci invading the brain (see diseases of the brain). The best time for undertaking a treatment is when joints or large pieces of the worm come away quite frequently of their own accord. We should never prescribe a treatment on the mere statements or suspicions of the patient. We must always convince ourselves with complete certainty of the presence of a tape-worm in the intestine.

We must finally mention that the only efficient prophylaxis against acquiring a tape-worm lies in entirely avoiding the use of raw or half-cooked beef or pork. The more widely spread the taking of raw meat is, as in Abyssinia, the more common are tape-worms in man. Certain callings, like those of cooks and butchers, are also especially exposed to infection.

2. **Round-worms.**

(*Ascaris lumbricoides.*)

Natural History.—Ascarides are pale-reddish, cylindrical worms, pointed at both ends, with the sexes in different individuals. The females are thirty or forty centimetres long, the males about twenty-five. At the cephalic end of the worm are found three lips furnished with fine teeth. The tail is straight in the females and curved in the males. In the female sexual organs (Fig. 46) sixty millions of eggs may develop, at a rough estimate. These eggs are often found in the fæces of people who have round-worms in their intestines (see Fig. 40, 8). They have a great capacity of resisting external influences, and a worm-like embryo develops in them in a few months. Their further fate, and the form and manner in which infection usually takes place in man, are not yet accurately known. It is probable the parasites are spread without any intermediate host by direct ingestion of the eggs which contain the embryos.

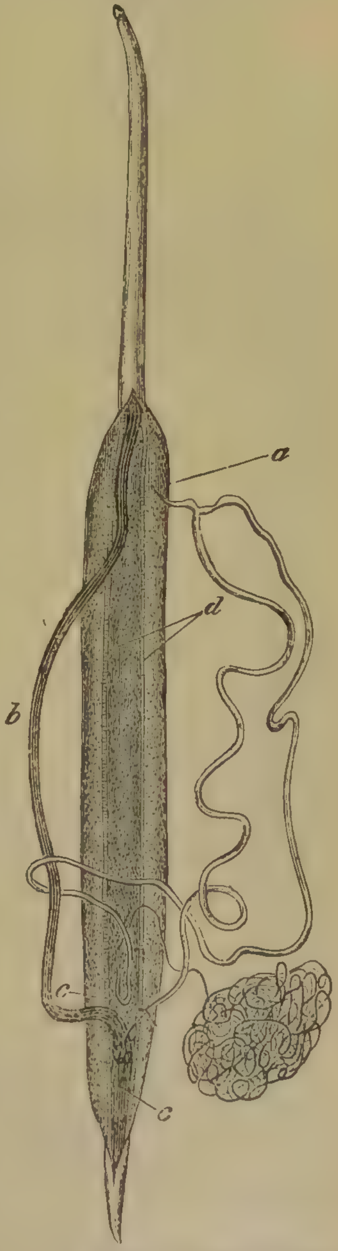


Fig. 46.—(From Heller.)—Ascaris lumbricoides. Female, 143 millimetres long. *a*. Vagina. *b*. Intestine. *c*. Boundary between the uterus and oviducts. *d*. Longitudinal bands. *e*. Coil of oviducts and ovaries.

The round-worms inhabit chiefly the small intestine. In severe vomiting they often reach the stomach and are vomited up. In individual cases they have been found in the bile-ducts, in the air-passages, and, after perforation of the intestine, in the abdominal cavity. The number of round-worms existing at the same time in the intestines may be very considerable. We find them most commonly in children and in adults from the lower classes. Round-worms have been repeatedly observed to crawl out of the anus, the mouth, or the nose of children during sleep.

The round-worm is also common in hogs and cattle as well as in man.

Symptoms.—In general, the round-worms are innocent parasites, which may exist in large numbers in the intestines without any bad results. Exceptionally they cause symptoms similar to those ascribed to tæniæ—abdominal pain, languor, itching of the nose, burning in the eyes, etc.—symptoms which are all ambiguous, and whose definite connection with the presence of round-worms it is hard to make out. The cases recorded in literature are quite numerous in which severe nervous symptoms have been caused by round worms and have disappeared after the removal of the parasites. However cautious we may be in accepting such statements, nevertheless their credibility can not be wholly denied. We would mention especially convulsions, epileptiform seizures, choreic and cataleptic conditions, contractures, and temporary mental disturbances, which are claimed to be excited by ascarides. Milder nervous attacks—like headache, vertigo, dilated pupils, and chills—are quite frequently seen in children with ascarides.

In some cases the presence of ascarides may excite much more severe symptoms by unfortunate accidents, as, for example, sudden suffocation from the entrance of a round-worm into the larynx. When a very large number of round-worms have been present in the intestine, severe symptoms of intestinal stenosis have been observed from their rolling together into a ball. If a round-worm crawls into the bile-ducts, it may give rise to jaundice, and even to the development of an abscess of the liver. In the abscesses of the anterior abdominal wall, usually termed "worm abscesses," the round-worms probably play a purely accidental part. We have to do in such cases with perityphlitic abscesses or with inflamed herniæ, which have perforated externally, by which the round-worms which are accidentally found in the intestines pass out, without having any causal relation to the abscess.

Treatment.—The oldest and most approved remedy against ascarides is worm-seed—santonica. This is best given in the form of an electuary—santonica, a drachm (grm. 5); jalap, fifteen grains (grm. 1); and syrup, an ounce (grm. 30), to be taken in three doses—in combination with a cathartic. Of late, worm-seed, on account of its bad taste, has been almost wholly replaced by santonin, which is derived from it. This is prescribed in one- or two-grain (grm. 0·05–0·10) powders, or still more frequently in the form of santonin troches ("worm-tablets"), which may be had of any apothecary. It is well to give santonin also in connection with a cathartic, like calomel. We let the patient take one or two doses of santonin in the morning for three days, and on the fourth we give a cathartic. Severe symptoms of poisoning—spasms—have been seen only occasionally from the careless use of it. Milder symptoms, like a yellowness of the urine and conjunctivæ, and xanthopsia, or seeing everything as yellow, are somewhat more frequent.

3. Oxyuris vermicularis.

(*Seat-worms. Pin-worms.*)

Natural History.—The oxyures are little round worms, the females ten or twelve millimetres long, the males only three or four (see Figs. 47 and 48). The eggs, when they reach the human stomach, develop very rapidly. The embryos, set free, collect in the small intestine and later in the cæcum, where they soon become mature. The impregnated female usually crawls down into the rectum, deposits her eggs there, and either crawls out of the anus herself, or, like the male, is evacuated with the fæces. The whole development of the oxyuris occupies about a fortnight. The total number of oxyures present in the intestine at the same time may be very considerable, so that "the whole mucous membrane of the large intestine is covered with them like fur."

FIG. 47.

Fig. 47. — Oxyuris vermicularis. Natural size. 1. Female. 2. Two males.

The infection by the eggs of the oxyuris probably takes place, as a rule, from one man to another, since the eggs stick to the hands (in scratching the anus), and are thus communicated to food, bread, fruit, etc. In children and dirty adults auto-infection may often be repeated in an analogous manner.

Symptoms and Treatment.—The oxyures found in the upper portions of the intestine and in the cæcum cause no symptoms whatever, but in the lower part of the rectum their presence causes local symptoms, especially a very severe feeling of itching and burning in the anus, which makes the child constantly scratch and dig with his fingers. This itching of the anus is most severe at night in bed. In girls the oxyures frequently travel into the vagina, by which an intense itching is also set up there, which sometimes leads to masturbation. In some cases in boys and men oxyures have been found to be the cause of abnormal sexual irritation.

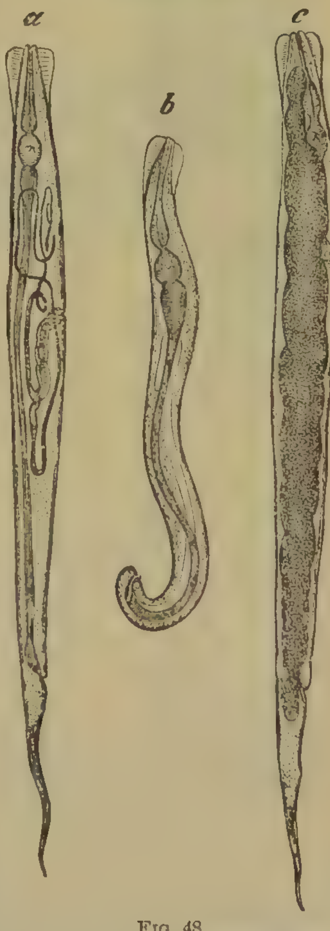


Fig. 48.

Fig. 48.—(From Heller.) Oxyuris vermicularis, enlarged. *a.* Mature female, not yet impregnated. *b.* Male. *c.* Female containing eggs.

The diagnosis of oxyures is not difficult. Our attention is called to the itching of the anus, and we look for worms. Single worms are easily found in the dejections, and often on the skin about the anus. The diagnosis is made more certain by finding the eggs (Fig. 40, 6) in the fæces under the microscope.

Treatment can remove the oxyures from the rectum with ease, but only with difficulty from the upper portions of the intestine, especially from the cæcum and the vermiform appendix. Santonin is generally used, but we must also prescribe large enemata of cold water and cathartics internally. Instead of ordinary water we may use soap-suds, vinegar-water, and, in severe cases, a weak solution of corrosive sublimate (1 to 10,000) in the enemata. The itching of the anus is relieved by rubbing on a little mercurial ointment.

[Enemas containing infusion of quassia, alum, eucalyptol, tannin, etc., are much in use. A plain enema should be given first, to unload the rectum and clean the membrane as far as possible, so that the anthelmintic may reach the worms when introduced.]

4. Anchylostomum duodenale.

(*Dochmius seu Strongylus duodenalis.*)

The *anchylostomum duodenale* is a worm first observed in upper Italy and in Egypt, which, singly or in large numbers, inhabits the upper portion of the small intestine, especially the duodenum, but also the jejunum and ileum. The male is six to ten millimetres long, the female ten to eighteen. At the cephalic end (Figs. 49, 50) is found a bell-shaped mouth-capsule, which is provided with two small teeth on its dorsal edge, and four larger curved teeth on its ventral edge. With this sucking and biting apparatus the worm fixes itself firmly, like a wet cup, on the intestinal mucous membrane, and is nourished by the blood which it sucks out. The places in the intestine to which an anchylostomum has fastened may be recognized in the cadaver as little ecchymoses. The worms sometimes bore completely into the inner part of the mucous coat.

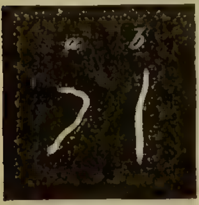


FIG. 49.

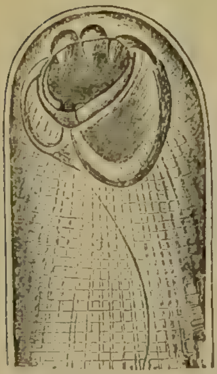


FIG. 50.

FIG. 49. — Anchylostomum duodenale. Natural size. *a* Male. *b*. Female.

FIG. 50.—(From HELLER.) Anchylostomum duodenale, enlarged. Head with bell-like mouth.

If the intestine harbors many anchylostoma, the small but constant loss of blood caused by them is not without influence on the organism. The symptoms of a severe anæmia gradually develop. Griesinger first made the discovery, in the year 1854, that the disease long known by the name of "Egyptian chlorosis" was caused by the anchylostomum duodenale. Since then confirmatory observations have been made in many parts of the tropics. Of late years the anchylostoma disease has become well known, because it occurred with great frequency among the Italian laborers employed in building the St. Gothard tunnel. In Germany, too, cases have repeatedly been detected, especially among brick-makers in the Rhineland and elsewhere, who work in wet clay-pits. The infection probably takes place chiefly from drinking impure, muddy water, in which the eggs of the anchylostomum are found.

The symptoms of the disease consist of a gradually increasing general anæmia, for which no special organic lesion can be made out objectively as a cause. The patient also suffers from very great general weakness and languor, constraint in breathing, palpitation, headache, œdema, etc. The changes in the blood (oligocythæmia, poikilocytosis) are precisely similar to those seen in pernicious anæmia. The disease may last for months, or even years, and it often ends fatally, if it be not recognized and treated in time.

Leichtenstern has made numerous and accurate observations with regard to the brickmakers of Cologne. He states that no symptoms are observed for three or four weeks after infection with the embryos of the anchylostomum. Some five or six weeks after infection, when the parasites become sexually mature and breed, there appear bloody diarrhœa, intestinal colic, and accompanying progressive anæmia. At this time there is probably more shifting about of the parasites in the intestine, while they later become more fixed. This explains why the disease evinces a more acute and severe character at first, and then takes on the form of a chronic anæmia, with great diminution or cessation of the bloody stools.

The diagnosis is easy if we only think of the possibility of anchylostoma. Many eggs may be found in the fæces, without great trouble; these are quite like the eggs of ascaris lumbricoides, only they are a little smaller. After using cathartics, the full-grown worms have often been found in large numbers in the patient's dejections.

If the trouble is correctly diagnosticated, treatment will usually give good results. We prescribe the same anthelmintics as for the other intestinal parasites, especially male fern in large doses, and also cathartics and enemata. In this way we often succeed in removing the parasites entirely from the intestinal canal, and thus bring about a complete cure, even in severe cases. Less certain in its effects is doliarina (made from ficus doliaria), of which the dose is one drachm (grm. 4·0) three times a day.

5. **Trichocephalus dispar.**

(*Whip-worm.*)

The trichocephalus dispar is a worm four or five centimetres long, whose anterior part is very thin, but whose posterior part is decidedly thick (Fig. 51).

The chief dwelling-place of the trichocephalus is the cæcum, where it is often found singly or in large numbers. It seems to have no clinical significance. At the most, if present in very large numbers, it may give rise to fæcal impaction, typhlitis, etc., but up to the present time no such occurrence has been actually demonstrated.

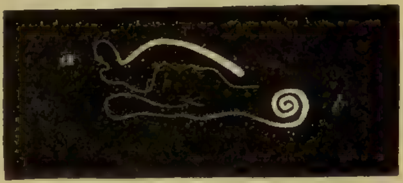


FIG. 51.—(From HELLER.) Trichocephalus dispar.

SECTION VI.

DISEASES OF THE PERITONEUM.

CHAPTER I.

ACUTE PERITONITIS.

Ætiology.—There are two ways by which inflammatory agents most frequently reach the peritoneum: one is from the gastro-intestinal tract, and the other—in women—is from the genitals.

All the diverse forms of ulceration which attack the digestive canal may involve the serous layer. In such a case an inflammation arises which is at first limited, but may under certain circumstances become more extensive. This inflammation may be regarded as analogous to that of the pleura in pneumonia; but the anatomy of the stomach and intestine is such that very often an ulcer in their walls ends in a complete perforation. If this occurs, the inflammatory germs contained in the primæ viæ at once escape into the peritoneal cavity and there excite an inflammation; which, from the specific character of its cause, is invariably purulent, and very frequently is at the same time septic or ichorous. The possibility of a peritonitis due to perforation, as a result of the various ulcerative processes of the stomach and intestines, has been frequently referred to in the previous sections of this work. Thus, it may occur in simple ulcer and in ulcerating cancer of the stomach; in typhoid, tubercular, or dysenteric ulceration of the intestine; in ulceration of the intestine above intestinal stenoses of many varieties; and in the small ulcers of the vermiform appendix due to the pressure of hard substances.

The female organs of generation are the other frequent source of peritonitis. In labor and premature delivery the genital tract is often directly infected. The infection may also occur, although much less frequently, at other times; for example, during menstruation. The various forms of inflammation which are thus set up, including endometritis, metritis, and parametritis, may in several different ways reach the peritoneum and excite peritonitis. A septic inflammation of the endometrium may involve the peritoneum by direct extension up the Fallopian tubes. In other cases it is through the lymph-vessels that a purulent metritis or parametritis spreads to the peritoneum. The larger parametritic abscesses may break into the peritoneal cavity. It is to be particularly noticed, however, that in many cases of septic puerperal peritonitis the uterus and its appendages are in a perfectly normal condition, having served merely as a gateway to the inflammatory agents without suffering any harm themselves.

Besides these two chief sources of peritonitis, numerous others are possible, although much less frequent.

Sometimes peritonitis is due to an extension of inflammation from other abdominal viscera. Hepatic abscess, suppurating hydatid cysts of the liver, ulcer of the biliary ducts, splenic abscess or infarction, purulent nephritis or pyelitis, abscess near the bladder or in the prostate, suppurating ovarian cysts, tubal pregnancy, psoas abscess, and Pott's disease—all these may produce peritonitis, either by direct extension or by perforation.

It is worthy of note that peritonitis may occur as a sequel of pleurisy. The pleural and peritoneal cavities are directly connected by the lymph-vessels of the diaphragm; and empyema as well as tubercular pleurisy (see next chapter) may spread to the peritoneum.

Penetrating wounds of the abdomen are a fruitful source of acute peritonitis. Surgical operations upon abdominal organs come under the same head. A large number of laparotomies proved fatal before Listerism was introduced, because the inflammatory germs thus admitted excited a diffuse septic peritonitis. Even tapping the abdomen for ascites may cause acute peritonitis if the trocar is not aseptic. Abdominal injuries, in which the walls are not penetrated, very rarely, if ever, give rise to peritonitis. One way in which they have been said to produce it is by exciting internal hæmorrhage. In the new-born, peritonitis sometimes results from infection through the navel.

Two diseases still remain to be mentioned, in the course of which acute peritonitis may be developed, although the occurrence is a rare one—acute articular rheumatism and nephritis. It may either be one of the symptoms of these diseases or an independent complication. We are somewhat in doubt as to how it arises. In acute articular rheumatism it must be regarded as analogous to the pleurisy and pericarditis which occur in the course of this disease, for they also involve serous membranes. We should likewise bear in mind the possibility that the inflammation may extend from the pleura to the peritoneum through the lymphatics. Acute peritonitis has now and then been observed in the various forms of nephritis, both acute and chronic, inclusive of amyloid disease. It usually proves fatal in these cases. Possibly the retention of urinary impurities in the blood plays some part in the development of this form of peritonitis.

Pathology.—Like the analogous inflammations of the pleura and pericardium, peritonitis is divided into different varieties according to the character of the inflammatory exudation. The nature of the exciting cause of most cases of peritonitis is such that by far the most frequent variety is the fibrino-purulent. If the process involves the entire peritoneum—that is, if there is a "diffuse general peritonitis"—we generally find upon opening the abdomen that the parietal layer of the peritoneum and the outer surface of the intestinal coils are distinctly reddened, from marked vascular injection. There may even be small ecchymoses here and there. The serous membrane is clouded, a result partly of desquamation of its endothelium, and partly of the more or less abundant fibrinous exudation which covers the peritoneum with a sheet of coagulated fibrin. Very often the coils of intestine have formed numerous adhesions with one another (compare pleuritic adhesions). In cases of brief duration these can still be easily broken up, but after a prolonged illness they are extremely firm. There is usually also some free, fluid, fibrino-purulent exudation in the abdominal cavity. Its amount varies greatly. Sometimes there is only a small quantity of opaque fluid in the dependent portions of the cavity; sometimes there are many quarts, causing great distention of the abdomen. The exudation seldom inclines to a sero-purulent character. It is usually predominantly purulent. Very often the purulent exudation undergoes decomposition into the offensive sanious fluid of septic peritonitis. This is particularly apt to occur when the disease originates from an intestinal perforation or from puerperal poisoning. The perforation through the walls of the intestine is sometimes so large as to admit considerable amounts of intestinal gases and fæces into the peritoneal cavity. It is also possible that the putrefaction of peritoneal exudations may generate offensive gases. In rare instances the exudation is hæmorrhagic; but most cases of hæmorrhagic peritonitis do not belong here, but come rather under the tubercular form (*vide infra*).

In severe and protracted cases of peritonitis the intestine is involved to a certain extent. There is a collateral inflammatory œdema of its walls, causing sometimes a considerable increase in thickness, while at the same time they may be non-resistant and easily torn. The weakness of the muscular layer of the intestine may amount to complete paralysis, and thus permit excessive intestinal tympanites, either diffuse or local.

Milder forms of general peritonitis with sero-fibrinous, or chiefly serous, exudation are relatively infrequent. Under this head would come certain apparently primary and usually chronic cases with favorable issue, and also the peritonitis which sometimes occurs as a sequel of an ascites which has existed for some time (see next chapter). Probably also in those rare cases where a peritonitis arising in the course of acute rheumatism has ended in recovery, the exudation has been sero-fibrinous.

We have spoken thus far of diffuse general peritonitis, but cases are not rarely seen of circumscribed or "encapsulated" peritonitis. Here, also, we have mild varieties with fibrinous exudations on the one hand, and on the other purulent inflammation. The milder inflammation is a result of the extension of the most varied forms of inflammation in neighboring organs. Thus, deep intestinal ulcers, for example, give rise to a mild circumscribed inflammation of the corresponding portion of the serous layer. A similar condition results from superficial splenic infarctions; from various hepatic diseases, when they reach the surface of the liver; and from numerous pathological conditions of the female genitals. In many of these cases the peritonitis takes a chronic course and leads to adhesions, and hence is called adhesive peritonitis.

Circumscribed purulent peritonitis has precisely the same ætiology as the general form, with this single difference, that firm adhesions are quickly formed around the spot whence the inflammation proceeds, limiting it and preventing it from involving the entire peritoneum. It occurs most frequently as a purulent perityphlitis (*q. v.*) consequent upon perforation of the vermiform appendix; and also as pelvic peritonitis, which is a possible sequel of most of the forms of puerperal inflammation to which the uterus and its appendages are liable. It may also follow perforative gastric or intestinal ulcer, hepatic abscess with perforation, and analogous affections. So-called sub-diaphragmatic abscess is a form of encapsulated purulent peritonitis.

Histologically considered, acute peritonitis is perfectly analogous to the inflammatory processes which attack other serous membranes. The endothelium becomes degenerated, and, for the most part, is cast off. There is an exudation from the blood-vessels of a fibrinous fluid, which is partly coagulable, and with this exudation round cells escape in greater or less abundance. In the further progress of the disease there is an inflammatory new growth of vascular connective tissue, which probably originates chiefly from the endothelium and the permanent tissue-cells, but, according to some, starts in part also from the wandering cells. The new formation of blood-vessels certainly seems to be chiefly due to budding from the capillaries of the serosa. Thus arise the adhesions of connective tissue and the false membranes found in chronic cases between the different coils of intestine. They lead in process of time to marked thickening and retraction of the omentum and mesentery (*peritonitis deformans*). Most cases of purulent peritonitis prove fatal in the early acute stage. If a case recovers, the exudation undergoes fatty degeneration, and its cellular constituents are thus disintegrated and then are absorbed.

The results of circumscribed purulent peritonitis are detailed in connection with the clinical history.

Clinical History.—1. *Acute General Peritonitis.*—The following description applies chiefly to the severe purulent form, the one by far most frequently met with. It occurs in most instances after perforation, in puerperal cases, and after external injuries, such as surgical operations. In most of these cases the peritonitis is a secondary disease, so that it must obviously be greatly modified in its general characteristics and behavior by the original trouble. In the first place, the onset is modified. Many cases of peritonitis due to perforation begin abruptly, the patient having been previously in perfect health. Thus, as already mentioned, the first indication of a gastric or duodenal ulcer may be given by perforation. Most cases of perforation of the vermiform appendix present equally sudden and unexpected symptoms.

There are many other cases where the symptoms of peritonitis supervene upon those of some grave disease already existing. For example, typhoid fever, intestinal tuberculosis or intestinal stenosis, may, by causing perforation, excite a peritonitis. Here the symptoms of this secondary disease may be more or less completely veiled by the other grave local and constitutional disturbances.

Again, an acute general peritonitis may, as we have already said, be the sequel to a local and circumscribed inflammation of the peritoneum. Thus, a purulent perityphlitis, or a purulent puerperal pelvic peritonitis, may finally become universal. In such unfortunate cases the change in symptoms is often gradual, and is not clearly pronounced.

We have now indicated certain variations from the general course of the disease; but, with these exceptions, almost every case of acute general peritonitis, whatever its ætiology, presents clinical symptoms which are so characteristic and typical that a general description of the disease will be both easy and advantageous.

The symptoms of acute peritonitis form two groups, the local and the constitutional. The latter are the result of the local disturbance acting upon the general condition of the patient.

Of the local symptoms, pain deserves to be named first. It is usually the earliest symptom; and, as the disease progresses, it is generally the excruciating abdominal pain which attracts most attention. The localization of the pain in the beginning of the illness may be of diagnostic value in doubtful cases, if such as to indicate the possible starting-point of the inflammation, for example, the vermiform appendix or a gastric ulcer. Later the pain extends over the whole abdomen. As a rule, there are brief remissions followed by fresh exacerbations. The pain is aggravated by voluntary movements, by deep inspirations, and probably by intestinal peristalsis. The abdominal tenderness is often extreme in peritonitis, and is very characteristic. The gentlest palpation is torture, and often the slightest pressure of the bed-clothes is almost unbearable. Frequently the greatest tenderness is in the umbilical region.

Acute peritonitis seldom exists without pain. The exceptions to this rule are chiefly seen in patients who are extremely prostrated, and whose sensibility and intelligence are much impaired. Here the peritonitis itself may escape notice.

Physical examination of the abdomen greatly aids the diagnosis in many ways.

As a rule, the abdomen is distended. This is an early symptom, and gradually becomes more and more pronounced. It is due mainly to the intestinal tympanites, which we have already mentioned, and which sometimes becomes very great if the muscular fibers of the intestine are paralyzed. In the later stages the liquid effusion into the peritoneal cavity of course contributes to the prominence of the abdomen, but even then the distention is seldom so uniform or so broad as in ascites. In peritonitis, coils of distended intestine can often be recognized by their characteristic contour through the abdominal wall. In general, if the abdominal wall is yielding and thin, the peritonitic distention will be greater, so that it is most marked in puerperal cases, where the preceding pregnancy has rendered the walls lax. In a person with powerful muscles and tense abdominal walls the convexity of the abdomen is seldom great. In some cases there is no convexity whatever. The walls may be as hard as a board, and the abdomen flat or slightly concave. In such cases diagnosis may be difficult. Sometimes, again, the original retraction of the abdominal walls is succeeded by more or less distention of the abdomen.

Percussion over the distended intestinal coils yields a resonant and usually tympanitic sound. It is not till a considerable amount of liquid effusion has collected that there is dullness, most marked in the dependent portions of the abdomen. If there is much tympanites, however, quite a large effusion may exist without being detected on percussion. Usually there is too much pain to permit a careful examination of the change of dullness consequent upon change of decubitus. In general, the numerous adhesions between the separate coils of intestine not infrequently interfere with the free motion of the peritonitic exudations.

Percussion not only gives information about the existence of a liquid, purulent effusion, but is also of value in determining the level of the diaphragm, as affected by abnormal abdominal distention. The upper limit of hepatic dullness is raised to the fifth or even the fourth rib. The heart is also pushed up. There is a tympanitic resonance above the margin of the ribs on the right side. The area of hepatic dullness is not only displaced upward, but is also evidently diminished. This is due in part to coils of distended intestine overlapping the anterior edge of the liver, and in part to the organ being tilted upward in such a way that its area of contact with the anterior wall of the body is less than normal. Various authors formerly laid great stress upon the total disappearance of hepatic dullness, regarding it as a sure sign that gas has escaped from the intestine into the abdominal cavity. The inference is not always correct. The liver may be displaced backward by coils of intestine, and hepatic dullness be thus abolished, although there is no air free in the peritoneal cavity.

If there is a considerable effusion, it is possible, as in ascites (*q. v.*), to get a sensation of fluctuation by gentle, quick palpation.

As a rule, auscultation of the abdomen does not throw much light on a case of peritonitis. In the distended coils of intestine we not infrequently hear all sorts of gurgling and splashing sounds. Sometimes we hear a peritonitic friction-sound, coincident with the movements of respiration and due to the rubbing against each other of two rough surfaces under the impulse of the diaphragm. In particular this friction-sound has been repeatedly heard over the liver.

In almost every severe case of peritonitis there are gastro-intestinal symptoms.

As to the stomach, vomiting is the most frequent and important symptom. Vomiting is often seen early in the disease, and recurs frequently as the illness progresses. It sometimes is spontaneous, and sometimes follows the ingestion of food. If spontaneous, the vomitus consists of watery mucus, usually of a greenish tinge. Why vomiting is so prominent in peritonitis we do not know absolutely. Apparently it is in part a reflex action, excited by the inflammation of the serous membrane. Possibly the external pressure of the exudation also affects the stomach. It must be added that vomiting may be absent in acute peritonitis. This is seen when the patient is comatose, and sometimes also when the peritonitis has developed upon perforation of a gastric ulcer, because the contents of the stomach are thus emptied out through the hole in its walls. The vomiting usually is accompanied by frequent eructations.

Of the intestinal symptoms, the reader has already become acquainted with the tympanites, and also with the fact that it is due mainly to a paresis of the muscular fibers of the intestine. This same muscular weakness furnishes an obvious reason for the persistent constipation usually observed in peritonitis; but we may have diarrhœa instead, from increased peristalsis and secondary intestinal catarrh.

The pushing up of the diaphragm has a noteworthy effect upon the thoracic organs. The lower lobes of the lungs are compressed, so that considerable dyspnœa results. The heart is likewise crowded upward, so that the apex-beat is usually to be felt in the fourth intercostal space.

Every case of acute peritonitis that is at all extensive has marked constitutional effects. These are in part the result of the wakefulness due to pain, and the restlessness and fever. But in all probability there are also decided reflex inhibitory influences, originating in the irritation of the peritoneal nerves and affecting chiefly the heart, just as Goltz in his well-known experiment killed a frog by blows upon the abdomen. There is no other disease, except internal strangulated hernia—and the effect of that is perfectly analogous—which produces general collapse so quickly as does peritonitis. The countenance is rapidly altered, the cheeks fall in, and the eyes become hollow. The nose grows sharp and cool, the lips dry and cyanotic. The skin of the extremities is also cool and bluish, as a result of impaired circulation. The patient is extremely feeble. The chief cause of all these symptoms is the excessive weakness of the heart. The peritonitis has hardly begun before we find the pulse small and soft. In many severe cases the pulse finally becomes almost imperceptible. At the same time the pulse-rate increases, as is usual in collapse from any cause, so that 120 to 140 beats per minute is not an exceptional rapidity.

The temperature varies greatly in different cases. It may be high in the rectum, although the skin feels cool. Still, very high fever is not usual; and there are often considerable remissions. We even frequently observe the subnormal temperature of collapse. The number of respirations per minute is usually 30 to 40. This increased rate is due not only to the compression of the lower lobes of the lungs, but also to the pain caused by full inspirations and to the impeded circulation.

The intellect remains in most cases almost unimpaired to the end. There may exceptionally be mild delirium, or an approach to stupor, toward the close.

The course of acute general peritonitis in the great majority of cases is unfavorable. With the appearance of the grave symptoms just depicted the prognosis becomes almost hopeless. The course of the disease is also comparatively rapid. Marked variations in the intensity of the symptoms are infrequent. The grave local and constitutional symptoms persist, and, as a rule, the patient dies at the end of a few (two to six) days. Still, it is not well to make general dogmatic statements as to the clinical history, for the ætiology of each individual case impresses upon it individual characteristics. A peritonitis resulting from gastric or intestinal perforation is usually quickly fatal. The same is true of almost all cases of puerperal septic peritonitis. In a few cases, however, the inflammation is limited, by the encapsulation of the exudation. These may finally end in recovery through perforation of the abdominal walls or perforation into the intestinal canal. Now and then an acute general peritonitis may assume a chronic form. The effusion is mostly reabsorbed, and the newly-formed adhesions and false membranes contract into firm bands of connective tissue. The liver, spleen, and other abdominal viscera acquire a tough coating of connective tissue. The omentum and mesentery are shortened and thickened. Indeed, the omentum may roll itself almost completely up. Although the clinical symptoms become less severe, weakness usually persists, with gradual exhaustion and death. Often the intestine is so bent or pinched as to give rise to grave symptoms from stenosis.

Recovery from acute general peritonitis is very exceptioal. If seen, it is usually in mild cases, such as sometimes occur after menstruation, abortion, or labor. Peritonitis as a complication of acute articular rheumatism is a very rare event. Its termination is generally favorable. In all cases of this kind the inflammation is probably not purulent, but sero-fibrinous.

2. *Acute Circumscribed Peritonitis.*—The local symptoms of this are essentially the same as we have just ascribed to the general form; except that, a smaller extent of tissue being involved, they are correspondingly limited. The pain and tenderness are confined mainly to one region, but its boundaries are never sharply defined. On palpation of this region, we find an increased resistance which is sometimes almost like that produced by a tumor. If there is an encapsulated effusion, we may detect fluctuation, particularly if the abscess is going to point outward. On percussion over the affected spot, there is either dullness or a muffled tympanitic resonance.

The constitutional symptoms are likewise those of general peritonitis, only usually less severe. Reflex vomiting does occur, but is seldom so persistent as in the diffuse inflammation. The physical weakness and symptoms of collapse are decided, but do not usually become extreme. There is generally an irregular fever, which may now and then assume an intermittent, pyæmic character. Most cases run a chronic course. If the illness be very much prolonged, death may finally ensue from general debility. Recovery is possible if the pus can be let out. This may be accomplished either by the surgeon or by Nature. Spontaneous discharge of the abscess may take place through the abdominal walls, into the intestine, or even, in rare instances, through the pleura into the lungs. But if the pus finds its way into the general peritoneal cavity, the peritonitis becomes diffuse and causes death.

To describe in detail each separate variety of circumscribed peritonitis would occupy too much space, and would also lead to useless repetitions. We have already spoken at some length of one especially important form—namely, perityphlitis. Perimetritis and pelvic peritonitis are chiefly puerperal affections, and are fully discussed by writers on gynæcology.

Abscesses which are very deeply situated—as, for instance, behind the stomach or in front of the spinal column—may prove very difficult to diagnosticate, being so far out of reach. Sub-diaphragmatic abscesses containing air deserve a brief mention (*pyo-pneumothorax subphrenicus*). They are sometimes observed as a result of perforation of the stomach or transverse colon. Lying between the liver and the diaphragm, they crowd the latter upward, and are liable to be mistaken for pyo-pneumothorax. Sub-diaphragmatic abscesses containing no air also occur, originating in the liver or spleen. Finally, there is a rare form of circumscribed purulent peritonitis to be noted, to which children seem especially exposed. It declares its presence by a painful fluctuating tumor above the left groin, which usually points into the rectum and ends in recovery.

Diagnosis.—The diagnosis of peritonitis is in many cases an easy matter, when we have the characteristic symptoms of tenderness and tympanites, vomiting, and collapse. Often the starting-point of the inflammation is equally obvious, in cases of secondary peritonitis supervening upon some disease which we have already clearly recognized, such as typhoid fever, gastric ulcer, or puerperal diseases. But where the peritonitis is apparently primary, we must inquire carefully into the previous history and the earliest symptoms of the attack, in order to form even a surmise as to ætiology.

The diagnosis is sometimes greatly obscured by the fact that under certain circumstances very similar symptoms may be excited by other disorders affecting the intestines. Thus, in typhoid fever there may be great tympanites and grave constitutional symptoms, with abdominal pain, so that peritonitis may be diagnosticated, while the autopsy, if there be one, discloses no signs of it. Deep ulcers of the intestine, however produced, may give rise to so great abdominal tenderness as likewise to simulate peritonitis. The grave symptoms of acute intestinal obstruction are often of such a character that it is impossible to determine whether the intestinal disorder has already induced a peritonitis or not. On the other hand, we have mentioned before that peritonitis attended by stupor and general depression may hide itself from the keenest eye, because the abdominal distention, tenderness, and other chief symptoms are absent.

It is not always easy to diagnosticate circumscribed peritonitis, even where the trouble is not deep-seated and therefore inaccessible. It is not infrequently mistaken for a new growth. In doubtful cases, a careful use of the aspirating needle is the more necessary, because the presence of an encapsulated peritonitic exudation in almost all cases demands operative interference. As to ætiology, it should be added that localized suppurative peritonitis, and less frequently chronic diffuse deforming peritonitis, may be due to actinomycosis (*vide* p. 274).

It is well to remember that a pregnant uterus and a distended and therefore painful bladder have each repeatedly been mistaken for peritonitis!

Treatment.—Although severe cases are generally almost hopeless, yet we must try to meet the symptomatic indications, and must do all in our power to promote a limitation of the process, if it be possible.

External counter-irritants or "revulsants" are seldom of much use. Painting with tincture of iodine and mercurial inunctions seem so utterly purposeless that they may be discarded. The local abstraction of blood can not be employed in an extensive peritonitis with constitutional prostration. It is only in a circumscribed peritonitis which is very painful, and where the general condition of the patient remains comparatively favorable, that local bleeding is to be considered. Under these circumstances, the application of eight to fifteen leeches sometimes causes decided abatement of the pain. The local application of ice to the abdomen is universally in vogue. It usually moderates the pain, and it may also have a beneficial influence in quieting peristalsis. Still, some patients can not bear ice, and sometimes hot cloths and poultices give great relief.

Of all internal remedies, there is but one of great value, namely, opium. This in large doses (a grain—grm. 0·05—of extract of opium every hour) almost always proves beneficial. It moderates both the pain and the vomiting or eructations; and also, by diminishing the peristaltic movements of the intestine, opium contributes in another way to assuage the suffering and possibly to limit the spread of the inflammation. Experience shows that almost all patients bear even very large doses of opium remarkably well in peritonitis. Perhaps this is because the drug is only slowly absorbed. To substitute injections of morphine for the opium is wise only in cases where we wish to produce narcosis as rapidly as possible, or where the vomiting does not prove amenable to the ordinary treatment.

Sometimes particular symptoms demand special attention. For vomiting we may employ, besides opium, bits of ice, or small quantities of "sherbet." If tympanites is excessive, we may try to remove some of the gas through a rectal tube passed as high up as possible. Many physicians also puncture the distended intestinal coils with a fine trocar. Collapse and cardiac failure require the exhibition of stimulants, such as champagne or other alcoholic liquors, or doses of ether or camphor given subcutaneously. It is generally very difficult to nourish the patient. As a rule, small quantities of ice-cold milk are the best of anything, as food.

The treatment of circumscribed peritonitis should conform in general to the above indications. In suitable cases operative interference may be of great value; but upon this point we refer to works on surgery.

CHAPTER II.

CHRONIC PERITONITIS. TUBERCULAR PERITONITIS.

Ætiology.—Chronic peritonitis, not tubercular, is a rather rare disease. It is found most frequently in post-mortem examinations of patients who have had for a long while ascites due to venous stasis—for example, in chronic cardiac or hepatic cases. The chronic peritonitis, however, is not, as a rule, the direct result of the passive hyperæmia in such cases, but is, as already hinted, due to the puncturing of the abdomen during life for the removal of the ascitic fluid. Exceptionally, a chronic peritonitis occurs as a sequel to some severe intestinal disorder, such as ulceration. Thus, chronic peritonitis is sometimes observed to follow typhoid fever.

Chronic peritonitis may furthermore be the result of an acute peritonitis. The latter seldom terminates in this way, but still it may, when rather mild and not quickly fatal. The encapsulated exudations of peritonitis usually persist a long while, as was implied in the preceding chapter.

In a few instances we can find no satisfactory cause for chronic peritonitis. It is sometimes ascribed to an injury. Alcoholic excesses are also said to predispose to the disease. Many of the apparently spontaneous cases, however, finally turn out to be tubercular.

The tubercular is the most frequent form of chronic peritonitis. It is often merely a part of the tuberculosis of serous membranes in general (*vide* pages 261 and 327), of which mention has been already repeatedly made. In these cases it is usually due to a conveyance of the process from the pleura through the diaphragm. Another way in which tubercular peritonitis may arise is by infection from neighboring tubercular organs. Tubercular intestinal ulcers are among the chief causes of this kind, the ulcer extending to the peritoneum; or the peritonitis may be excited by tubercular retroperitoneal or mesenteric lymph-glands. In women a tubercular peritonitis may be developed in consequence of tuberculosis of the genital organs. A tuberculosis of the uterus sometimes affects the Fallopian tubes by direct extension, and thence the virus enters the abdominal cavity and excites its specific inflammation. In conclusion, we have to mention that, in general miliary tuberculosis, the peritoneum also may be the seat of numerous tubercles. although these do not as a rule give rise to important symptoms.

Pathology.—After well-marked cases of chronic peritonitis, the peritoneum is usually found to be considerably thickened. The intestinal coils are joined to one another and to the neighboring organs by numerous and extensive adhesions. It is often a hard matter to disentangle the confused mass into which the intestines have been rolled. Sometimes the liver and spleen are covered by firm, tough capsules. The omentum and mesentery are much shrunken; hence the name *peritonitis deformans.* The mesentery may indeed be transformed into a single thick cord. As a rule, there is little liquid effusion, and perhaps none. In simple chronic peritonitis, such fluid as may be present is usually a cloudy serum, pus being seldom seen.

The milder forms of simple chronic peritonitis occur oftenest, as we have said, in cases of ascites due to venous stasis, after repeated tappings. It is frequently possible to detect the points where the trocar has pierced the internal layer of the abdominal wall, by adhesions, minute hæmorrhages, or other lesions. The false membranes which exist in these cases are often very numerous, but they are usually not dense and are easily separable. The serous liquid found in the abdominal cavity is partly a transudation, but contains clumps of fibrine in more or less abundance. In rare instances a peculiar form of chronic peritonitis has been observed as a sequel to punctures for ascites, called by Friedreich "chronic hæmorrhagic peritonitis with hæmatoma." In it almost the entire peritoneum is covered by a newly formed membrane permeated with large ecchymoses.

Tubercular disease of the peritoneum may be divided into two forms: tuberculosis of the peritoneum, which may be acute or chronic; and tubercular peritonitis, which is usually chronic. In tuberculosis the peritoneum is covered with numerous tubercular nodules, varying in size from a millet-seed up to a pea; but there is not much coincident inflammatory change. In genuine tubercular peritonitis, on the other hand, the inflammatory changes above described are well marked, while sometimes it requires a microscopic examination to demonstrate the tubercular nature of the inflammation, by the detection of tubercles and cheesy degeneration in the newly formed tissue. Tubercular peritonitis is usually rather chronic, so that the adhesions are numerous and strong. The amount of liquid effusion varies, being sometimes considerable and sometimes scanty. Just as in tubercular pleurisy, it is not rare for the exudation to be bloody. Tubercular peritonitis is quite often accompanied by hepatic cirrhosis (*q. v.*).

Clinical History.—Diagnosis.—If an acute peritonitis becomes chronic, the violent symptoms gradually abate, while another group of symptoms takes their place. In other cases the chronic disease develops gradually and insidiously.

The sensitiveness of the abdomen is never so extreme as in the acute inflammation. Sometimes, to be sure, the patient complains of dull pains and a sense of abdominal oppression, but quite often the pain is either constantly or at times insignificant. On physical examination, we usually find moderate distention of the abdomen. Frequently this is not perfectly uniform, certain coils of intestine being especially prominent. Occasionally there is no abdominal distention whatever, the belly is flat or concave, and the walls are tense and unyielding.

In many instances palpation furnishes very characteristic signs; for sometimes the thickening of the omentum and the numerous fibrous inter-intestinal bands above described can be felt through the abdominal walls as peculiarly resistant masses or uneven prominences. Indeed, if the omentum is rolled up, it may closely simulate a new growth. Not infrequently, particularly in tubercular peritonitis, the liver is enlarged so that its lower edge can be felt. But in other cases of chronic peritonitis there are no changes discoverable by palpation; or they may be concealed by an effusion or by the tenseness of the abdominal walls. A large exudation can be demonstrated by the great distention, or by its causing fluctuation, or by the signs yielded on percussion. In general, uncomplicated cases do not give rise to great accumulations of fluid. Such accumulations are almost invariably present when tubercular peritonitis and cirrhosis of the liver are combined; and here there are usually also passive congestion and enlargement of the spleen. It has been already stated that the distortions and flexions which the intestines may undergo in chronic peritonitis may result in obstruction. In the same way the duodenum or the ductus choledochus may be so occluded as to occasion persistent jaundice.

The objective signs of both the simple and tubercular forms of chronic peritonitis have been embraced in one description, because the abdominal signs of the two are identical. To differentiate between them, other factors must be considered. We regard the patient's constitution and general appearance, and inquire into his family history, or discover if there are other ætiological factors, such as previous tubercular disease. A careful thoracic examination is extremely important. If we find the signs of coincident pulmonary tuberculosis, and particularly of pleurisy, then it is almost indubitable that the peritonitis is tubercular. The character of the exudation is also of some importance, for if hæmorrhagic, as already stated, the peritonitis is probably tubercular. Tubercle bacilli are not usually present in the exudation of tubercular peritonitis.

To diagnosticate simple tuberculosis of the peritoneum, when not attended by marked inflammatory changes, is generally a difficult matter. Often it is absolutely impossible. Frequently there is no abdominal pain or tenderness whatever. The abdomen is usually but moderately distended, as a result of the effusion present.

Particular notice should be given to the chronic peritonitis of children. The occurrence of ascites in children between the ages of two and ten years has been observed repeatedly, both by other authors and by ourselves. The ascites, which may be quite considerable, can not be traced to any cause, and after a few months completely disappears. The child during this time is usually rather pale and languid, but not much emaciated, nor does he suffer great local discomfort. Since the cases often recover, their pathological anatomy remains obscure. Probably they are a mild form of simple chronic peritonitis. Still, of course, there may be other causes for the ascites, such as hereditary syphilitic disease of the liver.

In children, tubercular peritonitis plays an important part in general tuberculosis of the abdominal organs, a condition known as *tabes mesenterica*. In these cases the tuberculosis probably originates, as we have already said, in the intestine, so that usually we find the intestine, peritoneum, liver, and abdominal lymph-glands all simultaneously involved. The clinical symptoms are often mainly due to the peritonitis. The abdomen is distended and painful, and there is an effusion. Often there is also obstinate diarrhœa, as a result of tubercular intestinal ulcers, with persistent fever of an intermittent character, emaciation, and anæmia. The tubercular process may eventually involve the lungs, pleura, meninges, and other organs, or it may never extend beyond the abdomen.

As to the course of chronic peritonitis we have little to say. The simple chronic peritonitis may terminate in recovery, although on account of other co-existing lesions this event is rare, except in the special form which children present. Many cases of tubercular peritonitis prove fatal in a few months or weeks. In some instances, however, chronic tubercular peritonitis has a favorable issue, or at least there is very great abatement of all symptoms. This is particularly apt to be the case in what is called primary tuberculosis of the serous membranes in general (*vide supra*). If, in this disease, there is no simultaneous tuberculosis of the lungs, intestines, or other organs, then the final reabsorption of the exudation is possible, just as in tubercular pleurisy. It must be confessed that often the recovery is not permanent, for the tubercles may appear later in some other part of the body.

Treatment.—The means by which we can exercise a favorable influence upon the course of chronic peritonitis are scanty. Attention to nourishment and hygienic surroundings is very important; but, beyond this, treatment is mainly symptomatic. The chief local applications are poultices or fomentations, perseveringly employed. There is seldom such persistent and severe pain as to demand opiates, but they may be required for the diarrhœa which is apt to occur. Or, on the other hand, enemata and mild laxatives may be indicated. Among special remedies, the preparations of iodine should be mentioned; iodide of potassium and syrup of the iodide of iron sometimes are of apparent benefit. Arsenic may also be tried. Iron and syrup of the iodide of iron are also employed in the chronic peritonitis of children.

[Of recent years laparotomy has been repeatedly practiced in tubercular peritonitis, and has been held by some to be distinctly curative. In the opinion of the editor, what surgery has done for us in this affection is rather to demonstrate the curability of many cases than to work their cure. Formerly, if the diagnosis of tubercular peritonitis was made and the patient recovered, the very fact of recovery was held to invalidate the diagnosis. But the surgeon's knife has laid bare the miliary tubercles to the eye, given an exit to the serous exudation, and recovery has ensued. But we see other cases get well after simple tapping, and we see still others which present just as good a clinical picture of the affection and recover without interference of any kind. Such recovery may be temporary, tuberculosis breaking out afresh in the peritoneum or elsewhere, or it may be permanent. If, in spite of rest and appropriate general treatment, considerable effusion persists, aspiration or siphonage should be practiced. Laparotomy should probably be reserved for those rather rare cases which seem to be rapidly getting worse, and those in which the fluid reaccumulates after one or more tappings.

In expressing the belief that tubercular peritonitis recovers, the editor does not lose sight of the fact that multiple fibrous nodules occur disseminated over the peritoneum, indistinguishable by the unaided eye from tubercles.]

CHAPTER III.

ASCITES.

(*Hydroperitoneum.*)

THE name ascites is given to a collection of transuded serum in the abdominal cavity, due to venous stasis. The peritoneal veins belong to the portal system, so that among the diseases which lead to ascites those which impede the portal circulation are chief. As we shall see in the next section, ascites is, therefore, of frequent occurrence in cirrhosis of the liver, syphilitic disease of the liver, compression of the portal vein by tumors, thrombosis of the portal vein, and similar disorders. Ascites is also frequently present as one of the dropsical symptoms in general circulatory disturbances, such as cardiac disease or pulmonary emphysema, and in the various acute and chronic renal affections.

The clinical significance of ascites is due partly to the local discomfort occasioned by the presence of any considerable amount of fluid within the abdominal cavity. Small quantities of serum are often unnoticed by the patient; but, where many quarts (fifteen to twenty, or even more) of transudation exist, the abdominal walls become greatly distended, and the patient has a very troublesome feeling of pressure, weight, and tension. What is of still greater importance is the crowding upward of the diaphragm. Respiration is thereby not a little impeded. If the ascites is great, the lower lobes of the lungs are so compressed that a considerable degree of atelectasis is produced.

To demonstrate ascites by physical examination is possible only when a considerable accumulation exists. Then the belly is prominent, its walls are tense and shining, and, the base of the thorax being gradually distended by the pressure of the liquid, the lower part of the thorax seems much broader than the upper. Distended veins are usually visible through the skin of the abdomen, like blue lines, here and there. As soon as the abdominal tension has attained a certain degree, fluctuation can be perceived, by laying both hands upon the abdomen and imparting gentle but quick impulses to the fluid through the walls. Percussion gives a dull sound everywhere that the fluid is in contact with the abdominal walls. Gravity, of course, leads the liquid to occupy the dependent parts. In the dorsal decubitus, and when the transudation is of medium amount, the dullness is bounded in the central and upper parts of the abdomen from a region of tympanitic resonance by a line concave toward the head of the patient. The surface of the liquid being horizontal, of course the dullness reaches nearer to the thorax along the sides of the abdomen than in the central line. We would add, that where the layer of ascitic fluid is thin we can obtain dullness only by light, superficial percussion. If the pleximeter or finger is pressed deeply in, the fluid is crowded to one side, and we get a tympanitic sound from the underlying coils of intestine. A factor of great diagnostic value is the change of dullness on change of position of the patient. If he lies upon one side, the fluid seeks the dependent portions of the cavity, and gives rise to extensive dullness there, while the opposite side now yields a tympanitic resonance. Or, if he changes to the other side, it in turn becomes dull, and the side previously dull becomes tympanitic. Similar differences are found between the results of percussion in a horizontal and in a sitting posture. It is only when the accumulation is very abundant that there is dullness over the entire abdomen.

The signs mentioned enable us in most cases to make a diagnosis of ascites with ease and certainty. It is, indeed, not always easy to distinguish a transudation of serum from the exudation of chronic peritonitis, for, of course, either sort of fluid would yield the same physical signs. Only, the change in the area of dullness consequent upon a change of position is less pronounced in case of an exudation, because the peritonitic adhesions impede the movements of the fluid; and we have, besides, all the other symptoms to guide us; there may be pain, or thickenings of the peritoneum discoverable on palpation, or signs of tuberculosis; or, on the other hand, there may be some cardiac or hepatic disease, which would render ascites probable. If the fluid is drawn off, its character will sometimes aid us in diagnosis. Ascites yields pure serum, containing almost no morphological constituents. Its specific gravity is usually less than that of a peritonitic exudation, because it contains less albumen. We may say that the specific gravity of the fluid found in peritonitis is generally above 1018, and that of ascites about 1012, or even lower. Hæmorrhagic ascites sometimes occurs in anæmic patients who are suffering from marked portal obstruction, as we have ourselves seen, for example, in hepatic syphilis.

There may be equal difficulty in the exclusion of ovarian cysts, particularly since the cysts are sometimes so large as to fill the whole abdominal cavity. We must first map out accurately the dullness on percussion, and also see if it varies with changes of position. In cases of ovarian tumor, change of position does not make much difference. The resonance on percussion of the deepest and most dependent portions of the abdomen may be misleading, in this way, that even in ascites a narrow zone here may be tympanitic. This should be remembered. Thus, just above the symphysis, there is sometimes a tympanitic resonance in ascites which might readily be mistaken for a proof of the existence of an ovarian tumor. The explanation is that in the places indicated a coil of intestine with a short mesenteric attachment may remain in contact with the abdominal wall in spite of ascitic accumulations. Further aid in the differential diagnosis is to be obtained from the history of the case (place where the swelling began), from a consideration of possible causative diseases, and from a vaginal examination. In ascites the uterus is freely movable, while in case of ovarian tumors it is often bound down by adhesions. Further particulars may be sought in books on gynæcology.

The treatment of ascites, of course, depends largely upon the disease of which it is a symptom. As to the symptomatic treatment of ascites itself, we will confine ourselves to a few words about tapping. This operation is indicated when the local disturbances caused by the ascites are great; that is, if there is an unbearable sensation of pressure and tension, and, above all, if the crowding up of the diaphragm causes much dyspnœa. The instrument to be used is a common trocar of medium size. Usually the patient is tapped lying on his side in bed, at the most dependent point in the lateral portion of the abdominal walls; but it is also a good way to make the puncture in the median line, about half-way between the umbilicus and the pubes, with the patient in a chair. The operation is easy, and almost free from danger. We may allow large amounts of liquid (five or ten quarts, or more) to flow slowly away at one tapping. Over the puncture we put a piece of sticking plaster, or, if great caution is to be exercised, an antiseptic bandage. Often the fluid trickles out through the opening, because the abdominal walls have lost their elasticity on account of the persistent distention. We may then employ a "circumvoluted suture" to close it. After tapping, the laxness of the walls is favorable to palpation of the abdominal organs.

Inasmuch as tapping does not remove the cause, there is in most cases a very rapid reaccumulation of fluid. Thus the system is deprived of much albumen, and nutrition is impaired, so that not infrequently the operation is followed by decided loss of strength. Therefore, we should not tap in ascites, as a rule, unless the indications for the operation are urgent.

[Before puncture the precaution should always be observed to see that the bladder is empty.

If, as is very frequently the case, the fluid continues to drain away through the puncture after the trocar is withdrawn, good rather than harm results, provided the danger of irritation of the skin and of bed-sores is kept in mind and guarded against, and an instrument of moderate size is used.

Flint advocates early and repeated tappings if the fluid causes discomfort and does not yield to diuretics or cathartics. The pressure is removed in a measure from the abdominal and thoracic organs, and nutrition is thus promoted. The fluid is likely to return, but does not always do so, or may do so only slowly. He reports cases in which, after repeated removal, the fluid ceased to return and the patient remained apparently well. The result must depend, of course, chiefly on the underlying cause, which is sometimes very obscure.

In cases of cirrhosis the same principles govern Flint's treatment.]

CHAPTER IV.

CANCER OF THE PERITONEUM.

CARCINOMA[1] is the only new growth of any practical importance to which the peritoneum is liable. Primary endothelial cancer, analogous to the growth which attacks the pleura, is very rare. Cancerous growths here are usually secondary to cancer of the stomach, intestine, pancreas, liver, or some other organ. Often the secondary nodules are numerous, and almost as small as peas, presenting what is called miliary carcinosis of the peritoneum. Separate nodules of larger size are less frequent. These may be found in the omentum, in Douglas's pouch, around the navel, or in other situations. Colloid cancer attains the most diffuse and extensive development of any variety. The retroperitoneal lymph-glands may also present at the same time large cancerous growths. Often the development of cancer in the peritoneum is attended with pronounced inflammatory disturbances —that is, we have a cancerous peritonitis.

The symptoms of peritoneal cancer resemble in many points those of chronic tubercular peritonitis. Simple miliary carcinosis may be very insidious and give rise to no special symptoms, so that it often is unsuspected. In many cases a moderate amount of fluid collects in the abdomen, and this, if we are aware of the existence of a primary cancerous growth, may lead us to surmise a secondary peritoneal carcinosis. The symptoms are much more pronounced if there is cancerous peritonitis. In that case there is usually very severe pain, marked abdominal distention, and constipation. We may sometimes feel the larger nodules in the omentum or upon the inner surface of the anterior wall of the abdomen, or even those in the lowest part of the abdomen, by palpation through the vagina. If the exuded fluid be drawn off, it is sometimes merely serous, but it may be hæmorrhagic. When the new growth has been diffuse, and particularly in case of colloid cancer, the exudation has repeatedly been found to present a milky opacity. Sometimes this fluid also has been tinged with blood. The opacity is due to fat, from fatty-degenerated and disintegrated cancer cells. Occasionally the microscope reveals characteristic cancerous elements in the fluid.

The diagnosis can not be made with any positiveness unless, as a sequel to a primary cancerous growth already demonstrated, we observe the evident tokens of peritoneal disturbance, such as free fluid and pain. Other points are the patient's age, cancerous cachexia, and secondary glandular enlargements, particularly in the groins.

Treatment must be confined to efforts at mitigation of the suffering. Warm applications, morphine, and supporting measures are chiefly employed.

SECTION VII.

Diseases of the Liver, Bile-ducts, and Portal Vein.

CHAPTER I.

CATARRHAL JAUNDICE.

(*Icterus catarrhalis. Gastro-duodenal Catarrh with Jaundice.*)

Ætiology.—When discussing intestinal catarrh, we mentioned that inflammation of the duodenum may invade the secretory ducts of the liver, and, above all, the ductus choledochus communis. This complication would be of little clinical importance did it not in many instances prevent the flow of bile into the intestine. Such an obstruction at once gives pathological interest to a catarrh of the bile-ducts, because it entails a series of very important clinical symptoms. In this case the cause of the biliary stasis is purely mechanical, and any closure of the hepatic ducts, however produced, gives rise to identical symptoms, which vary, if at all, only in their duration and intensity. Catarrhal jaundice is therefore only one form, though the most frequent form, of what is called hepatogenous jaundice. Accordingly, we will in this chapter describe in detail the general phenomena common to all cases of hepatogenous jaundice, that we may avoid needless repetitions hereafter.

The causes which lead to a gastro-duodenal catarrh, with consequent jaundice, may be the same which produce ordinary gastric catarrh—such as errors in diet. But we must emphasize the fact that we see catarrhal jaundice so often without any striking cause, as strongly to suggest the thought of some specific lesion, some special pathogenic agent. The precise facts are indeed yet to be learned; still it has been often observed that at many times (particularly in spring and autumn) it assumes epidemic proportions. The infectious nature of the jaundice is still more probable in those cases where the disease is decidedly endemic. In barracks, prisons, and single houses quite important endemics of jaundice have been seen, the only explanation for which could be found in assuming the existence of some local source of infection. In a few cases, epidemics of catarrhal jaundice have followed revaccination, compelling one to think that possibly the pathogenic agent was transferred by inoculation. Patients themselves not infrequently refer to taking cold, or to mental emotion (particularly anger), as circumstances promoting the disease.

Another cause is the rather frequent occurrence of duodenal catarrh as a result of passive congestion. This is seldom very intense. It is especially frequent in heart disease. Again, the slight jaundice quite often seen in the course of many acute diseases, and of lobar pneumonia in particular, must be ascribed to catarrh.

Pathology.—As in catarrhal affections of most other mucous membranes, the post-mortem signs of catarrh of the bile-ducts are not always striking, for the swelling and injection subside considerably after death. The usual method of testing the patency of the common duct is by pressing upon the gall-bladder to see if its contents can be squeezed out into the intestinal canal. If a catarrh has closed the common duct, the bile is not discharged at once. A firmer pressure drives out from the opening of the duct a plug of tough, white mucus, upon which the bile follows; but such a plug is not present in every case, by any means, for even a simple swelling of the catarrhal membrane suffices to obstruct the flow of bile.

If the biliary passages are slit open, the common duct is found more or less filled with tough, white mucus. Usually the portion which lies in the intestinal wall, the so-called intestinal portion, is most affected. Behind the occluded part the ducts are distended, if the biliary retention has been chronic. This distention may involve even the smallest ducts, which lie in the liver itself. As a consequence, the liver is somewhat enlarged and has a diffuse bilious tinge. If the obstruction persists for a long while, which is very exceptional in simple catarrhal jaundice, a portion of the hepatic cells are destroyed by the pernicious influence of the retained secretion. The lost parenchyma is replaced by new-formed connective tissue. For further particulars, see the chapter on biliary cirrhosis.

Clinical History.—Inasmuch as catarrh of the ductus communis is almost always consequent upon a gastro-duodenal catarrh, the first symptoms are usually referable to the latter disease. It is indeed seldom that the attack begins with marked gastric disturbances, like violent vomiting and gastralgia, but almost invariably the jaundice is preceded for a variable period by indisposition, as shown by malaise, languor, anorexia, a bad taste in the mouth, nausea, gastric oppression, eructations, and sometimes temporary vomiting. Then comes the first evidence that the catarrh has invaded the common duct, in a jaundiced hue of the skin and of the visible mucous membranes. In many cases, however, the initiatory gastric symptoms are almost wholly absent, so that the disease begins immediately with the appearance of the jaundice.

The pressure in the secretory ducts of the liver is extremely low, so that even the catarrhal swelling of the mucous membrane and the collection of viscid mucus in the common duct suffice to impede in a marked degree the further outflow of bile into the intestinal canal. As a rule, however, the retention of bile is not complete, or, if so, only for a time. Still a considerable amount of bile collects, and distends even the intra-hepatic ducts. As soon as this stasis has reached a certain point, the stagnant bile is absorbed by the hepatic lymph-vessels. Thus the bile and all its constituents are poured into the blood by way of the thoracic duct and carried to all parts of the body.

No more than a few days need elapse before the bile-pigments are absorbed into the tissues, and give rise to the evident yellow color of the skin and mucous passages which we call jaundice. Usually the yellowness of the conjunctiva is the first thing to attract attention. Later the entire skin becomes yellow, and the same color is plainly visible in the mucous membrane of the mouth and throat, especially after we have produced temporary anæmia by pressure, as in the lips. Of course, the internal organs, which we can not see, are likewise stained. Any abnormal collection of liquid will also have a marked yellow color. The cornea, the peripheral nerves, and the cartilages alone escape unstained. In other parts we may not only find this diffuse impregnation with the biliary pigments, but even solid granules of the latter.

A jaundiced patient often presents other indications of the presence of biliary coloring matter than the color of his skin. If the jaundice is at all chronic, there is almost invariably an itching of the skin, which may be very troublesome. It may be so bad at night as to disturb sleep. The scratching thus induced often causes numerous excoriations and fissures, which may even occasion quite large furuncles. Urticaria is also sometimes observed. A peculiar disease of the skin, which has been chiefly described by English authors in connection with jaundice, is called *xanthelasma*. It presents bright-yellow spots, usually somewhat elevated, which are found mainly on the eyelids, though also on other parts of the body.

The remaining symptoms of hepatogenous jaundice may be divided into two groups. The first group comprise the symptoms excited by the presence of the biliary constituents, and particularly of the biliary acids, in the blood, while the second group are due to the lack of bile in the intestinal canal.

We have seen that, when the biliary outlets are occluded or narrowed, the constituents of the bile are absorbed by the lymphatics. We have already learned in part what becomes of the bile-pigments thus conveyed into the blood-vessels. The presence of the bile-acids in the blood is also of considerable clinical importance. Physiology has shown that these acids possess certain poisonous properties, and, among others, the power to destroy red blood-corpuscles. But in reality few if any blood-corpuscles are destroyed by the bile-acids in the blood, because they are too much diluted, and, besides, seem in large part to be quickly decomposed after absorption. These acids do really, however, excite certain nervous centres in a way to give rise to decided clinical symptoms. The most frequent effect is that produced by the cholate of sodium upon the cardiac ganglia, or possibly also upon the center for the vagus, and it is evinced by a slowing of the pulse. This is an almost invariable phenomenon, provided there be no fever or other complication, and is seen not only in simple catarrhal jaundice but in all cases of hepatogenous icterus. The pulse-rate is from 64 to 50, or even less. Slight irregularity in the heart's action is not infrequent. There are certain other nervous disturbances often seen in jaundice and referable to the presence in the blood of biliary constituents, and in particular of biliary acids. Sometimes there is a striking languor and muscular weakness, or headache, or the patient is "out of sorts." Grave nervous symptoms, sometimes seen in jaundice and grouped under the name of *cholœmia*, are discussed in another chapter. It also deserves a brief mention here that many cases with marked jaundice have a noticeable tendency to bleeding—that is, a sort of "hæmorrhagic diathesis." Hæmorrhages into the skin and in the viscera are quite often seen, and also epistaxis and analogous occurrences.

We now come to the second group of symptoms, which result from lack of bile in the intestinal canal. It will be easy to understand these if we briefly review the physiological functions assigned to the bile which is poured into the alimentary canal. Bile plays an important part in the digestion of fat, emulsifying it, and promoting its absorption into the chyle-ducts. Now, in hepatogenous icterus this work remains undone, as is shown by the fatty stools. From time immemorial the white clay-colored stools of jaundice have been well known, and are employed as the best measure of the completeness of biliary retention. The light color of the stools is partly due to the lack of biliary pigment, for it is that chiefly which imparts to normal fæces their dark-brown color; but the characteristic white clay color is due exclusively to the presence of undigested fat in large amounts. We have ourselves performed the experiment of putting a patient with extreme hepatogenous icterus upon a diet containing as little fat as possible, and have found that the stools then became light brown, and not at all like clay. Upon microscopic examination of the fæces in jaundice, sheaf-like aggregations of crystals are not infrequently observed. These were formerly supposed to be tyrosine, but Oesterlein has shown them to be in reality magnesia-soap.

The retention of bile has some further effects besides retarding the digestion of fat. Bile possesses decided antiseptic properties, and thus, to a certain extent, protects the contents of the primæ viæ from putrefaction. In hepatogenous jaundice, therefore, the processes of decay are abnormally active; the fæces are unusually offensive, and there is an excessive generation of gas, often leading to flatulence and tympanites. The bile also undoubtedly stimulates peristalsis, and so in jaundice there is often constipation.

One important function of the bile remains to be considered—namely, that by precipitating pepsine it terminates peptic digestive action. Kühn has shown how necessary this is to the normal processes, because pepsine will destroy pancreatine, and therefore interferes with pancreatic digestion. Hence we are justified in assuming that in well-marked jaundice the digestive action of the pancreatic juice on fat and albumen is probably disturbed, even though the pancreas itself secretes normally. Often, and in catarrhal jaundice probably as a rule, the pancreatic duct is obstructed as well as the biliary, so that not only the bile, but also the pancreatic juice is retained. Just how far the digestive disturbances are to be ascribed to the lack of each of these secretions we are of course unable to determine.

We must now inquire what becomes of the absorbed bile. As to the biliary acids, we have already said that they probably undergo decomposition. Of the other constituents, including the taurine and cholesterine and the pigmentary matters, we know the fate of the last-named only—that is, we have learned how Nature seeks to rid herself of this foreign substance. As soon as the amount of bile-pigment in the blood and tissues becomes considerable, excretory efforts are made, in which the kidneys take the chief share. Certain changes take place in the urine almost simultaneously with the first appearance of a jaundiced hue in the skin; and these changes are due to the urine containing excreted biliary coloring matter.

The urine of jaundice is generally recognizable from its color, which is a dark brown, like beer. The foam caused by shaking it is not white, but distinctly yellow. A bit of white filter-paper dipped in the urine is stained yellow. If the urine is mixed with chloroform in a test-tube, the chloroform dissolves the pigment, and, on being allowed to collect at the bottom of the tube, displays a deep-yellow color. This is known as the "chloroform test." Another reaction which usually gives a satisfactory result, but not always, is Gmelin's. If urine containing bile-pigment is slowly poured down the sides of a test-tube containing some fuming nitric acid, the zone between these two liquids exhibits a fine play of colors. The effect of the acid upon the biliary pigment is to produce a number of colored rings, the highest and most characteristic of which is green; next comes blue, then violet and red. Gmelin's test often shows very prettily if one filters the urine and then adds a drop of nitric acid to what remains upon the moist filter-paper. The characteristic colored rings form around this drop.

The biliary acids also may be detected in the urine of jaundice; but the process is somewhat tedious, and the knowledge gained is of no practical importance.

The urine very often contains morphological elements which are characteristic. Nothnagel was the first to describe minutely the icteric casts—that is, hyaline casts which usually have a yellow tinge and quite often are completely covered with dark-yellow granules. The urine may contain a little albumen also, but this is not constant.

[The presence or absence of albumen depends largely on the amount of the biliary constituents and on the length of time they continue in action on the kidneys; their effect on these organs is more or less that of an irritant.]

The sweat-glands also take part in the excretion of bile-pigment. The latter can be demonstrated in the perspiration of jaundiced persons, as well as in their urine. Not infrequently the patient's linen is colored yellow by the sweat. On the other hand, no bile-pigment is found in the tears, saliva, gastric juice, or secretions other than those mentioned.

Having now considered the phenomena common to all cases of hepatogenous icterus alike, we revert to the subject of simple catarrhal jaundice. The prodromal gastric symptoms usually last a few days, more rarely a week or two, when the skin becomes evidently jaundiced and the other results of the icterus are also seen. The urine grows dark with biliary pigment, the stools become light-colored and more or less clay-colored. The nervous system is not usually seriously deranged, but still most patients feel very languid, and have anorexia and a tendency to constipation. The pulse becomes somewhat slower than normal, and sometimes the temperature also is subnormal, say 97° or 98° (36°-36·5° C.).

In most cases the physical examination of the liver is of interest, the organ being, as already mentioned, enlarged from the retained bile. Accordingly the lower boundary of hepatic dullness usually extends two or three finger-breadths below the edge of the ribs, and not infrequently the lower margin of the organ can be plainly felt. Often the gall-bladder is so distended, both by bile and by the mucus which it itself secretes, that it projects from under the edge of the liver. In such cases, as Gerhardt tells us, we may sometimes make out by percussion a convexity in the lower line of hepatic dullness, which corresponds to the gall-bladder. If the abdominal walls are lax, we may even feel the distended viscus. As a rule, there is not much distress in the hepatic region, although now and then there is a certain sensation of pressure or tension.

The symptoms depicted seldom last longer than a few weeks. Usually a patient who takes proper care of himself begins to feel better in even less time. The urine grows lighter colored, the stools darker, and the pulse more rapid. The yellow color of the skin often remains visible for quite a while, although gradually diminishing, even after the patient feels perfectly well; but at last the jaundice disappears also and recovery is complete. Relapses are indeed possible, particularly after errors in diet; but they are rare.

The termination of catarrhal jaundice is, therefore, almost invariably favorable. The entire course of the disease occupies about three to six weeks, rarely a longer period. It is a very exceptional occurrence, but one which we must always think of as possible, for this apparently mild and secure condition to be suddenly merged into the grave, pernicious variety of jaundice. (See the chapter on acute yellow atrophy of the liver and pernicious jaundice.)

Diagnosis.—Catarrhal jaundice is usually easily diagnosticated. The diagnosis is made chiefly from the course of the disease—the development of jaundice, preceded by gastric symptoms, in a previously healthy person, and generally in a youthful individual. It is very important to exclude other conditions which might occasion jaundice. We must consider, therefore, whether the history of the case suggests the presence of gall-stones (hepatic colic), and be vigilant in our physical examination to detect a possible cirrhosis or new growth. In the case of elderly patients, particularly, it is not rare for what was at first regarded as an attack of ordinary catarrhal jaundice eventually to disclose itself as a grave chronic disease. We should not make a diagnosis of catarrhal jaundice until we have carefully weighed all the rational and objective signs.

Treatment.—Most cases of catarrhal jaundice terminate favorably and require no active treatment. Rest and prudence are indicated, and the diet should be carefully regulated, that the gastro-duodenal catarrh may not be aggravated. Fat must not be eaten, for, as we have seen, it is not assimilated, and only excites abnormal processes of decomposition in the intestinal canal. Lean meat, bread, soups, if not too rich, vegetables, preserves, and lemonade or tamarind-water are allowable.

We should also employ internal remedies to mitigate the catarrh. The various stomachic tonics are frequently prescribed. Rhubarb is a favorite drug. A very good medicine is Carlsbad water, or the artificial Carlsbad salts, of which latter the dose is half a teaspoonful to a teaspoonful, in a tumbler of warm water, before breakfast. The alkalies not only exert a direct beneficial influence upon the gastric mucous membrane, but are also useful as laxatives. More obstinate constipation may call for castor-oil, calomel, or rhubarb.

Lately much enthusiasm has been displayed about the treatment of catarrhal jaundice by large enemata of cold water. The injections are said to overcome the biliary retention by exciting peristalsis, and possibly by also promoting the secretion of bile. Once a day a quart or two of water, at 60° to 70° (12°-18° R.), is injected, and is retained as long as possible. The good effect is said to be observable in a few days, both in the general condition of the patient and in the diminished amount of bile-pigment in the urine, as well as the darker color of the stools.

The effort has also been made to empty the gall-bladder by manipulation. Gerhardt states that sometimes the distended viscus can not only be felt through the abdominal walls (*vide supra*), but it can be so firmly compressed as to squeeze its contents into the duodenum. Sometimes the obstruction is said to yield suddenly, as if a plug were driven out. This method has not been universally adopted. It seems applicable only in a limited number of cases, and is probably not free from danger. Several authorities have recommended external faradization as a means to stimulate the gall-bladder to contract and discharge its contents. We believe that few will adopt the suggestion.

APPENDIX.

ACUTE FEBRILE JAUNDICE. WEIL'S DISEASE.

Within a few years an acute infectious disease has become known, which invariably causes jaundice and may therefore be properly described in this connection. It was first described by Weil, then by Fiedler and others.

The disease is most frequent in the months of summer. It attacks by preference young and middle-aged men. Fiedler was struck by the frequency with which butchers suffered from it. The symptoms usually begin suddenly. Extreme chilliness, fever, headache, and malaise are almost always present at the onset. Jaundice usually appears on the second day, and it may become severe. Its immediate cause is doubtless obstruction of bile, for the stools are colorless, and the urine contains an abundance of bile-pigment. The constitutional symptoms remain for several days quite severe. The patient complains of violent headache, wakefulness, and vertigo. Sometimes there is evident stupor or mild delirium. Upon physical examination we not infrequently notice herpes on the lips, besides the icterus. The tongue is coated. There is nothing unusual about the lungs or heart, except that the pulse is apt to be quite rapid. The abdomen is not particularly distended. The liver is often enlarged, but not always. An acute splenic tumor is very often, but not invariably, present. There is usually diarrhœa. Vomiting may occur. There is generally albuminuria; and not infrequently the existence of a nephritis is shown by the blood and casts found in the urine. A very characteristic symptom is the violent pain in the muscles, particularly in the calves, of which most patients complain.

With these symptoms the disease persists for from five to eight days, during which period the fever is often very considerable. Temperatures of 105°-106·8° (40°-41° C.) are not rare. Then the fever falls by lysis, although seldom with perfect regularity. At the same time the other symptoms abate also, and convalescence ensues after an illness of ten to fourteen days in all. Many milder cases recover even sooner. An unfavorable termination seems to be quite exceptional.

It is highly probable that this perfectly specific disease, the most important symptoms of which are, as we have seen, jaundice, fever, swelling of the spleen, albuminuria, and pain in the muscles, is an acute infectious process; but we do not yet possess any precise information as to its ætiology.

The treatment must be purely symptomatic. In the beginning calomel is useful. Later on, hydrochloric acid, rhubarb, and the like are prescribed. The headache is relieved by an ice-bag, the pain in the muscles by rubbing with chloroform liniment.

CHAPTER II.

BILIARY CALCULI.

(*Hepatic Colic. Cholelithiasis.*)

Ætiology.—Although gall-stones are of very frequent occurrence, we do not yet possess much information as to their causation. It is only possible to state a few factors which very probably promote their formation.

Biliary retention certainly acts in this way, both directly and by leading to an increased consistency and increased concentration of the bile. As a result, certain constituents which were before held in solution are thrown down. And yet this cause, however potent, can not be regarded as the only one. The chemical examination of gall-stones leads plainly to the conclusion that their formation must be preceded by certain abnormal chemical processes of decomposition and of transformation. We can not otherwise explain why the constituents of gall-stones should differ, as they do in many ways, from the matters which normal bile holds in solution. For example, the pigment in gall-stones is never found unchanged, but invariably exists in composition with lime. Now, normal bile contains only a trace of lime, so that long ago Frerichs suggested that the lime comes from the mucous membrane of the gall-bladder. It is an important fact that the cholesterine, and probably also a portion of the pigmentary matters, are held in solution in normal bile by the combination of sodium with the biliary acids which it contains. If this sodium salt were decomposed from any cause, the matters named would naturally be precipitated. The decomposition of the salts formed by the bile-acids is greatly promoted if the bile acquires an acid reaction; but of the circumstances in which this last-mentioned change occurs we do not yet have any accurate knowledge. Possibly fermentation may have something to do with it. The idea that often a clump of mucus forms the nucleus of a biliary concretion lacks chemical confirmation, for mucus is never found in the stones.

We have rather more knowledge as to predisposing causes than about the chemical processes involved in the formation of gall-stones.

Age seems to be an important factor. The great majority of patients are over forty. Gall-stones are much less frequent between twenty and forty years of age; and in children they are very rare, although they have been observed in the new-born. One reason why elderly people are so liable to this trouble is said be the senile weakness of the muscular fibers of the gall-bladder. Thus stagnation and retention of bile are promoted. It has also been suggested that in old age the bile may contain an excessive amount of cholesterine and lime.

Sex also has a decided influence. All authors agree that gall-stones are more frequent in females than in males, the proportion being about three to two. An explanation of this fact has been sought in the sedentary life of women, and particularly in the mechanical effect of tight lacing, impeding the outflow of bile.

Much has been said about certain peculiarities of bodily temperament in relation to cholelithiasis. Obesity, gout, and chronic endarteritis are said to favor the formation of gall-stones. A like influence is ascribed to excessive indulgence in meat and fat, as well as to gluttony in general, and to lack of exercise; but the real importance of these various factors is not at all definitely determined.

Diseases of the liver and bile-ducts do undeniably promote cholelithiasis, for they interfere in many ways with the discharge of bile from the ducts and from the gall-bladder. Thus they may cause compression or obstruction of a duct, or may lead to degenerative changes in the tissues of the gall-bladder. The view is quite generally held, but is apparently incorrect, that even a simple chronic catarrh of the bile-ducts tends to cause gall-stones. Gall-stones and catarrh of the ducts are indeed often found to co-exist, but it is probable that the catarrh is not the cause, but the result, of the presence of gall-stones.

Occurrence, and Chemical and Physical Properties of Gall-stones.—The place where gall-stones are most frequently found is the gall-bladder. We may find in it any number, from one or two up to a hundred and more. The size varies from that of a grain of sand up to that of a hen's egg. The large stones may completely fill the gall-bladder; and sometimes the smaller stones are numerous enough to fill it also. The stones usually lie free in the bladder, although exceptionally they may be found adherent to its walls. Rarely the bladder presents a diverticulum, in which a stone has been formed. The lining membrane of the viscus often suffers such mechanical irritation from the stones as to present quite a severe catarrhal inflammation. Sometimes there is even a more or less extensive necrosis (*vide infra*).

Stones which are found in the larger bile-ducts were not formed in them, but have become wedged in them while on their way to the intestine. This condition is described as an impaction of gall-stones. Gall-stones are quite often found in the liver itself, and frequently in large numbers. These concretions may measure half a centimetre to a centimetre in diameter. In such cases the small intrahepatic bile-ducts are usually a good deal widened; or occasionally they present niches in which the stones lie. As a rule, the hepatic parenchyma surrounding the stone is in a state of chronic or acute purulent inflammation (*vide infra*).

In form, gall-stones vary infinitely. The smallest are irregular masses, well described by the name of "gall-sand." The larger stones are more or less round, or oval, or polyhedral. The polyhedra are usually due to the mutual rubbing and pressure of a number of stones upon one another. In color, the stones vary according to the amount of pigment they contain, from almost black or dark brown to a lighter greenish or bright yellow shade. A fresh gall-stone always sinks in water; but when dry, gall-stones contain air and generally float. On cross-section, they are found to be either homogeneous or composed of layers. As a rule, there is a nucleus, darkly pigmented, which is surrounded by a lighter-colored envelope, itself either made up of concentric layers or evidently crystalline. Often the outermost layers are still distinguishable as peculiar darker and harder strata.

As to chemical composition, gall-stones are usually divided into several groups. By far the most frequent variety is made up of cholesterine and pigment mixed together in greatly varying proportions. The pigment is part of it in its natural state and part of it combined with lime. On the average, stones contain about seventy to eighty per cent. of cholesterine. Besides these two chief ingredients, most stones also contain lime and magnesium. In color, they are light or dark according to the smaller or greater proportion of coloring matter they contain. Less common are stones of pure cholesterine. These are usually found singly, are soft, and often are almost transparent. Most cholesterine stones have a nucleus of pigment and lime in combination. Pure pigmentary concretions are rare, and are generally small, like coarse sand. A still greater rarity is a stone made up entirely of lime. Such a stone is small and very hard.

It should also be added that just as is the case with renal calculi, an organic cementing substance can be detected by the microscope in gall-stones. This is in part itself calcified, in part it unites the separate crystals (Posner).

Pathological and Clinical Consequences of Gall-Stones.—Gall-stones may remain for a long while in the gall-bladder, and even in the liver, without causing the slightest unpleasant symptom. It is not at all unusual to find gall-stones unexpectedly at an autopsy.

In other cases, however, gall-stones occasion severe suffering, and sometimes even death. These grave phenomena may be due either to certain mechanical conditions, like impaction, or occlusion of the bile-ducts; or to secondary inflammation resulting from their presence. These two kinds of disturbance must now be discussed in detail.

Gall-stones not infrequently leave the place where they were formed. Stones formed in the liver are gradually driven onward by the current of the bile, and pass through the hepatic duct into the common duct and the intestine. The more numerous concretions formed within the gall-bladder also often change their location. What should make them move is not perfectly determined. Probably there are various factors; in the first place, contraction of the muscular coat of the gall-bladder; and then probably the weight of the stone; and the pressure of the diaphragm and the abdominal walls, as in breathing, defecation, and vomiting. When once a stone has entered the biliary passages, we must regard the secretion behind it as the chief propulsive power; for neither the cystic nor the common duct possesses muscular fibers.

Small calculi may effect their escape without exciting any attention; but the passage of larger ones produces a very characteristic set of symptoms, known under the name of hepatic colic. Attacks of pain are frequently, though not always, the first symptom of the passage of gall-stones. The intensity of the suffering varies greatly in different cases. It may be so mild and ill-defined as not to suggest its true cause, or it may be unbearable.

A typical attack of hepatic colic either begins suddenly, or it has for prodromata nausea, chilliness, and mild constitutional disturbance. The most frequent time for its occurrence is a few hours after dinner. The pain may be violent from the start; or, if at first less severe, it quickly reaches an extreme degree. It is usually felt chiefly in the epigastrium and the right hypochondrium, but radiates thence backward and toward the shoulders, or even into the right arm. The pain comes in paroxysms, being worse each time till it becomes extremely severe. General convulsions have been repeatedly observed as a result of the pain, particularly in nervous individuals. Quite often there is a severe rigor. Vomiting is also common. Usually there is constipation. As a rule, there is great constitutional disturbance; there are extreme languor and weakness, with an appearance which suggests collapse. The pulse is small and somewhat accelerated; less frequently, it is slower than normal. The temperature is normal. During the rigor, however, it may be elevated to 104° (40° C.) or more. On physical examination, the liver is more or less enlarged; and sometimes it is possible to feel a full and distended gall-bladder, or at least to find a dullness on percussion corresponding to it. Jaundice often appears toward the end of the attack, but not invariably. Of course it can be caused by a calculus only when it blocks up the hepatic or common ducts. Occlusion of the cystic duct would not produce it.

The duration of an attack is, in mild cases, only a few hours, and in severer ones seldom more than one or two days. Probably the pain ceases the instant the stone has successfully passed the final narrow portion of the common duct, just above its opening into the intestine. Perhaps relief is sometimes due to the stone's slipping back again into the gall-bladder. If the fæces are carefully examined after an attack is ended, we often find one or more calculi. The best method of search is to pour the fæces upon a sieve, after adding water to them. In one or two instances, gall-stones have passed into the stomach and been vomited up. The interval between different attacks varies. Sometimes months or years intervene, sometimes only a little while. Not infrequently there is a quick succession of attacks, and then none for years or even for a lifetime. In the intervals the patient may feel perfectly well, but he may have a slight jaundice or an enlarged liver or chronic digestive disturbance.

Permanent impaction of a stone at any point in the biliary passages gives rise to quite different phenomena. Usually the violent symptoms of hepatic colic persist for some days, but then abate, leaving behind only a dull pain subject to occasional exacerbations. Sometimes the symptoms almost all vanish, in case the passage is not entirely occluded. If, however, no bile whatever can pass, further trouble is to be expected. If the calculus lies in the cystic duct, mucus collects in the gall-bladder and gradually distends it. The coloring matter of the retained bile is gradually absorbed, so that finally the contents of the gall-bladder is an almost colorless mucous fluid. This condition, of course, arises just the same from occlusion of the cystic duct due to any other cause. It is termed dropsy of the gall-bladder. Sometimes the distended viscus can be felt through the abdominal walls. If a gall-stone lodges in the hepatic duct, or, as is much oftener the case, in the common duct, and dams up the bile, chronic jaundice is inevitable.

Another series of very important symptoms is due to the secondary inflammation or ulceration resulting from the impaction. A gall-stone may excite a secondary inflammation, no matter where it lodges, whether in the gall-bladder or the ducts, or in the liver itself. The phenomena are quite analogous to the inflammation which a fæcal calculus excites in the vermiform appendix (*vide supra*). The first effect of the stone is purely mechanical. Its pressure causes a simple necrosis of the mucous membrane. The inflammation and ulceration are developed secondarily around the necrosed tissue; but once started, they may spread. The germs which excite the inflammation are probably in all cases derived from the intestinal canal. As long as the ulcerative process is confined to the mucous membrane there are no special symptoms; but quite often it gradually involves the deeper tissues or invades neighboring organs. In such cases the number of possible events is almost infinite. We shall mention here only a few of the most frequent and important.

If the gall-bladder or one of the large ducts is perforated, the bile flows into the abdominal cavity. Gall-stones also have more than once escaped in the same way. Such a perforation is almost invariably followed by a purulent and usually quickly fatal peritonitis. This is not excited by the bile itself, for normal bile does not provoke inflammation, but it is due to septic matter (or decomposed bile), which also gains access to the peritoneum. Rarely there is perforation outward. The inflamed gall-bladder forms adhesions to the abdominal walls, the ulceration slowly progresses, and finally reaches the surface of the body. Thus a genuine "external fistula of the gall-bladder" may be formed, through which bile and calculi are discharged. More frequent than either of these occurrences is perforation into neighboring organs, and into the duodenum in particular. Virchow, and more lately Fiedler, have pointed out that we can not explain the appearance of large gall-stones in the fæces on any other supposition than that of duodenal perforation; for it is hardly supposable that calculi the size of a walnut or even larger should pass through the bile-ducts if in a normal state. Of course, the bile can also escape through this same abnormal opening, whereupon the symptoms of biliary retention and those due to lack of bile in the intestines cease. There have been a few cases of similar perforation into the stomach and the colon, and even into the portal vein and the urinary passages.

The clinical symptoms of all these secondary inflammatory and ulcerative processes may, of course, vary greatly. Sometimes the symptoms are for a long time so indefinite that no diagnosis can be made with any degree of certainty. There are abdominal pain, occasional febrile attacks, constitutional derangement, and anorexia—symptoms which, to be sure, indicate some serious trouble, but do not reveal its nature. The case is different if there be a previous history of colic, jaundice, the appearance of gall-stones in the stools, or similar more definite indications. If perforation into the abdominal cavity takes place, acute and severe peritonitis is, as we have said, almost inevitable. If a perforation into other organs occurs, the only sign by which it can be recognized is the discharge of gall-stones through some unusual channel—for instance, through the abdominal walls, or the œsophagus, or the urinary organs. The "biliary abscesses" which may be produced in the liver by gall-stones will be considered under the general head of hepatic abscesses. It is worth while to mention, that in rare instances large gall-stones which have reached the intestinal canal cause obstruction of the gut. There have also been a few cases of secondary inflammation and ulceration of the intestine due to gall-stones.

Diagnosis.—It is evident from what has been already said that often the diagnosis of cholelithiasis is easy and indubitable, while in other cases the symptoms and course of the disease are obscure and ambiguous. The attacks of colic are certainly the most characteristic symptom. We should therefore make it a rule, in case of severe paroxysmal pain in the region of the stomach or liver, to think of the possibility of gall-stones. Any mild jaundice accompanying such an attack makes the diagnosis more probable. For absolute certainty, however, we need to find the stones in the dejecta; but even without this, in most of the typical cases, the disease may be correctly diagnosticated. It is most easily confounded with cardialgia, intestinal colic, renal colic, and attacks of pure "neuralgia in the distribution of the hepatic plexus." Observation of the patient for a considerable time may often be required in order to reach a definite conclusion.

It is but seldom that physical examination of the liver in this disease yields decisive results. Still it is possible, as has been said, that a gall-bladder distended with calculi may be felt through the abdominal walls. Sometimes we can even hear with a stethoscope the friction of the stones upon each other.

Prognosis.—We have already enumerated the manifold dangers which are incident to cholelithiasis, but on the whole these untoward results seldom occur. As a rule, the termination is favorable. Either the calculi escape in some way, and there is complete recovery; or at least the symptoms abate, and the patient feels as well as ever, although the possibility of a relapse hangs over him.

As to the separate symptoms, the colic itself is very seldom dangerous. In a very few cases of extreme severity there has been a fatal collapse. Permanent obstruction of the common duct is worse, because it greatly impairs nutrition, and also leads to secondary changes in the liver (*vide infra*). The most favorable direction for a perforation to take is into the small intestine. It would seem that the fistula thus originated may even heal up perfectly. The ulcerative process may cause an unfavorable result by entailing a cicatricial closure of the common duct.

Treatment.—Our first efforts must be directed to a relief of the symptoms excited by the gall-stones. We must further endeavor to promote their discharge from the body and to prevent the formation of new ones.

It is the hepatic colic which most often demands therapeutic interference. The most important and indispensable remedy is opium or morphine. If there is violent pain, a grain of opium (0·05 grm.) is usually required every hour or two. If there is vomiting, or if immediate relief is called for, a sixth to a third of a grain (0·01–0·02 grm.) of morphine may be injected subcutaneously. Other narcotics, like chloral and belladonna, are seldom needed. In the way of external applications to the hepatic region, warm or hot poultices serve the best purpose.

Some few patients get more comfort from an ice-bag. Usually some relief can be obtained from a mixture of equal parts of chloroform and olive-oil rubbed gently in over the liver. Sometimes relief is obtained by a prolonged warm bath. For violent emesis, opium, potassic bromide, or bits of ice may be administered. In collapse, we must exhibit such stimulants as wine, strong black coffee, or even ether or camphor subcutaneously. After the colic is over, some mild laxative, such as a mineral-water, is generally given, to favor the evacuation of such calculi as may have entered the intestinal canal.

[When giving repeated doses of opium for biliary or renal colic, it should be remembered that the pain will cease abruptly as soon as the stone ceases to obstruct the passage, and that severe toxic effects of the drug may then appear. In bilious colic the distance to be traversed by the stone is relatively short, and in that affection especially the inhalation of ether or chloroform is sometimes the best, and, indeed, the imperative treatment.

The operation of cholecystotomy has now won for itself an enviable position in surgery, and in the hands of a skilled surgeon gives permanent relief at relatively small risk to cases of recurrent and persistent gall-stone impaction which are quite beyond the reach of internal remedies.]

The second indication, namely, to prevent the formation of fresh concretions, is best met by certain alkaline mineral waters. Just how they act is uncertain, but that they do exert a favorable influence has been satisfactorily established by experience. The springs of Carlsbad have gained the highest reputation of any. If the patient's circumstances permit, it is always best to send him to Carlsbad. During the "cure" there it often happens that a large number of gall-stones are passed with comparatively little discomfort, and the patient not infrequently returns home to enjoy for years, or even for life, freedom from his old trouble. Vichy is good, as are also Kissingen, Homburg, Marienbad, and Ems. If the patient can not travel, he must drink Carlsbad water at home for a month or six weeks.

All the other remedies are of dubious efficacy. "Durande's remedy," which is a mixture of three parts of ether and two parts of oil of turpentine, is much in vogue: twenty or thirty drops are to be given two or three times daily for a long while. The internal use of chloroform is also recommended. Ten or fifteen drops in some mucilaginous vehicle are given three or four times a day. Of newly suggested remedies, salicylate of sodium deserves especial mention. It certainly promotes the secretion of bile. It is given in long-continued small doses (30 grains to 1 drachm—2·0–4·0 grm.—*per diem*). Italian physicians highly praise the free use of olive-oil, say six or seven ounces (200·0 grm.) daily in divided doses. It is said that in obstinate and severe cases of hepatic colic the oil sometimes causes marked improvement.

[Most of the oil is passed in lumps, which have a superficial resemblance to gall-stones, and are sometimes called such.]

If perforation, peritonitis, or any other special complications arise, they are to be treated on general principles.

CHAPTER III.

SUPPURATIVE HEPATITIS.

(*Hepatic Abscess.*)

Ætiology.—Exclusive of traumatism there are two ways by which bacteria may penetrate into the liver, there to excite a suppurative inflammation—namely, by the blood and through the bile-ducts. In the circulatory system the main route is by way of the portal vein, by which germs from the intestines reach the liver. This explains why many ulcerative processes in the intestine, like severe dysentery, are followed by hepatic abscess; and why purulent pylephlebitis (*q. v.*) and other suppurative processes within the portal system may have a similar sequel. In general pyæmia, the germs must take a very circuitous route in order to reach the liver. They must, on leaving the primary abscesses, first enter the veins and the lungs, and then gain the liver by way of the hepatic artery. It has been well known for a long time that suppurating wounds of the head are followed by hepatic abscess with comparative frequency. Perhaps it may exceptionally happen that infectious matter enters the hepatic veins by "retrogressive embolism" from the vena cava.

The germs which make their way into the liver from the bile-ducts invariably originate in the intestine. In these cases the hepatic inflammation is almost invariably preceded by disease of the biliary passages. The most frequent cause by far of this variety of hepatic abscess is the formation of gall-stones. The two most essential factors are the mechanical injury caused by the concretion—that is, necrosis from pressure—and the decomposition of the retained bile.

Among us, hepatic abscesses are rarely occasioned in other ways than those indicated; but in the tropics it is said that quite a large number of apparently primary hepatic abscesses are met with. Their origin is not yet explained, although in this case also there is most probably an invasion of pyogenic bacteria from the intestine.

Pathology.—The smallest and as yet imperfectly developed abscesses best illustrate the mode of formation. We find the blood-vessels choked with micrococci, and the cells of the surrounding parenchyma void of nuclei and in process of disintegration. Along the course of the blood-vessels nuclei are very abundant. These are due to white corpuscles which have escaped through the vascular walls. The cells and the liquid exudation rapidly increase, and there is complete destruction of the hepatic parenchyma, and the formation of an abscess in its place. This may extend indefinitely in all directions. Large abscesses may at last involve an entire lobe. In other cases the suppurative process is limited by encapsulation. Sometimes quite large portions of the liver become necrotic and slough off, under the influence of what is called "sequestrating" suppuration. We almost invariably find some shreds of hepatic tissue in the pus of hepatic abscesses. Where the abscess is due to gall-stones, the latter are found in the pus.

Small abscesses may be absorbed, but they are often merely symptomatic of pyæmia or some such disease, which is itself incurable. Larger abscesses may point into neighboring organs. If they are discharged into the abdominal cavity, diffuse peritonitis follows. The most favorable termination, and one repeatedly observed, is perforation through the abdominal walls, after these walls and the liver have been joined by adhesions. They may also break into the pleural cavity, the pericardium, the intestine, and the pelvis of the right kidney.

Clinical History.—An absolutely complete clinical description of hepatic abscess is impossible, because, as we have seen, it may be a symptom of such diverse pathological processes. Hepatic abscesses are often found post mortem which had given no previous indication of their presence; this is frequently true in pyæmia. In other cases there are symptoms, in part directly referable to the seat of inflammation, and in part due to its influence upon neighboring organs.

Enlargement of the liver can often be made out by percussion or even by palpation. It is the result of swelling and hyperæmia involving the entire organ. Extensive abscesses may give much more definite signs of their presence, however, if situated near the anterior edge of the organ. They are sometimes felt through the abdominal walls as hemispherical and actually fluctuating tumors. It is not so very rare for tropical hepatic abscess to attain these dimensions.

Pain in the right hypochondrium, although it may be entirely absent when the abscesses are small, even if they are numerous, is often violent and persistent when the abscess is large. It is excited by the tension of the peritoneal covering of the liver, or by a perihepatitis. The pain often radiates,—and with especial frequency, it is said, into the neighborhood of the right shoulder.

The course of the fever may prove a strong diagnostic point. When the abscess is chronic and encapsulated there may, it is true, be no fever whatever; but, as a rule, fever does exist, and often presents a very characteristic intermittent character. There are great elevations, usually ushered in by a chill, and succeeded by deep depressions of temperature accompanied by perspiration. If the hepatic trouble is merely a symptom of general pyæmia, then the fever is to be ascribed to the latter; but if there are signs of a severe local hepatic disease, such as pain, enlargement, and jaundice, and if these febrile attacks come on at irregular intervals, we should always consider the possibility of abscess of the liver. In the cases of large tropical abscess this sort of fever is the rule. It is most frequent with us in cases of purulent pylephlebitis and of abscess excited by gall-stones. The "*fièvre intermittente hépatique*" of the French is in most instances due to the presence of gall-stones in the liver, with secondary suppuration.

Among the secondary symptoms of hepatic abscess jaundice is prominent. It is not invariably present, however, occurring only when the abscess has compressed some large biliary duct, and has thus given occasion to the absorption of bile by the lymphatics. In rare instances the abscess compresses the portal vein and thus causes ascites. There may be pulmonary symptoms of considerable importance, even when there are no actual pulmonary complications. This is because the right half of the diaphragm is crowded up by abscesses projecting from the convex surface of the liver. Hiccough is sometimes a source of distress, and may be due to the pressure of the abscess upon the stomach. Vomiting is also a rather common and often very troublesome symptom.

There is almost always great constitutional disturbance. The patient has no appetite, and loses flesh, particularly if there are frequent febrile exacerbations. Often there are severe nervous attacks. Very exceptionally, the disease remains latent for a long while, and does not disturb the general health to any great extent.

The course of the disease depends mainly upon the nature of the original disturbance. Severe pyæmic cases, in the course of which hepatic abscesses develop, are generally brief, and are almost invariably fatal. Abscesses due to gall-stones, and the large abscesses which are apparently idiopathic, are generally chronic, lasting for weeks, or even for many months. Cases exhibit manifold diversities, according to the position, size, number, and sequelæ of the abscesses. Among the possible results, we would once more call attention to perforation into neighboring organs. If the pus is discharged externally, recovery may ensue; as also if the pus reaches the intestinal canal or the bronchi, which seldom happens. Perforation into the abdominal cavity always excites a fatal acute peritonitis. As a general rule, hepatic abscess finally proves fatal, recovery being exceptional. Death is due either to the gradual loss of strength or to some complication.

Treatment.—Local bleeding, counter-irritation, purgatives, and emetics are among the remedies which are advocated, but we can hardly expect them to exert much influence upon a hepatic abscess. The best way is to treat the case purely symptomatically, seeking to keep up the patient's strength and mitigate his suffering until, if we are very fortunate, we have a chance for operative interference. When once the other symptoms are re-enforced by the discovery on palpation of a fluctuating tumor, the diagnosis is established, and the pus should be evacuated and the cavity drained. Particulars about the operation should be sought in works on surgery. More than one case of the large tropical abscess has been cured in this way; but the cases which are most common among us—namely, embolic abscesses and those excited by gall-stones—hardly ever afford any opportunity for surgical interference.

CHAPTER IV.

CIRRHOSIS OF THE LIVER.

(*Chronic Diffuse Interstitial Hepatitis. Laennec's Cirrhosis. Gin-drinker's Liver.*)

Ætiology and Pathology.—Cirrhosis of the liver is usually defined as a diffuse interstitial inflammation, chronic in duration, and resulting in a secondary atrophy of the true hepatic parenchyma. This conception makes the disease perfectly analogous to " chronic interstitial inflammation " of the kidney and many other organs. Weigert's careful study of the processes of " chronic interstitial nephritis " has shown that at least a large part of the changes which take place in the connective tissue are not primary, but secondary, and the consequence of a primary destruction of the genuine renal parenchyma. The question naturally suggests itself, whether the same may not be true of the apparently closely allied phenomena of hepatic cirrhosis. We feel justified, therefore, in seeking the origin of the disease in a primary injury and consequent partial destruction of the hepatic cells, whereupon follow a secondary proliferation and final contraction of the interstitial tissue, just as is seen in lesions of the parenchyma of the kidneys, spinal cord, and heart.

Such a conception would be extremely compatible with one fact about the ætiology of the disease, namely, that chronic alcoholism is universally regarded as a potent predisposing cause. Hence the English name, " gin-drinker's liver." The harmful influence of alcohol can be appreciated, if we remember that on being absorbed it is carried directly by the blood-vessels to the liver. The stronger liquors are more potent for evil in this direction; wine and beer are not harmless, however. According to the usual view of the disease, the poison excites a chronic inflammation of the connective tissue; while, according to the new view, the alcohol exerts a specific injurious influence upon the hepatic cells proper, impairing their nutrition, and finally causing their destruction. That the disease attacks the periphery of the lobules and the interlobular connective tissue is equally consonant with either theory. It is well known that the capillary anastomoses of the portal vein are situated between the lobules.

The abuse of alcohol is by no means the only cause of cirrhosis, for quite often the disease attacks persons in whose case no such ætiology is possible. In such instances we are seldom able to demonstrate the real cause. The excessive use of spices, and other analogous substances, has sometimes been regarded as causative. It is also said that sometimes malaria, and the acute infectious diseases, leave behind them a tendency to cirrhosis. The form of cirrhosis which follows diseases of the bile-ducts, and also "syphilitic cirrhosis," will receive separate consideration.

Cirrhosis of the liver is seen much oftener in men than in women, and usually occurs in middle life.

The anatomical changes are generally divided, without regard to the way in which they are brought about, into two stages. In the first stage the liver is uniformly enlarged, resistant, with its edge blunt, and its surface at first perfectly smooth, but later presenting little dimples. On section, the increased consistency, or "interstitial induration" of the liver, can be readily perceived. The acini are separated from one another by a relatively thick band of grayish-red interstitial tissue, and are at first readily distinguishable. Later on, the interstitial hyperplasia invades the acini themselves, and they cease to be discernible. The microscope shows that the cause of this increase in size and firmness of the organ is due exclusively to the abundant cellular infiltration and the new formation of connective tissue between the individual lobules. The neighboring cells of the parenchyma exhibit signs of disintegration, undergoing either simple atrophy or else fatty degeneration.

The second stage corresponds with the process of contraction of the newly formed connective tissue, but in this stage the destruction of the proper hepatic tissue has already assumed grave proportions. On the old theory, the parenchyma perishes because of the great disturbance of circulation in the portal capillaries, great numbers of which are obliterated by the shrinking of the connective tissue. Under this process of contraction the liver undergoes progressive atrophy, and its surface becomes mammillated. The size of the nodules varies. The size of the whole organ may be reduced one half, or even more. Frequently its general contour is considerably modified. Upon microscopic examination, we now find merely vestiges of parenchyma, embracing which are wide, firm bands of connective tissue. Even within the acini there is decided interstitial hyperplasia along the blood-vessels. Brown masses of pigment are often found here and there, which have been left behind by the hepatic cells now destroyed. Regenerative changes can also be detected quite frequently. The most common of these is the formation of small biliary passages in the broad bands of interstitial tissue.

The division of hepatic cirrhosis into two stages is somewhat diagrammatic, for there is really no sharp dividing line between them. The same liver may in different places illustrate both stages simultaneously. Thus, the surface is often distinctly granular, while the liver as a whole remains hypertrophic.

Clinical History.—The onset of the disease is usually insidious. At autopsies, quite an advanced stage of cirrhosis is sometimes found, to which not a single clinical symptom had pointed; and it is often observed that the duration of unambiguous symptoms is much shorter than the degree of anatomical change discovered post mortem would have led us to expect.

It is, however, true that certain prodromata may appear long before the genuine cirrhotic symptoms; but there is generally room for doubt whether these prodromata are excited by the incipient hepatic disease or whether they are not due to other coincident affections, such as the chronic gastric or intestinal catarrh which drunkards so often have. There are anorexia, nausea, epigastric uneasiness, eructations, constipation, and sometimes vomiting. There is evident constitutional disturbance in many cases, but in others the strength is unimpaired. The severer symptoms usually date from the time when disturbance of the portal circulation arises. We have already stated that the diseased process is most active in the interlobular connective tissue—that is, where the portal capillaries are situated. When the contraction of the connective tissue has resulted in the destruction of a large number of these portal capillaries and the minute veins from which they spring, the portal circulation is inevitably impeded, and there arises a passive congestion of the whole portal system. The signs of this are soon manifest.

The stasis in the veins of the peritoneum is, as a rule, the first to attract attention, from the ascites which it occasions. The distention of the abdomen and the sensation of weight and pressure due to this effusion, are often the first things which excite the patient's attention and lead him to seek medical advice. Later on, the ascites sometimes becomes enormous, causing immense swelling and extreme tension of the abdominal walls, and, of course, proportionate discomfort. Proper nursing and internal treatment may diminish the ascitic effusion, but they will seldom wholly remove it. It quite often remains nearly uniform, until finally, for some reason, there is a change for the worse.

Next to ascites, the most important symptom of portal obstruction is enlargement of the spleen, which is due both to the increased amount of blood in the organ and to a diffuse hyperplasia of its tissues. As a rule, the increase in size is considerable, amounting sometimes to two or three times the normal dimensions. The demonstration of splenic tumor is of great diagnostic importance, but is often a difficult matter. Percussion and even palpation are greatly interfered with by the co-existing ascites. On the whole, however, palpation is the more reliable. Pain or other subjective symptoms are rarely observed. Exceptionally, there is no enlargement of the spleen. This may be due to the firmness and thickness of its capsule, or to the general marantic condition of the patient.

The venous congestion of the stomach and intestine excites catarrh, which is evinced by anorexia, nausea, and irregularity of the bowels. Usually there is quite obstinate constipation, but there may be persistent diarrhœa. None of these symptoms occupy the foreground of the clinical picture, however, both because they are frequent in all grave chronic diseases and because many patients have had digestive derangements long before these severer troubles began. A more significant symptom, if it occurs, is hæmorrhage. This is now and then occasioned by the extreme gastro-intestinal congestion. The bleeding may be from either the stomach or the intestine, and is sometimes a relatively early phenomenon. Sometimes there is a capillary oozing sufficient to tinge the stools day after day for some time. Very rarely there is hæmorrhage from the œsophagus.

The patient may be moderately jaundiced even in the common form of cirrhosis. This is sometimes due to duodenal catarrh. Often there is no jaundice whatever; or there may be a slight yellowish tinge of the skin, in addition to the dirty, grayish, earthen color which not infrequently characterizes the cirrhotic. Perhaps the jaundice is due in many cases to stenosis of the intra-hepatic bile-ducts, involving biliary retention.

The above signs of portal obstruction will often render the diagnosis of hepatic disease extremely probable, but we should always endeavor to confirm our opinion by physical examination of the liver. In the later stages of the disease, and particularly if there be great ascites, our efforts may be fruitless; but at first, or after paracentesis has been performed, percussion and palpation may yield valuable information. In the earliest stages the liver is large. Hepatic dullness reaches some ways below the edge of the ribs, and we can often feel the lower edge and anterior surface of the organ. Later on we find the surface irregular and rough. If we can feel these little nodules or prominences through the abdominal walls, as we sometimes can, of course the diagnosis of cirrhosis of the liver is nearly certain. As already mentioned, it often happens that irregularities are already to be felt upon the surface of the organ while it still remains hypertrophic. The demonstration of atrophy by percussion in the later stages of the disease is less reliable. The ascites often interferes with such an attempt. We may also be misled by coils of intestine distended with gas and perhaps lying in front of the liver. If, however, after guarding against error, we constantly find the area of hepatic dullness diminished, the sign has some value.

General nutrition is usually much impaired in the later stages of the disease. At first the patient may retain vestiges of his former corpulence, but finally he grows emaciated. Anasarca may exceptionally occur toward the close; but there is frequently considerable œdema of the lower extremities, and even of the scrotum and the dependent portions of the abdominal walls. The cause of this is a purely local one—the pressure of the ascites impedes the return of blood from the lower limbs to the heart.

Occasionally there are ecchymoses into the skin, the mucous membranes, the retina, and other parts. These are probably due to malnutrition of the vascular walls.

Unless there are complications, there is no fever. Respiration may be impeded and accelerated on account of the upward displacement of the diaphragm. The pulse is usually small, and often somewhat more rapid than normal.

At first the urine presents no characteristic changes. When the ascites has become considerable and there is œdema, the urine grows scanty, dark, and concentrated, and often has an abundant sediment of urates. It should be mentioned that earlier observers found a diminished excretion of urea. This is perhaps due to a disturbance of the urea-generating function of the liver, about which both earlier and more recent investigators agree. In a few instances the urine has been found to contain a trace of sugar.

It remains for us to describe briefly the collateral circulation which may be developed in cirrhosis, so as to enable the blood of the portal system to reach the systemic veins. The clinical history of the disease does not indicate that this attempt at compensation is very successful. We may have: 1. Communications between the veins of the mesentery and of the abdominal walls. 2. Communications between the coronary vein of the stomach and the veins of Glisson's capsule on the one hand, and the phrenic veins on the other. 3. Anastomoses between the internal hæmorrhoidal and the hypogastric veins. 4. As pointed out by Baumgarten, enlargement of the not yet completely obliterated umbilical vein in the ligamentum teres. Through all these the blood may flow from the portal system into the veins of the abdominal walls—that is, in the reverse of the normal direction. In cases of portal obstruction the veins of the abdominal walls are often much enlarged, and this may be partly due to the changes just enumerated. In some instances the veins around the umbilicus have been strikingly tortuous and swollen; this condition has been termed "*caput Medusæ*."

The complications which sometimes occur are probably in part due to the same injurious influences as the cirrhosis. This is apparently true, for example, of the cardiac hypertrophy, the contracted kidney, and the chronic pachymeningitis. An interesting combination is the simultaneous occurrence of cirrhosis and chronic tubercular peritonitis. Various observers as well as ourselves have observed this with comparative frequency. The explanation is unknown. Probably cirrhosis is the primary lesion, and it promotes the development of peritoneal tuberculosis through the passive congestion of the portal system. Sometimes, however, the order of development seems to be reversed.

As to the general course of the disease, its duration can not easily be determined because the onset is usually insidious. As a rule, the disease lasts one to three years, or rarely longer. In many cases the symptoms are insignificant for the first six to eighteen months. Then the disorder takes on a severer form, perhaps rather suddenly. Ascites appears, for example. These graver symptoms persist, till after a few months the patient dies. The course of the disease reminds one of cardiac cases, where for a long while the compensatory changes avert any distress, till on a sudden the circulatory disturbances become pronounced and persist to the end.

The prognosis is always unfavorable, at least when the symptoms have once become well marked. It may be that in the earlier stages the disease can be checked or even permanently cured; but even this is open to doubt. No case recovers in which the diagnosis of cirrhosis is certain.

Death is due either to intercurrent disease, or more often to gradually increasing exhaustion. In some few cases severe cerebral symptoms suddenly appear: there are coma, general convulsions, and delirium; and these usually are soon fatal. Just how these nervous phenomena originate we do not certainly know (see the chapter on acute yellow atrophy of the liver).

Diagnosis.—The diagnosis is seldom very easily made. It becomes extremely probable if a patient who is known to have been addicted to alcohol gradually develops ascites and splenic tumor, and has a liver which presents distinct evidences of disease, such as an irregular surface. Often, however, we are left in doubt, because one or another of these more characteristic symptoms can not be clearly made out. Often a patient does not come under observation till a considerable ascitic effusion has already taken place, so that physical examination of the liver and spleen is rendered very difficult. Then we must first exclude any general disturbance of circulation as a cause of the ascites. If the heart, lungs, and kidneys are found to be normal, and if there is no œdema in the upper half of the body, it is very probable that there is a local derangement of the portal circulation; but we have still to determine whether the cause of this derangement is cirrhosis of the liver. This may be assumed to be the case if the whole course of the disease warrants the assumption, and if the history furnishes that most frequent of all ætiological factors, chronic alcoholism. Otherwise we must be cautious, for portal obstruction with precisely similar results may be due to other causes—like the external pressure of tumors or portal thrombosis. Many forms of hepatic syphilis (*q. v.*) can not be differentiated from cirrhosis by mere clinical observation of the hepatic disorder. Here it is only the ætiology and the demonstration of other signs of syphilis that can justify the assumption that the disease in hand is of specific origin.

It is also very difficult in many instances to exclude chronic peritonitis. The ætiology may aid us. Other points are, that in chronic peritonitis there may be tenderness on pressure, the abdominal distention is less uniform, and there is no enlargement of the spleen. The combination of cirrhosis and chronic tubercular peritonitis can not be diagnosticated with any approach to certainty unless we find both the symptoms of cirrhosis and pronounced indications of a tubercular affection. In such a case coincident pleurisy is significant of tuberculosis.

Treatment.—As we know the causes which sometime excite cirrhosis of the liver, an obvious prophylaxis consists in avoiding them; and, even after the early symptoms of the disease have appeared, alcohol should be forbidden, as well as spices and other similar articles, in the hope that we may thus do something to prevent the extension of the abnormal process.

If the disease has already made considerable progress, treatment becomes purely symptomatic. Iodide of potassium is said to exert a favorable influence upon cirrhosis of the liver, but its powers are doubtful. Probably it does good only in syphilitic hepatitis. Of the individual symptoms the results of portal congestion deserve the most consideration. It is important that the patient should enjoy complete physical rest, and that his bodily vigor should be promoted in every way possible. Even by these means we are sometimes able to diminish, or at least prevent the increase of, the ascites and other effects of portal obstruction.

Further remedies are given with the purpose (1) to deplete the congested portal system, and (2) by promoting the watery excretions to cause reabsorption of the ascitic effusion. Depletion is usually attempted with purgatives, the action of which is expected to diminish the high tension existing in the portal vein. The custom is an old one. In the milder cases, just developing, salines are recommended, usually in the form of mineral waters. The continued use of small doses of calomel (say 2 or 3 powders a day, each containing one half to one grain—0·03–0·05 grm.) seems to be sometimes advantageous. But if the ascites is already great, it is claimed that drastic remedies sometimes prove beneficial. Gamboge is reputed to be particularly appropriate in cirrhosis. We should not persist in the use of such drugs, however, if they disturb digestion.

The second indication of promoting watery discharges is fulfilled by diuretics. Besides the usual remedies of this class, like potassic acetate and squills, copaiba and its resin have been especially recommended for the various forms of ascites by English authors. The dose [of the resin] is about fifteen grains (grm. 1) daily, best given in capsules. In some cases this remedy has caused a rapid increase in the amount of urine and an accompanying diminution of the ascites. The improvement is not permanent, however. Good diuretic effects are sometimes obtained with calomel (*vide* p. 306).

If the ascites is so excessive as to occasion much local discomfort and to impede respiration, the removal of the fluid by paracentesis may afford relief. The details of this proceeding were given in the last section. Many physicians recommended tapping as early as possible, before it is absolutely necessary. The relief is said to be more lasting in that case; but, as a rule, the abdomen generally is quickly filled again. Possibly the application of an elastic bandage after the fluid is removed may retard its reaccumulation by the pressure thus exerted upon the abdominal cavity.

Special symptoms may sometimes demand attention. They are to be treated according to general principles.

CHAPTER V.

BILIARY AND HYPERTROPHIC CIRRHOSES OF THE LIVER.

THERE are two forms of cirrhosis which differ in many respects from the disease just described: they are called biliary cirrhosis and hypertrophic cirrhosis of the liver. Charcot and other French investigators were the first to call attention to them. Since then the literature of the subject has become quite considerable, but all doubts and differences of opinion are not yet settled. We shall try to state the most important points in what follows.

Whenever there is retention of bile in the liver for any length of time, no matter what causes it, certain changes result. The small and the medium-sized bile-ducts become distended, and granules of pigment are deposited, both in the interlobular connective tissue and within the acini themselves. Besides this, however, and undoubtedly because of the noxious influence of the retained bile, the hepatic cells undergo destructive changes. In accordance with the general rule, connective tissue gradually fills the gaps thus left in the parenchyma, and, more than this, the interstitial hyperplasia is so great as to increase the size of the organ.

If, therefore, there is persistent obstruction of the common duct by a gall-stone, or a cicatricial stenosis, or a tumor pressing from without upon the duct, the liver will, in all such cases, be found to be larger, firmer, and richer in fibrous tissue than normal—in a word, "cirrhotic." Hence this condition does not represent an independent disease, but is a result of chronic biliary retention, in whatever way occasioned. It is appropriately termed "secondary biliary cirrhosis." That retention is really the cause of this change has been proved by experiments, for it has been shown that ligature of the common duct in animals causes well-marked biliary cirrhosis.

This secondary cirrhosis is due to occlusion of the large bile-ducts. There is also a rare primary form of the biliary cirrhosis, usually known as hypertrophic cirrhosis. French authors have given it the name of "*cirrhose hypertrophique avec ictère*," out of regard to its most important clinical symptom. That there is an essential difference between this form and the ordinary "atrophic" cirrhosis of Laennec is manifested by the clinical behavior of the disease.

Often this disorder attacks hard drinkers, but they are not its only victims. While, in the common form of cirrhosis, ascites is usually the earliest grave symptom of disease, in hypertrophic cirrhosis a slight jaundice generally appears simultaneously with the first indefinite symptoms of pressure in the region of the liver, languor, and anorexia. This jaundice rapidly increases, and persists throughout the illness. In ordinary cirrhosis there may be, as we have said, hardly any jaundice, or, at any rate, it is a rather late symptom, and even then it is seldom extreme. On the other hand, ascites may be slight or absent in hypertrophic cirrhosis. It is true that there have been cases with great ascitic effusion, but it never comes till the disease is quite far advanced.

On physical examination the liver is usually found to be considerably enlarged, and its surface is smooth, as a rule, or rarely rough. In general there is said to be this important difference between the ordinary and the hypertrophic forms, that in the latter the newly formed connective tissue evinces little tendency to contraction, so that the liver remains large, even late in the course of the disease, and does not shrink. Somewhat too much stress has been laid upon this point. If, in many cases of hypertrophic cirrhosis, the liver has remained large to the end, this is probably in part due to an early death, before there was opportunity for much shrinkage. Cases that lasted longer have presented a contracted liver.

It must be said that the pathological appearance of the liver, particularly in the later stages of the disease, affords no certain evidence as to whether the cirrhosis was of the ordinary or of the "primary biliary" variety. Clinically, however, the two forms present such important diversities as to justify the distinction made. Of course, the clinical peculiarities of primary biliary cirrhosis must be due to some anatomical lesion. The most noticeable peculiarity of this sort is, that the development of connective tissue in hypertrophic cirrhosis is more active within the lobules than is the case in ordinary cirrhosis. Probably the hyperplasia in hypertrophic cirrhosis is most vigorous at first around the small biliary ducts, and thus leads to a retention of bile within the minutest biliary passages, with consequent jaundice, while the ramifications of the portal vein are not encroached upon till the process is far advanced. Whether these two forms of cirrhosis are merely modifications of one disease, or whether they are two independent disorders, is as yet unsettled. Certainly there are transitional forms.

As to the other clinical symptoms of primary biliary, or hypertrophic, cirrhosis we need say little. The most noticeable symptoms besides the hepatic enlargement and the jaundice are the effects of the jaundice itself—namely, digestive disturbances, slowness of the pulse, and nervous disorders. Of the occasional disturbances in the portal system, we have mentioned the ascites already. Still more frequent, and usually earlier in the time of its appearance, is chronic passive congestion of the spleen, with enlargement.

The entire duration of the disease is about one or two years; but it may last much longer. The prognosis is almost always bad. Occasionally a case will exhibit marked temporary improvement or an apparent arrest of the disease. Death comes as a result of gradual exhaustion, or is suddenly ushered in by coma, convulsions, and other grave nervous symptoms, usually ascribed to cholæmia (*vide infra*).

The diagnosis of hypertrophic cirrhosis can sometimes be made with considerable positiveness, and sometimes can merely be regarded as probable. The gradual development and persistence of jaundice and the presence of an enlarged liver and spleen, but usually without ascites, would suggest the disease strongly; but in some cases it is often impossible to exclude the existence of some mechanical obstruction in the larger biliary passages, such as gall-stones or new growths.

The treatment should conform to the principles laid down in the chapters on jaundice and ordinary cirrhosis of the liver. Sacharjin has lately recommended the frequent use of small doses of calomel—one grain (0·06) several times a day.

CHAPTER VI.

ACUTE YELLOW ATROPHY OF THE LIVER.

Ætiology.—Acute fatty degeneration of the liver occurs both as a primary disease and as secondary to other hepatic disorders, or as a symptom of constitutional diseases. Secondary acute fatty degeneration in rare instances accompanies severe acute infectious diseases, like typhoid fever, recurrent fever, septicæmia, and puerperal disease. It also appears, with equal rarity, in the course of cirrhosis of the liver or of persistent biliary retention; and it is a constant phenomenon in acute phosphorus poisoning. Indeed, the effects of phosphorus resemble the symptoms of primary acute yellow atrophy in many ways so closely, even post mortem, that formerly the two were often confounded.

Primary acute yellow atrophy of the liver is an extremely severe disease which almost invariably leads to speedy death. There is generally no determinable cause, and its victims are struck down in blooming health. It is so rare that not much over two hundred cases have thus far been reported. It is most common in young adult life, say between the fifteenth and thirty-fifth year. Children and elderly people have been occasionally attacked. Females are much more liable to the disease than males; and pregnancy increases the predisposition to it.

As we have said, we can not as a rule find any exciting cause. It is stated that sometimes the onset has been preceded by some violent emotional excitement, or excess in alcohol, or the like; but how important these factors may be is not at all clear.

It is an interesting fact that sometimes the disease becomes rather more frequent than usual, and endemic. For instance, several members of one family will be attacked. This favors a view as to the nature of acute yellow atrophy which a majority of the present investigators seem inclined to adopt. The view referred to is suggested not only by the whole course of the disease, but by the pathological appearances, and places it in the category of acute infectious diseases. It must be confessed that as yet we know nothing about the intimate nature of the infection. Klebs maintains that he has discovered micrococci in the hepatic blood-vessels; but thus far the observation lacks confirmation.

Pathology.—The chief change found post mortem is in the liver, and has determined the name given to the disease.

The liver is much atrophied, sometimes being only one half or one third its normal size. This makes its capsule often seem contracted and wrinkled. The organ is usually soft and flabby, so that in some places it seems as if the finger could be pressed into it. The color of the surface, and for the most part of the cross-section also, is yellow, like ochre or saffron; but the cut surface may be parti-colored, having red and yellow spots interspersed. Hence the names "red atrophy" and "yellow atrophy." The arrangement and relative extent of these patches may vary exceedingly. The red places look as if they had collapsed, and seem tougher than the yellow. They correspond, as we shall soon see, to the more advanced stages of the affection, while the yellow spots have undergone less change. The lobules are, as a rule, no longer distinguishable by the naked eye. Such lobules as can still be made out seem abnormally small and have a gray periphery.

On microscopic examination, we find that the essential change is an intense and uniform fatty degeneration of the hepatic cells, affecting the entire parenchyma. But few cells still retain their normal condition. The others are filled with large and small fat-globules, and many are already suffering evident disintegration and absorption. Where the degeneration is furthest advanced, fat, detritus, and pigment alone remain. Inasmuch as the lymphatics rapidly absorb and remove the fatty and albuminoid granules, there is finally little left except blood-vessels and connective tissue. The blood-vessels are frequently quite congested, and thus occasion that red color which the naked eye detects in the more advanced, broken-down portions. Frerichs made an interesting discovery, which deserves mention, of the existence of leucine and tyrosine crystals both in the parenchyma and in the blood-vessels. Bilirubin crystals also are sometimes found in the detritus, and more rarely in the interior of the hepatic cells.

Not only the liver, but many other organs present fatty degeneration: the heart in particular, the kidneys, and rarely the muscles; but the process is always most intense in the liver. The skin (*vide infra*) and most of the viscera are evidently tinged with jaundice.

Acute splenic tumor is invariably present. This suggests that the disease may be infectious. That the disease is a constitutional one is also to be inferred from the numerous ecchymoses in the skin and the interior of the body, especially in the mucous membrane of the stomach and intestines, in the serous membranes, in the pelvis of the kidneys, and the kidneys themselves, and more rarely in the brain and heart. This, again, is like the grave septic diseases. The blood itself is dark, with few clots. Leucine and tyrosine have repeatedly been detected in it. The peritoneum and other serous cavities sometimes contain a considerable amount of serum.

Clinical History.—The disease is usually divided into two stages, the first of which corresponds to the milder prodromal symptoms, the second to those severe symptoms which are alone characteristic. In many instances, however, the first period is wanting, or is so brief that the patient is plunged almost without warning into the gravest condition.

The prodromata in most cases consist of constitutional disturbances and mild gastro-intestinal symptoms. The patient is languid, without appetite, and disinclined to exertion. There are headache, nausea, vomiting, and sometimes moderate fever. In a few days jaundice usually appears. This is almost invariably taken for an ordinary catarrhal attack.

After some days, or it may be weeks, the second stage begins. The chief characteristic of this is the occurrence of grave nervous symptoms. First there is violent headache, with sleeplessness and marked restlessness. The intellect is usually somewhat dulled even now, and articulation is slow and clumsy. The mental confusion usually advances very rapidly to a noisy and violent delirium. The excitement becomes at time maniacal. The patient screams and storms, and can hardly be kept in bed. Often there are convulsive twitchings of individual muscles; and there may be typical epileptiform attacks, but this is not common. After one or two days, or rarely longer, the excitement abates, and is followed by sopor, which soon passes into deep coma. At death the patient is usually perfectly unconscious. It is exceptional for the excited stage to be wanting; in such cases the first nervous symptom is sopor.

The cause of the nervous symptoms has not yet been explained in a way to silence discussion. The same theories which have been set up to account for the grave form of jaundice in general (see appendix to this chapter) have also been employed to elucidate the nervous phenomena of acute yellow atrophy. Thus, some refer them to cholæmia, some to acholia, and still others to acute cerebral anæmia. It seems to us worth considering whether the cerebral disturbance in acute yellow atrophy of the liver may not be due to the constitutional infection (or intoxication) which we have seen to be so probable.

The jaundice, which is present even in the first stage, afterward usually deepens. The urine contains bile-pigment, and many investigators have also found bile-acids in it. If these latter are present, it suggests that the jaundice is not hæmatogenous—that is, the result of a destruction of red blood-corpuscles and the transformation of their pigment into biliary coloring matter—but is rather due to a retention of bile. Just how this retention arises we do not yet know for certain. The obstruction can not be in the large bile-ducts, for the gall-bladder is usually found empty. Hence the cause of the retention of bile and of the jaundice is probably a derangement of the smaller biliary passages within the liver. We should add that in a few rare cases there has been little or no jaundice.

On physical examination of the liver during the last stage of the disease, there is usually a striking diminution of hepatic dullness, corresponding to the atrophy of the organ. Generally the first change to be detected is a contraction of the left lobe, as shown by the development of tympanitic resonance in the epigastrium. At the commencement of the illness, the hepatic dullness is normal or slightly increased in area. If the disease proves very rapidly fatal, the organ may never become very small. In most cases, though by no means in all, there are pain and tenderness in the hepatic region, but these are seldom so great as in phosphorus poisoning.

The enlargement of the spleen has been already mentioned as an almost constant symptom of the disease. Even during life some increase of the area of splenic dullness can usually be made out, and sometimes the spleen can be felt under the edge of the ribs.

The occurrence of the hæmorrhages, which have already been referred to under the pathological lesions, can often be demonstrated during life. The cutaneous ecchymoses can, of course, be seen, and the hæmorrhages in the mucous membranes may give rise to hæmatemesis, bloody stools, bleeding from the female genitals, or epistaxis. The hæmorrhages are probably due to the impaired nutrition and diminished resisting power of the vascular walls occasioned by the grave constitutional disturbance.

The condition of the urine in acute yellow atrophy is very interesting. The amount is either normal or slightly diminished, and the specific gravity is somewhat increased. Often there is a trace of albumen. We have already mentioned the presence of bile-pigment. The point of chief interest, however, is one that Frerichs discovered and various others have since confirmed, and is the great diminution of urea and the appearance in its place of several other substances, which are likewise the products of the decomposition of albuminoid matter, and represent, in all probability, the first steps in the formation of urea. Of these substances, the most important are leucine and tyrosine. Their characteristic crystals can often be detected by the microscope in the urinary sediment (see Fig. 52). The crystals may also be obtained by allowing a drop of the fresh urine to evaporate slowly upon an object-glass. There is a chemical test for them which we can not here describe. There are some other abnormal constituents to be found in the urine besides leucine and tyrosine; but what their significance is we do not know. Among these are sarcolactic acid, oxymandel acid, peptonoid substances, and large amounts of kreatine.

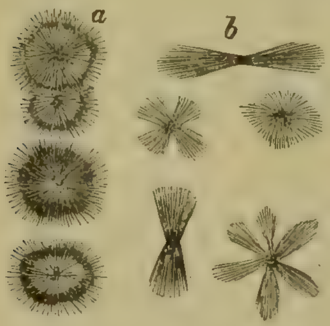


FIG 52.—*a*. Leucine crystals. *b*. Tyrosine crystals.

It at once suggests itself that this disappearance of urea and appearance of leucine and tyrosine, which are regarded as preparatory stages in the formation of urea, gives valuable support to Meissner's and Von Schröder's idea that this substance is manufactured in the liver.

As to the other organs little need be said. Vomiting is very frequent in the second stage, as well as in the first. It usually ushers in the severe cerebral symptoms. The stools are, as a rule, clay-colored, as is usual in jaundice. There is generally constipation. The pulse is rapid, often reaching 140 to 160 beats per minute, and is also small and compressible. It is this acceleration of the pulse, contrasting with its usual slowness during the first stage, which, along with the vomiting, announces the onset of dangerous symptoms. The pulmonary signs are seldom marked, although there may be bronchitis or a pneumonia due to the inhalation of foreign substances. During the coma which precedes death respiration is usually hurried, and often deep and noisy. Sometimes it is irregular.

The temperature is generally approximately normal. Toward the fatal termination there may be a subnormal temperature. Still more frequently the temperature rises before death, and even sometimes grows higher after death, reaching 107·5° (42° C.) or more.

In case the disease attacks a pregnant woman, abortion or premature delivery is almost certain to occur.

The entire duration of the disease depends mainly upon the length of the first stage. This may be entirely absent, or may be brief, or may occupy several weeks. The duration of the second stage, reckoning from the occurrence of grave cerebral symptoms, is generally only a few days (two to four), rarely a week. The termination is invariably fatal. In the few cases of recovery reported there is doubt about the correctness of the diagnosis.

The diagnosis can not be made till the second stage. The symptoms of the first stage are indistinguishable from those of simple catarrhal jaundice. With the development of the grave symptoms all doubt usually vanishes. The general course of the disease, the deep jaundice, the cerebral disturbances, the cutaneous ecchymoses, and the character of the urine, form a clinical picture resembling no other disease. The only cases where there can be any uncertainty about the diagnosis are the exceptional ones in which there is no jaundice. Acute phosphorus poisoning (*q. v.*) is in many respects very similar in its phenomena, but can generally be differentiated, even if the history of the case is not conclusive. The chief points are that in phosphorus poisoning the liver remains of large size for some time, and is very painful; that the nervous symptoms much less frequently assume the form of maniacal excitement; and that in but few cases is there any large amount of leucine and tyrosine in the urine.

After what has been said, we need hardly add that **treatment** is unavailing. Usually laxatives are employed, e. g., calomel. The nervous symptoms are combated by an ice-cap and baths and narcotics; the vomiting, by opium and bits of ice; and the cardiac weakness, by stimulants.

APPENDIX.

PERNICIOUS JAUNDICE. CHOLÆMIA AND ACHOLIA.

Reference has been repeatedly made in the preceding chapters to the possibility of the sudden supervention of grave nervous derangements in the course of various hepatic diseases. These nervous symptoms resemble one another so much in the different instances of their occurrence that we are forced to believe them always due to the same cause.

These symptoms are relatively most frequent where there is chronic biliary retention. Whether this retention be the result of obstruction of the common or the hepatic duct, or of stenosis of the biliary passages from a carcinoma involving the opening of the common duct into the duodenum, or that duct itself, the patient may quite suddenly fall into a condition which in many respects corresponds to the second stage of acute yellow atrophy just described. Grave cerebral disturbances declare themselves, with delirium, convulsions, and coma. There are hæmorrhages into the skin and into the mucous membranes, and in a few days the patient dies. Usually the end is attended with high fever. We have ourselves seen a temperature of 107·4° (41·9° C.) in a case of cancer at the duodenal extremity of the common duct. It is this group of symptoms which is usually termed pernicious jaundice; but almost precisely similar phenomena may suddenly appear in hepatic cirrhosis, when there is no great degree of jaundice, if any.

Just what produces these grave results in acute yellow atrophy and the other disorders just enumerated, we are not certain. Three theories have been propounded in explanation. The first theory, the latest champion of which is Leyden, attributes pernicious jaundice to cholæmia—that is, to the accumulation in the blood of the constituents of bile, and in particular of the biliary acids, as a result of absorption. This accumulation, it is said, is promoted by the paralyzing effect of the jaundice upon the activity of the kidneys. Opposed to this theory is the fact that these same symptoms may occur where there is no marked hepatogenous jaundice.

Traube has suggested that as a result of the great impairment of nutrition there is a cerebral anæmia, which in its turn brings on the nervous attacks. Cohnheim also advocated this view, with some modifications.

The view which we are most inclined to accept is that of Frerichs. He has given to the group of symptoms under discussion the name of acholia. These symptoms he ascribes to the pernicious influence of those substances which under normal conditions are manufactured by the liver into bile, but which in such cases accumulate in the blood and the tissues. As Frerichs himself has said, and we believe very justly, this view should be extended to include all other transforming functions of the liver, and especially the production of urea. It is also very possible that in addition to the acholia, cholæmic poisoning may sometimes exert a simultaneous influence.

The termination of cholæmia or acholia is almost always as unfavorable as that of acute yellow atrophy. In cases of this kind there is usually marked fatty degeneration of the hepatic parenchyma to be found post mortem.

CHAPTER VII.

ICTERUS NEONATORUM.

(*Jaundice of the New-born.*)

FREQUENTLY the normal red color of the skin in children changes on the second, third, or fourth day after birth to a distinctly yellow, jaundiced hue. The yellow tinge is deeper on the face and trunk than on the extremities. There are no special digestive or constitutional disturbances. Still it may be taken for the rule that weaklings more often present this jaundice than do vigorous babes. The abnormal hue is almost certain to vanish in a week or two, and leave no sequelæ. The termination is unfavorable in those instances alone where there is some special complication, not directly connected with the jaundice as such.

The ætiology is a disputed matter. A large number of all sorts of theories have been set up, no one of which to this day has gained universal acceptance. Formerly there was considerable tendency to regard the jaundice as hæmatogenous—that is, due to the transformation of the pigment of broken-down blood-corpuscles into bile-pigment. Points which seemed to support this view are the light (not jaundiced) color of the urine and the yellow color of the stools (from bile). But more accurate examination has shown that the urine does contain biliary pigment, as do also the kidneys of such infants as happen to die during the existence of the jaundice; and the biliary acids have been clearly shown to be present in the serous transudations. It may therefore be considered certain that icterus neonatorum is hepatogenous; but how the biliary retention and consequent absorption are caused we do not know. Perhaps at first the bile is not ejected properly, from weakness, or the ducts may be narrow, or temporarily plugged by desquamated epithelium. Birch-Hirschfeld has called attention to the fact that after birth there is a tendency to considerable passive congestion of the liver, with resultant œdema of Glisson's capsule and pressure upon the interlobular bile-ducts. We should also consider, as Hofmeier points out, that probably during the first few days of extra-uterine life there is a comparatively large amount of bile secreted, occasioned by the destruction in considerable numbers of the red blood-globules.

It is well to mention in conclusion that in very rare instances there is complete congenital stenosis, or even entire absence, of the larger bile-ducts. Then deep jaundice comes on soon after birth, and is persistent. The children become extremely emaciated, and, after a few weeks, inevitably perish.

CHAPTER VIII.

SYPHILIS OF THE LIVER.

Ætiology and Pathology.—Syphilitic disease of the liver occurs both when the syphilis is acquired and when it is congenital. Congenital syphilitic disease of the liver may be diffuse or localized, and causes a cellular infiltration in either case. If the changes are extensive, the organ is hard and considerably enlarged; or, if the newly formed connective tissue has undergone contraction, the liver is smaller than normal, and its surface is uneven. In some cases of hereditary syphilis, distinct gummata of considerable size have been observed.

In acquired syphilis, hepatic disease is one of the so-called tertiary symptoms, and does not usually develop, at least to any great extent, until several years after infection. Indeed, it may be a very late result. It may take the form either of a diffuse syphilitic hepatitis, or of circumscribed gummata or syphilomata. The diffuse hepatitis does not present essentially different anatomical appearances from those of ordinary cirrhosis. The gummata are the most characteristic, and the most important clinically. They may form separate tumors the size of an apple or even larger. The convex surface of the organ, particularly that portion near the suspensory ligament, seems to be a favorite location for the new growth. The same is true of the porta hepatis, where Glisson's capsule enters the liver. At the autopsy the gummata have in most cases already begun to undergo contraction. If so, the liver is usually smaller than normal, and traversed in various directions by deep furrows, which divide it into lobes. These furrows are due to firm cicatricial bands, among the fibers of which we may sometimes find necrotic and cheesy vestiges of the gumma proper. Often there is evident syphilitic endarteritis in the smaller and sometimes also in the larger branches of the hepatic artery and portal vein.

Clinical History.—Circumscribed syphilitic changes in the liver often give rise to no symptoms whatever. It is only when the disease comes to disturb the portal circulation that a series of symptoms result, which, for evident reasons, may be analogous in all essential points to the effects of ordinary cirrhosis. Whenever the syphilitic growths contract so as to obliterate a large number of branches of the portal vein, or whenever a gumma happens to be so situated as to compress the trunk of the portal vein itself, then the well-known results of portal obstruction are inevitable, the chief being ascites and enlargement of the spleen. The disturbance of circulation often gives rise also to anorexia and digestive disorders. Experience shows jaundice to be rare in hepatic syphilis, but it may appear when the lesions involve the larger bile-ducts or a considerable number of the smaller biliary passages.

On physical examination, the results vary according to the form and the stage of the disorder. Sometimes the larger gummata may be plainly felt through the abdominal walls, usually as flattened hemispheres. Frequently, also, we can feel the edge of the enlarged organ, and can detect that the edge is less sharp than normal. In other instances the separate elevations and prominences can be made out. The area of dullness on percussion of course varies in different cases.

It deserves mention that hepatic syphilis quite often causes severe pain, sometimes over the entire region of the liver and sometimes in just one spot. Pain is by no means felt in every case. With the pain there may be great tenderness on pressure.

The course of the disease is usually tedious, and may occupy many years. Probably, too, lesions exist in many cases long before there are any symptoms. Just as in cirrhosis, ascites is usually the first thing to attract the patient's attention. Improvement and temporary arrest of the disease are more frequent than in ordinary cirrhosis. Still, in most cases, where the lesions are at all extensive, the termination is unfavorable.

The **diagnosis** is not always easy. Usually the objective changes in the liver, the ascites, and the enlarged spleen, indicate hepatic trouble, but we are often unable to determine just what the trouble is. Naturally, the ætiological factors are of great importance. In a toper we would think rather of the common form of cirrhosis. If there is a syphilitic history, or if we find scars in the throat, irregularities in the surface of the bones, or other signs of a specific dyscrasia, we would naturally ascribe the hepatic disorder to the same cause. As to special signs, if the prominences on the liver are rather large in contrast to the smaller granulations of common cirrhosis, syphilis is somewhat more probable. Severe pain in the right hypochondrium also suggests syphilis rather than cirrhosis. It should also be considered that the course of hepatic syphilis is usually much more protracted than that of ordinary hepatic cirrhosis.

Treatment.—Whether we feel certain that syphilitic hepatitis exists, or merely suspect it, specific treatment should be tried. Mercury and potassic iodide should both be given, but probably the iodide is the more valuable of the two. But these remedies can be successful only when the gummata are still in process of formation. Our therapeutic efforts produce no impression upon the cicatricial bands, the contraction of which is the main cause of derangement. Hence we see why the results of treatment in advanced cases are seldom very favorable.

For symptomatic treatment, the reader is referred to cirrhosis of the liver.

CHAPTER IX.

CANCER OF THE LIVER AND BILE-DUCTS.

Ætiology and Pathology.—Primary cancer of the liver is very rare, but secondary or metastatic cancer of this organ is met with comparatively often. The chief explanation of this latter fact is the slowness of the blood-current in the liver, which favors the deposition of the cancerous germs suspended in the blood.

Secondary hepatic cancer may be a sequel to primary cancer of any organ. It is most often seen, however, when the primary growth lies within the portal system, in the stomach, intestines, rectum, œsophagus, or pancreas. In some instances the projection of the primary growth into the lumen of a branch of the portal vein has been directly demonstrated, thus furnishing an obvious source for metastasis. The secondary cancers in the liver may be very numerous. They are found both within the organ and upon its surface. If superficial, they form flattened protuberances, which are often dimpled in the middle. If the new growth is extensive, the liver may be greatly enlarged, so as to occupy a great part of the abdominal cavity.

As we have said, primary cancer of the liver is very unusual. It may occur either in the form of separate large nodules, or as a more diffuse cancerous infiltration pervading the greater part of the organ. Histologically considered, the primary growths are of cylindrical-cell carcinoma, apparently originating from the epithelium of the minute bile-ducts, but also, according to some authors, sometimes starting from the cells of the parenchyma.

Primary cancer of the larger bile-ducts is of more frequent occurrence than genuine primary hepatic cancer, and therefore it is of more importance clinically. The gall-bladder may also be the seat of primary carcinoma. From these sources may proceed abundant metastatic growths in the liver itself.

As to the ætiology of hepatic cancer we can be brief. The disease is most frequent in advanced life, from forty to sixty, following in this the general rule for cancer. Special causes are not known. It sometimes seems possible to trace a hereditary predisposition to it. In many cases gall-stones seem to start up the development of carcinoma.

Clinical History—Diagnosis.—Many small nodules of cancer, as well as large masses which are favorably situated, may exist in the liver without exciting any symptoms. If there is an undoubted primary cancer in another organ, such as the stomach, we must always remember the possibility of metastatic growths in the liver; but they can not be proved to exist, unless they alter appreciably the size or shape of the organ. Sometimes they may be inferred, when we observe either ascites and enlargement of the spleen from pressure on the portal vein, or jaundice from pressure on the bile-ducts.

On palpation, we are often able to make out one or more tumors plainly in hepatic cancer. These tumors are in the region of the liver, and they are directly connected with it, as we can prove by marking out its limits by percussion and palpation. Another characteristic sign is that almost all hepatic tumors can be felt to move with respiration, on account of the inspiratory depression of the diaphragm pushing down the liver and all that is joined to it. Percussion over a hepatic tumor almost invariably gives flatness, in contrast to the muffled tympanitic resonance of many gastric tumors.

The most characteristic condition is not a very rare one; in it the liver is the seat of a very large number of cancerous nodules. In such cases the organ is usually much enlarged. Often we can detect by mere inspection a great, irregular prominence in the hepatic region, pressing forward the flabby, atrophied walls of the abdomen, and reaching down to the level of the umbilicus, or even lower. On palpation, we can feel most of the anterior surface of the liver, and the separate cancerous nodules scattered over it. These usually are as large as walnuts, or even apples. The lower or anterior margin of the liver can often be made out plainly, and it also is often the seat of nodules; and we can sometimes feel nodules on the lower surface of the organ.

The other clinical phenomena in hepatic cancer have several causes: (1) The primary disease, such as cancer of the stomach; (2) the general cancerous cachexia, as shown by languor, emaciation, and possibly a slight œdema of the ankles; and (3) the possible compression of the blood-vessels or bile-ducts. This compression is not infrequent, and produces a moderate or even a large ascitic effusion. Even in these instances the spleen is seldom much enlarged as a result of the passive congestion, because the universal emaciation and anæmia counteract the tendency to increase in size. Jaundice is relatively more frequent in cancer of the liver than is ascites. It is caused by compression either of the hepatic duct or of the minuter bile-ducts. On the other hand, however, we can easily see that hepatic cancer may exist without producing either icterus or ascites.

The differential diagnosis of hepatic cancer from cancer in other organs is sometimes extremely difficult. This is particularly true of pyloric cancer, and especially so when the pylorus has become adherent to the liver, which often is the case. Cancers of the omentum and of the colon sometimes simulate hepatic cancer, but they seldom move so decidedly in respiration as do hepatic tumors. Given a new growth in the liver, it is usually comparatively easy to distinguish between carcinoma and other tumors. The benign growths, like adenoma, are so rarely found in the liver that they can actually be almost disregarded. If there are syphilitic growths, we may be aided by the history of the case and other signs of syphilis, or by the eventual contraction and atrophy of the organ. Echinococci have, as a rule, a much more regular shape, like a flattened sphere. Large abscesses are rare in our latitudes; and if they occur, usually the ætiology is significant. They also frequently cause fever and rigors, which cancer does not.

When we have decided that cancer of the liver is present, the question arises, Is it primary or secondary? In the first place, primary cancer here is so rare that the probabilities are in favor of a secondary growth. Not infrequently the primary tumor can not be detected during life. Thus a small cancer of the stomach, or a flat œsophageal cancer, or carcinoma of the pancreas, are all easily overlooked. If we find many nodules in the liver and no primary trouble elsewhere, then there comes the possibility that there is primary cancer in the gall-bladder or the bile ducts. In rare instances palpation may discover the gall-bladder in a state of cancerous degeneration close underneath the liver; but usually the viscus will be small and contracted, and the flat and ulcerating growth is not noticeable, except from the inside. It is particularly in those cases of hepatic cancer where there is great and persistent icterus, and no evidence of carcinoma in any other organ, that we should be most apt to think of primary cancer of the bile-ducts.

The duration of hepatic cancer is usually not prolonged. The first decided evidences of its existence no sooner present themselves than marasmus and cachexia rapidly develop. The fatal end comes in a few months, or at latest within a year.

The prognosis is hopeless. Treatment can avail only to alleviate somewhat the patient's sufferings.

CHAPTER X.

ECHINOCOCCUS OF THE LIVER.

Natural History and Pathology.—Inasmuch as it is the liver which suffers most frequently from invasions of the echinococcus, we will here state the main general points relative to the troubles produced by this parasite.

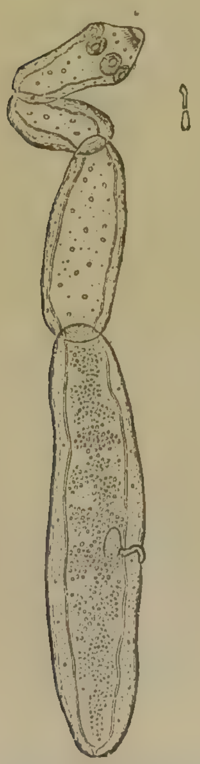


FIG. 53.—(From HELLER.) Tænia echinococcus, enlarged. Above, at the right, echinococcus, of natural size.

The *tænia echinococcus* (see Fig. 53) is a small tape-worm about four millimetres long, and composed of three or four joints. It inhabits the intestinal canal of the dog. Man becomes infected by the ingestion of the eggs of this tapeworm into the stomach. The striking prevalence of the disease in Iceland is explained by the fact that the inhabitants live in constant contact with their numerous canine friends. Among us the echinococcus is comparatively rare.

If a human being has become infected, the blood-current carries the embryo into some organ. In a great majority of cases it passes through a branch of the portal vein into the liver and there fastens itself; but the echinococcus may be developed in other organs—like the lungs (*vide* page 252), the bones, the brain, and the kidneys. A hydatid cyst develops from the embryo, and is filled with a non-albuminous fluid. The cyst is composed of an external cuticle of lamellated structure, and an inner, parenchymatous layer, which contains muscular fibers and blood-vessels. Surrounding the cyst, as it lies in the infested organ, there is gradually developed a thick capsule of connective tissue.

After the cyst has continued its growth for some four to six months, being now about the size of a walnut, there are generated upon the inner surface of the capsule, from the parenchymatous layer, so-called breeding capsules, containing numbers of echinococcus-heads, or "*scolices.*" Each scolex has four suckers and a circlet of hooks. It can draw itself into the breeding capsule and also produce a prominence upon the outer surface of the latter (see Figs. 54, 55, and 56).

Usually the primary cyst gives rise to secondary "daughter vesicles," and these to "granddaughter vesicles." Some of these are formed in the cuticle, others from the breeding capsules. In man they generally grow inward—that is, are endogenous (*echinococcus hydatidosus*)—and finally become detached. Hundreds of them may sometimes be found free in the liquid contents of the cyst. In animals the daughter vesicles are more often exogenous (*echinococcus veterinorum seu granulosus*). A peculiar kind of echinococcus, which was formerly regarded as a kind of new growth, is that called by Virchow *echinococcus multilocularis*. This gives rise to a hard tumor, which is composed of vesicles the size of a pea, and which seems to grow along the lymph-vessels, and possibly in the blood-vessels also.

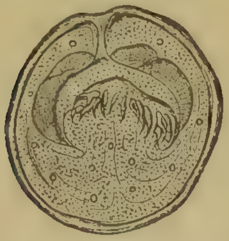


FIG. 54.

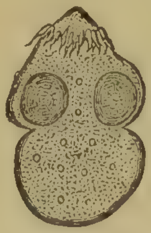


FIG. 55.

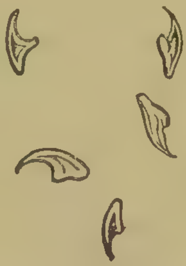


FIG. 56.

FIGS. 54 and 55.—(From HELLER.) Echinococcus scolices, free, drawn in and turned outward.
FIG. 56.—Echinococcus hooklets.

The growth of a hydatid cyst is slow, and may continue for years. It may finally attain the size of a child's head. At last, however, the echinococcus dies. The cyst then undergoes considerable though gradual contraction, and both walls and contents become calcified.

Clinical Phenomena.—As long as the cyst in the liver retains moderate dimensions, there is usually no discomfort. Frequently the cysts perish and become calcified, without having ever attracted attention, and are found post-mortem merely by accident.

If the cyst becomes very large, it causes a sensation of pressure and pain in the hepatic region. In rare instances, unusually large cysts, if situated on the convex surface of the liver may crowd up the diaphragm so as to compress the lower portions of the lung and induce dyspnœa. Again, the cyst may be so situated as to compress the portal vein or a large bile-duct. Then appear ascites and enlargement of the spleen, or jaundice, as the case may be.

It is an important fact that sometimes the cyst ruptures and discharges its contents into neighboring parts. Thus in repeated instances the pleural cavity has been invaded; also the lungs, as evidenced by the expectoration of vesicles; the intestinal canal, with the appearance of vesicles in the stools; the bile-ducts, followed by jaundice and the eventual appearance of vesicles in the intestine; and the vena cava inferior, causing sudden death from pulmonary embolism. Sometimes the cyst pushes outward through the abdominal walls, and terminates in recovery. Exceptionally, the echinococcus-sac undergoes purulent inflammation, with all the symptoms of a hepatic abscess.

A multilocular echinococcus usually excites grave disturbance. The liver is decidedly enlarged, and generally is firm and smooth, not uneven, to the touch. As a rule, there are jaundice, swelling of the spleen, and ascites, and accompanying these a gradual loss of flesh and strength, ending fatally.

A diagnosis is sometimes easily reached if the cysts can be felt upon the surface of the liver. Usually the tumors are flat or globular, and of firm consistence, though often evidently elastic. A peculiarly characteristic sign, though one which is seldom obtainable, is the "hydatid thrill." It is felt upon giving the tumor a quick, short blow with the flat of the hand. The diagnosis is certain if, in any way, echinococcus vesicles are discharged. Aspiration for purposes of diagnosis has been repeatedly performed. The fluid thus evacuated is light-yellow, almost always non-albuminous, and, upon microscopic examination, sometimes presents fragments of the lamellated membrane, or some of the hooklets. But if we do not find these morphological elements, we are by no means warranted in excluding echinococcus. Chemically, the fluid should yield sugar and succinic acid. This fact may perhaps aid diagnosis.

Frequently it is difficult to distinguish between an echinococcus of the liver and other hepatic disorders. We may need to consider all the circumstances—causation, age, fever, shape of the tumor, or perhaps the results of an exploratory puncture. Large cysts, which crowd themselves upward into the pleural cavity may simulate a pleuritic effusion.

Treatment.—The administration of internal remedies is extremely unreliable. Iodide of potassium and mercury are especially recommended. It is, however, operative interference alone that holds out any promise of success; but this has its dangers, and should, therefore, be held in reserve till the symptoms become grave or very distressing. For details of the numberless methods of operation which have been proposed, we would refer to works on surgery; but we may mention here that simple aspiration of the contents of the cyst sometimes affords permanent relief; the cyst becomes obliterated, and there is complete recovery. In a few cases, after the sac has been emptied, tincture of iodine has been injected. Most of the other methods aim (1) to bring about adhesion of the sac to the abdominal walls, and (2) to lay it open and evacuate the contents. At the surgical clinique in Leipsic the favorite and a very satisfactory method is to employ a caustic paste, made with chloride of zinc, which slowly effects an opening of the cyst, and, by the adhesive inflammation it excites previously, fixes the cyst to the abdominal walls. Simon's method is to thrust in several trocars, at intervals from one another, and allow them to remain till adhesions are formed. Then the points of puncture are united by an incision, the sac emptied, syringed out, disinfected, and allowed to heal gradually.

[The experience of Australian surgeons with hydatid disease is very large, and Thomas believes that the cure, after tapping, is often illusory, and that failure follows in at least forty per cent. of the cases. He has collected sixty-eight cases of hydatid diseases treated by laparotomy, with only seven deaths. In twenty-one cases treated as in Leipsic (Volkmann's method), the mortality has been nineteen per cent.]

CHAPTER XI.

CIRCULATORY DISTURBANCES IN THE LIVER.

1. Hepatic anæmia is seldom extreme except in cases of profound general anæmia, and has no clinical importance, so far as we are aware.

2. Passive congestion of the liver is of frequent occurrence and is of importance. It may arise in any disorder which disturbs the systemic circulation. It is oftenest seen in connection with heart disease, particularly mitral disease. It also follows pulmonary emphysema and chronic processes which result in contraction of the lungs. The liver is enlarged and engorged. The hepatic veins being situated in the center of the lobules, this central portion becomes darkly pigmented, while the periphery of the lobules seems lighter colored. The peripheral cells may even appear distinctly yellow, from a fatty infiltration which is not infrequent. In this way the cut surface comes to present that variegated appearance which has led to the name of "nutmeg liver." If the venous stasis be per-

sistent, there is considerable atrophy of the hepatic parenchyma, involving especially the cells near the center of each lobule. Thus the liver atrophies, and its surface may become slightly granular. This is the "atrophic nutmeg liver."

The clinical phenomena are chiefly those due to the hepatic enlargement. If chronic cardiac disease, emphysema, or some analogous trouble has occasioned congestion of the liver, the area of hepatic dullness is increased, and frequently we can feel the edge or even a portion of the anterior surface. In pronounced cases the organ may extend almost a hand's breadth below the ribs. Often there is a slight jaundice. Sometimes it is quite marked. It is probably due to the distended blood-vessels compressing the smaller bile-ducts. We have already mentioned how characteristic, in many cardiac cases, is a complexion presenting both cyanosis and jaundice.

Quite often the congestion, if great, produces subjective disturbances. There is a feeling of pressure and weight in the hepatic region; and if the capsule of the organ is tightly stretched, there may be actual pain.

The prognosis and treatment depend, of course, upon the primary disorder.

3. About active hyperæmia of the liver we have little definite information. Formerly there was a great deal said about it, as one of the conditions in "abdominal plethora." Active hyperæmia is most frequently assumed to exist in case of those who are good livers and of sedentary habit. In such, we are told, the temporary physiological hyperæmia which attends digestion passes on into a permanent congestion of the liver. Thereby the organ is enlarged, there are painful sensations in the right hypochondriun, digestive disturbances, and occasional slight jaundice. The abnormal condition just described is certainly often met with in practice, but it would seem hardly possible to draw a clear dividing-line between active hyperæmia of the liver and other disturbances which give rise to similar symptoms. Such are chronic gastric and intestinal catarrhs; cardiac hypertrophy and functional cardiac derangement, with passive congestion of the liver; fatty liver; and incipient cirrhosis.

A prominent factor in the production of active hyperæmia of the liver is also ascribed to the ingestion of such matters as are said to "irritate" the liver, like the various spices, coffee, and, above all, alcohol.

It should also be noted that the liver may be much engorged in many acute infectious diseases, particularly in pernicious malarial diseases and in typhus or typhoid fever.

It is also maintained that the hyperæmia may result from the cessation of hæmorrhages elsewhere, such as the catamenia or bleeding from hæmorrhoids. The facts that have been brought forward to sustain this view are none of them conclusive. We will mention that the "menstrual jaundice" which occasionally appears when the menses are scanty or absent has been referred to a vicarious hyperæmia of the liver.

It is, of course, impossible to make general statements about the course and duration of active hyperæmia of the liver. The treatment of the first variety mentioned—namely, that arising from an improper mode of life—demands careful regulation of the diet, abundant exercise in the open air, like horseback-riding, and laxatives. We may give rhubarb, aloes, or a course of the waters at Carlsbad, Marienbad, Kissingen, or Homburg.

CHAPTER XII.

ATROPHY, HYPERTROPHY, AND DEGENERATIONS OF THE LIVER.

1. **Simple Atrophy of the Liver.**—Simple atrophy is not of rare occurrence, being seen in senile marasmus, and in malnutrition from almost any cause. The degree of atrophy varies. The borders of the organ are much wrinkled. The lobules seem decidedly smaller than normal, and even the individual cells that still remain are atrophied and also usually deeply pigmented.

The condition does not of itself give rise to any special symptoms. The area of hepatic dullness is usually lessened, but this sign is too ambiguous ever to justify us in making from it a diagnosis of hepatic atrophy. Perhaps there is some value in the alleged lighter color of the stools, as indicating a diminished secretion of bile.

2. **Hypertrophy of the Liver.**—Even under normal circumstances the liver undergoes quite marked alterations in size. The exact point, therefore, where an abnormal hypertrophy begins can not be set. Sometimes the autopsy reveals an unusually large liver, of which there had been no indications during life, and for which no cause can be made out.

There are certain diseases in which enlargement of the liver is found with comparative frequency: diabetes mellitus, chronic malarial poisoning, leukæmia, and sometimes rachitis. Topers quite often have enlarged livers, which as a rule present simple hypertrophic changes. Occasionally a liver has been reported as showing spots of localized hyperplasia, which may form flattened prominences upon the surface of the organ.

Hypertrophy is to be diagnosticated only when palpation and percussion give proof of an enlargement, and yet amyloid, hypertrophic cirrhosis, and other diseases which cause an increase in the size of the liver, can be excluded. The ætiology of the case should also be considered.

3. **Fatty Liver.**—This name is applied to excessive, diffuse, fatty infiltration of the hepatic cells. The size of the organ is increased. It is firm, anæmic, and of a uniform yellow color, both externally and upon section. The microscope shows that the cells of the parenchyma are filled with large and small globules of fat. The fat is most abundant toward the periphery of the lobules.

The causes of fatty liver are by no means clear. Sometimes it is found in cases of general obesity, where we may assume that the amount of fat which the liver receives as nourishment is abnormally great; but often we find a liver that contains comparatively little fat in those who have a well-developed panniculus adiposus and much fat in other organs. Topers may have a decidedly fatty liver. The occurrence of fatty liver in the cachectic, and particularly in the consumptive, is remarkable; and individuals suffering from cancer, or marantic children, may also exhibit the same change. We have no intimate knowledge of the conditions that prevent, in such cases, the oxygenation of the fat which comes to the liver from the ingesta or from other organs.

We do not know that the fatty liver is in any way functionally impaired. The only clinical indication, therefore, of its existence is the increased bulk of the organ. In phthisis we may sometimes feel pretty certain that the liver is fatty, if an increase in bulk can be demonstrated, and if other causes for this enlargement, like amyloid, appear improbable. If the anterior edge of a fatty liver can be felt, it is usually found to be noticeably thick and blunt.

The treatment of fatty liver is to combat the original disease.

4. **Amyloid Liver** (*Waxy Liver*).—Amyloid degeneration of the liver is almost invariably a part of extensive amyloid disease, involving also the spleen, kidneys,

intestine, and other organs. The disease occurs chiefly in certain cachectic conditions, such as chronic suppuration, as in caries and persistent empyema, and also in chronic pulmonary tuberculosis, and constitutional syphilis.

The amyloid liver is usually increased in bulk. The organ may even become almost double its normal size. It feels very firm and hard, its surface is perfectly smooth, and its edge is slightly thickened. The cut surface presents a characteristic grayish-brown "waxy" appearance.

The microscope shows that the degenerative process attacks chiefly the walls of the hepatic capillaries, the hepatic cells proper showing infrequent and slight amyloid changes. Very often the cells of the parenchyma are atrophied and somewhat infiltrated with fat.

The diagnosis of amyloid liver requires (1) the demonstration by palpation and percussion of hepatic enlargement. We can often feel a large part of the anterior surface and the margin of the hard and firm organ. The liver may reach as low as the level of the umbilicus. The diagnosis further demands (2) that some disease which predisposes to amyloid be present, and (3) that there be evidence of the degenerative process in other organs: the spleen should be enlarged, and the kidneys secrete albuminous urine.

The other symptoms, as well as both prognosis and treatment, are determined mainly by the nature of the causative affection. About amyloid disease in general see the chapter on amyloid kidney (page 864).

CHAPTER XIII.

ANOMALIES IN THE SHAPE AND POSITION OF THE LIVER.

1. **Corset Liver.**—The constant pressure of the lower ribs against the liver, as a result of tight lacing, often produces an atrophy of the hepatic parenchyma from pressure, as shown by a deep furrow crossing transversely the anterior surface of the organ. This "corset furrow" lies chiefly in the right lobe. Its usual situation corresponds to the margin of the ribs, and the atrophy may be so extreme that the liver is divided into a large upper part and a small, usually roundish, lower portion, connected by a narrow isthmus of tissue. At the atrophic place, the connective-tissue capsule of the liver is almost always much thickened. Often the lower section can be bent upward as if attached by a hinge.

This deformity of the liver is found quite often in elderly females, and rarely in men, as in soldiers. Unless extreme, it can not be detected during life, and causes no discomfort. Even the bad cases do not, as a rule, occasion any special symptoms; but they can be clearly made out if the abdominal walls are lax. The deep transverse furrow can be felt, and also the lower section, with its usually blunt edge. Particularly in the case of old women we must bear this condition in mind, else we might easily confound it with some enlargement of the liver, like amyloid or passive congestion, or even new growths.

In not rare instances there are clinical symptoms. A constant sensation of pressure and pulling is felt in the hepatic region; and sometimes, as a result of venous stasis, there is a temporary but decided swelling of the isolated portion, and possibly violent pain and indications of irritation of the peritoneum, such as vomiting and an approach to collapse. Usually rest in bed and cold applications give speedy relief; but relapses are possible.

2. **Movable Liver.**—A movable or "wandering" liver is of very rare occurrence, and has thus far been seen only in women. Its cause is not perfectly deter-

mined. Probably the suspensory ligament is abnormally long. The liver does not occupy its usual position, but lies deep in the lower part of the abdomen. Here it can be plainly felt, and, by external pressure, it can generally be brought back into its normal place with considerable ease. It is always abnormally movable, and can be shown to change its position when the patient changes from one side to the other.

In most cases a movable liver causes considerable discomfort, particularly pain and digestive disturbances. The only way of affording relief is by applying a bandage which may maintain the organ in its proper position.

CHAPTER XIV.

SUPPURATIVE PYLEPHLEBITIS.

(Purulent Inflammation of the Portal Vein and its Branches.)

Ætiology.—Purulent pylephlebitis is seldom a primary, idiopathic disease. In most instances it is due to the propagation of a suppurative inflammation of neighboring tissues to the walls of the vein. The main trunk of the portal vein is rarely directly attacked. Usually the process originates in the hepatic branches of the vein or in the veins of the portal system, and thence extends to the larger vessel.

Perityphlitic abscess is the most frequent source of suppurative pylephlebitis. The inflammation involves a mesenteric vein, and thence extends upward. Other causes are gastric ulcer, intestinal ulcers, as in dysentery, splenic abscess, and purulent inflammation at the porta hepatis or within the liver itself, as in abscess due to gall-stones. The mode of production in these cases is precisely analogous to that in perityphlitic abscess; but they are rare.

A special form of pylephlebitis is observed in the new-born. Here the inflammation originates in the umbilical vein, and we need hardly say that the cause is a septic infection through the navel.

In rare instances it has been found that pylephlebitis has resulted from the penetration into a vein of some foreign body that had been swallowed, such as a pin. Here, too, the true factors in producing the inflammation are, of course, the bacteria which adhere to the foreign body.

Pathology.—Where the inflammation has attacked the vascular walls, the vein is thickened, and often the surrounding connective tissue is infiltrated with pus-cells and mottled with minute ecchymoses. If the vein is cut open, the intima is seen to be opaque and often superficially ulcerated. The lumen of the vessel is filled with a thrombus, which is usually to a great extent in a state of purulent softening, so that offensive purulent or sanious fluid flows out. The course of events is as follows: First, the wall of the vein becomes inflamed. As a consequence of this, a thrombus forms at the same place. The bacteria penetrate this thrombus and occasion its purulent softening.

The extent of a pylephlebitis naturally varies in different cases. As a rule, little fragments become detached from the thrombus and enter the liver, producing metastatic abscesses. Secondary suppuration may also occur in the lungs, kidneys, brain, and joints, so that we have all the anatomical characteristics of a general pyæmia.

Clinical History.—Inasmuch as the primary, causative disease may be very different in different cases, it is impossible to delineate the disease comprehensively. It is, however, frequently ushered in by a number of symptoms, which render a diagnosis possible, at least in some cases, if the original disease has been recognized.

The symptoms of suppurative pylephlebitis are in part due directly to the local disease itself, and in part are occasioned by the general pyæmia. One of the local symptoms is pain in the epigastrium. This is rare. It may radiate downward or laterally, according to the starting-place and extent of the inflammation. An inevitable result of the portal thrombosis is portal obstruction. The spleen becomes considerably swollen, and, if the disease be not too quickly fatal, there is an evident effusion into the peritoneal cavity. The splenic enlargement can not be regarded as due merely to venous stasis, but is in part the "acute splenic tumor" of constitutional septic conditions. If the inflammation spreads from the branches of the portal vein to the neighboring bile-ducts, jaundice results. This is seen quite often. Sometimes it is also due to the hepatic abscesses, or to a gall-stone which happens to cause trouble simultaneously. Now and then there is no jaundice whatever.

Of the pyæmic symptoms, hepatic abscesses come first. They are due, as we have said, to the conveyance of septic matter directly into the liver by emboli. The one almost constant sign of their occurrence is a decided enlargement of the liver. Where there are no hepatic abscesses, the organ usually retains its normal bulk.

The course of the fever is very characteristic. As in other pyæmic conditions, there are almost invariably great elevations, to 106° (41° C.), or higher, accompanied by rigors, and followed by marked remissions, with profuse perspiration. These onsets of fever occur at irregular intervals, either daily, or every two or three days.

There are at the same time indications of constitutional septic infection, which keep increasing in severity. The pulse grows rapid and small. Intelligence is impaired. Somnolence and delirium come on, and the strength rapidly fails.

There are other symptoms. Vomiting is frequently seen. The bowels are seldom constipated, but usually relaxed. The dejections may contain blood, because of the venous stasis. In some cases the inflammation extends so as to produce a fatal general peritonitis. It is noticeable that the urine is generally scanty, and the amount of urea is strikingly diminished.

The disease usually runs a rather acute course. On the average, it lasts about two weeks, but may occupy three or four weeks, or even a longer period. It is invariably fatal. At least, no cases of recovery are known.

The diagnosis can sometimes be made with considerable positiveness. In other instances it is impossible to exclude other pyæmic conditions, or abscess due to gall-stones, etc. Important factors are the origin of the trouble—if it can be made out—the pyæmic rigors, the enlargement of the spleen and liver, jaundice, epigastric pain, and the evidences of general sepsis.

Treatment is unfortunately almost entirely useless. The fever is not affected even by large doses of quinine. All we can aim at is to support and relieve the sufferer as far as possible.

CHAPTER XV.

THROMBOSIS OF THE PORTAL VEIN.

(*Chronic Adhesive Pylephlebitis. Pylethrombosis.*)

Ætiology and Pathology.—Like suppurative pylephlebitis, chronic portal thrombosis is not an independent disease, but is the sequel of a great variety of pathological conditions. Marantic thrombosis is of rare occurrence, and is usually formed toward the close of life, so as not to be of practical interest. Apart from this, almost all cases of thrombosis of the portal vein are due to a compression of the trunk of that vessel or one of its main branches. This most often occurs in certain chronic hepatic diseases which involve a mechanical stenosis, either of the smaller branches of the portal vein within the liver, or of the vein itself, with resulting coagulation of the blood within it. Chief among these diseases are cirrhosis and syphilis of the liver, which have repeatedly been observed to entail portal thrombosis; but other diseases in the neighborhood of the vein may produce a similar effect. New growths of various kinds may press upon the vessel, or chronic inflammatory hyperplasia of the connective tissue at the porta hepatis may act in the same way. This is illustrated in chronic peritonitis, whether circumscribed or diffuse, an example of the former being sometimes seen as an effect of duodenal ulcer.

It was formerly held that many forms of so-called "lobulated liver" were due to a primary adhesive pylephlebitis. This is erroneous. These cases are probably all due to some primary hepatic disease, usually syphilitic. The size of the liver is little influenced by obstruction of the portal vein, even if long continued, for the hepatic artery suffices to supply all the blood required by the organ.

The anatomical changes in pylethrombosis do not differ essentially from those seen in thrombosis of any other vein. If fresh, the thrombus is still red; later it grows harder, paler, and more friable. If the thrombosis has existed a long while, the clot becomes completely organized. We have observed this even in the main trunk of the portal vein.

Clinical History.—The symptoms of portal thrombosis are those occasioned by the obstruction, and therefore such as we have already repeatedly met with, in connection with various hepatic diseases. The intensity and extent of these results, as well as the time occupied in their development, depend, of course, upon the place and size of the clot. If it is the portal vein itself which is attacked, and if the thrombus is extensive enough to obstruct the flow of blood, then the signs of venous stasis are evident throughout the portal system. The spleen becomes much enlarged, as can be easily demonstrated by percussion and palpation. Soon ascites appears, as a result of the passive congestion of the peritoneal veins; and from a similar condition of the gastro-intestinal veins arise catarrhal disorders, like diarrhœa; or, not so very exceptionally, there is repeated gastric and intestinal hæmorrhage.

As we have seen, a collateral circulation may be developed (*vide* page 481), by which the venous blood of the portal system is enabled to reach the systemic veins. This explains why some of the symptoms of venous stasis may temporarily (perhaps permanently) vanish. We saw one case of portal thrombosis, the sequel to what was apparently a syphilitic disease of the liver, where a quite large ascitic effusion appeared some six or seven times at intervals of three to six months, and under proper treatment as often abated. The patient did not die till the illness had lasted six years, and tapping had been demanded some fifteen times. At the autopsy the trunk of the portal vein was found to be converted into a fibrous cord, with a lumen which barely admitted a knitting-needle. It is the development of a collateral circulation which causes the frequent distention of the veins in the abdominal walls. Sometimes these enlarged veins take the form of the "*caput Medusæ*" mentioned above.

In simple pylethrombosis there are no local symptoms such as pain. The condition of the liver depends upon the primary disease. It is possible that a moderate atrophy of the entire organ might at length ensue if the portal blood were permanently cut off from it. But, as we have said, any cirrhotic changes, or any "lobulation," are not to be regarded as the result, but as the cause of the thrombosis, or at least related to the cause.

The course and duration of the trouble are according to the nature of the original, causative trouble. No general statements can be made.

The diagnosis of thrombosis of the portal vein is usually extremely difficult, and can really hardly ever be made with absolute certainty. We may, indeed, recognize readily that there is some decided obstruction to the portal circulation; but whether this be due to a thrombus, or to compression of the portal vein, or to the obliteration of a large number of the smaller branches of that vein within the liver, we can very seldom determine. Pylethrombosis may be regarded as probable, if no other possible cause of the portal obstruction seems likely, and if we are able to discover a cause for thrombosis, like a former attack of circumscribed peritonitis.

The prognosis is always unfavorable, although there may be, as we have said, great temporary improvement. Treatment must be symptomatic, and follows in the main the principles set forth under cirrhosis of the liver.

APPENDIX.

DISEASES OF THE PANCREAS.

THE few facts of clinical importance that are known about the pathology of the pancreas are given below.

1. **Hæmorrhages into the Pancreas** of small size occur as one symptom of a general hæmorrhagic diathesis, or as the result of excessive passive congestion. They are of no special importance. Klebs and Zenker have described a few cases, however, where there was extensive hæmorrhage into this organ, and where this was the only discoverable cause of death. The patients had been previously well and vigorous, although decidedly obese, and had died suddenly. Perhaps the speedy termination was caused by the influence of the hæmorrhage upon the semilunar ganglion or solar plexus. The cause of the hæmorrhage could not be determined.

2. **Atrophy of the Pancreas.**—The organ may share in a general marasmus. There is also extreme atrophy of the pancreas in those who have died of diabetes mellitus (*q. v.*). What relation this change bears to the diabetes is not known.

3. **Pancreatitis.**—A few cases have been reported of what would seem to be a primary acute pancreatitis. The disease is certainly very rare. It begins with violent colicky pains in the epigastrium. Vomiting and collapse soon follow. The pulse grows small, the extremities become cool, and death is speedy. At the autopsy the pancreas is found to be much enlarged, and mottled with ecchymoses, or it even presents scattered foci of suppuration. The ætiology is unknown. Secondary abscesses of the pancreas are not infrequent in pyæmia.

[Fitz, in the Middleton-Goldsmith lecture for 1889, threw much light on the ætiology and diagnosis of acute pancreatitis, which he subdivides into three anatomical forms—the hæmorrhagic, the suppurative, and the gangrenous. He shows that the affection is not so rare as has been supposed; that its victims are usually in middle life, fat, and good livers; that it commonly originates by the extension of a gastro-duodenal inflammation along the pancreatic duct.

"If the case does not end fatally in the course of a few days, recovery is possible, or a recurrence of the symptoms in a milder form takes place, and the characteristics of a subacute peritonitis are developed."

In the differential diagnosis irritant poisoning, perforation of the digestive or biliary tracts, and acute intestinal obstruction are to be considered. The location of peritonitic symptoms, their suddenness of onset, the absence of apparently sufficient cause, and the age and habit of the individual are important points.]

Chronic interstitial pancreatitis sometimes results from the extension of chronic inflammatory processes affecting neighboring parts. Friedreich states that it sometimes is a primary disease in topers. Syphilitic lesions of the pancreas have been observed, occasioning contraction and induration. None of these changes give rise to special clinical symptoms; or, if there were a single distinctive symptom, it would be one common to all sorts of grave pancreatic disorder—namely, the appearance of a large amount of fat in the stools. As we know, the pancreatic juice is an important factor in the digestion of fat, so that it is very natural for any great derangement of the organ to have this result; and yet the bile alone may render the ingested fat capable of absorption, so that in repeated instances there have been no fatty stools when the pancreas has been completely atrophied or degenerated.

4. **Cysts of the Pancreas.**—After closure of Wirsung's duct by scars, concretions, or the like, cysts of the pancreas may form, as a result of the damming up of the secretion. These may become so large as to be felt as great tumors through the abdominal walls. In some instances they have been operated upon with success.

5. **Cancer of the Pancreas.**—Primary cancer is the most frequent, and therefore clinically the most important disease of this organ. As a rule, the new growth is situated in the head of the pancreas. It is usually of the medullary variety, though occasionally colloid. It may involve neighboring parts by direct extension, and a great many organs by metastasis; for example, the liver, peritoneum, and lymph-glands.

The clinical symptoms of cancer of the pancreas are very seldom so decided as to justify a positive diagnosis. Sometimes the secondary nodules can be detected in the liver, peritoneum, and elsewhere. Then we are left in doubt about the seat of the primary growth. Or the primary tumor may be plainly felt through the abdominal walls; but then we can hardly ever exclude cancer of the stomach or of the omentum, and neighboring parts.

The symptoms of pancreatic cancer, as a whole, resemble closely those occasioned by most cancers of abdominal organs. Usually the patient is elderly. The first symptoms are loss of flesh and strength, or are the result of compression. Often there is complaint of a persistent dull pain in the epigastrium. If the portal vein is pressed upon by the tumor, ascites appears. If the common duct is compressed, there is jaundice. Marasmus increases, and usually at the end of six months or a year the patient dies.

The diagnosis can be said to be somewhat probable in those cases only where there are fatty stools, where a tumor can be felt in a position corresponding to the pancreas, and where primary cancer of any other organ is unlikely; but usually, as we have said, the symptoms are very ambiguous. There have been no fatty stools in a number of cases, even where the cancer was extensive.

The prognosis is absolutely bad. The treatment is merely symptomatic, with the aim of lessening the patient's suffering.

DISEASES OF THE NERVOUS SYSTEM.

I.—The Diseases of the Peripheral Nerves.

SECTION I.

DISEASES OF THE SENSORY NERVES.

CHAPTER I.

GENERAL REMARKS UPON THE DISTURBANCES OF SENSIBILITY.

THE disturbances of sensibility, like all other functions of the nerves, are manifested in two directions. Under pathological conditions we observe either an abnormal diminution or a complete absence of sensibility—anæsthesia—or a morbid increase—hyperæsthesia. While, in anæsthesia, the ordinary or even the strongest irritations which excite the sensory nerves produce only a weak and insignificant sensation, or even no corresponding sensation at all, in hyperæsthesia very severe and painful sensations are caused by weak irritations. The "symptoms of sensory irritation" are to be distinguished from hyperæsthesia, although they are often present along with it. By this term we mean sensations which cause internal irritation, not from without, but from certain abnormal morbid conditions in the nerve itself. In the region of cutaneous sensibility, with which we shall chiefly concern ourselves in what follows, these symptoms of sensory irritation show themselves partly as actual pain and partly as the so-called paræsthesiæ—that is, abnormal sensations in the skin, which are termed "formication" (the crawling of ants), "prickling," "numbness," "a furry feeling," etc.

The Different Varieties of Cutaneous Sensibility and the Methods of testing them.—As is known from physiology, the irritation of the sensory cutaneous nerves produces in us a number of sensations, differing in quality according to the manner of action of the irritation. If, therefore, we would obtain an accurate estimate of the condition of the patient's cutaneous sensibility, we must make a special test of all the different forms of sensation, for we often see that the disturbances of sensibility do not involve all the forms mentioned alike, but that one kind of irritation is followed by perfectly normal sensations, while there is more or less complete anæsthesia for another kind. We term such partial anæsthesias of the skin, which are manifest toward only one form of irritation, "partial paralyses of sensation." Such partial paralyses will be much more easily understood if the remarkable statements of Blix, Goldscheider, and others are confirmed. According to these statements, the different qualities of cutaneous sensation are transmitted to the consciousness by special nerve-fibers, so that there are in the skin special nerves for the tactile sense, for the sense of cold, for the sense of heat, etc. Such a condition is analogous to the well-known specific energy of the different fibers of the optic nerve to colors, assumed by many physiologists. The separate varieties of cutaneous sensibility, and the methods of testing them, are as follows:

1. TACTILE SENSIBILITY.—The examination of the tactile sensibility—that is, of the sensibility of the skin to simple contact—is usually performed by repeatedly touching or stroking the part of the skin to be tested with the finger, a fine brush, or some other blunt object (not of metal, in order to exclude sensations of cold) while the patient's eyes are shut, and making the patient say whether he has felt the touch or not. When it is necessary to call the patient's attention to the investigation, it is always best to do so afresh by asking "Now ?" and then either really touching the skin or else asking the question when only pretending to do so. In this way we are safest from error, which is otherwise easily produced by a lack of attention or of practice on the part of the patient. All accurate tests of sensibility must be repeatedly performed and controlled in order to obtain accurate objective results.

If, as in most cases, we have to do with disturbances of sensibility which affect only a part of the skin, we must make comparative tests of sensibility on the healthy and, if possible, symmetrical portions of the skin. Slight disturbances of sensibility are then often shown in this way, that the patient feels almost every touch on the affected part; but the sensation always seems to him more indefinite, blunter—in short, "different" from that on the corresponding normal portion of the body.

Besides simply touching the skin, we also try how far the patient is able to distinguish the form and certain external peculiarities of objects by the aid of his tactile sense. We touch the skin with smooth and rough (woolly) or round and angular objects, and see whether the patient can distinguish them respectively with his eyes shut, and also whether he can distinguish between the head and the point of a pin, etc. If we are testing the sensibility of the fingers, we may put different well-known objects—like coins, rings, or keys—into his hands, and let him name them with his eyes shut. We may also try the last method of testing by the aid of a number of wooden geometrical objects, cubes, octahedra, or cones.

2. SENSE OF LOCALITY.—Under normal conditions, as we know, not only do we feel the touch of an object, but we can tell with a good deal of accuracy the place on our skin which was touched. This power we term the ability to localize our sensation. In nervous patients we often see that, while cutaneous sensibility is still present (we refer not only to tactile sensibility but also to the other forms), it is localized more poorly and with less accuracy than is the case under normal conditions.

In the simple test of the tactile sense we may also examine, at least roughly, the power of localization if we make the patient also state where the touch is felt, or if we ask him to designate with his hand as accurately as possible the part of the skin touched. A more accurate method, much used in nervous pathology, was proposed by E. H. Weber. It consists in determining the smallest distance which must separate two simultaneous cutaneous irritants from each other, in order that they may be perceived as two locally distinct sensations. Weber has found that this distance differs very much in different parts of the body, and from this he has divided the whole surface of the skin into so-called tactile circles. As data for the examination of patients, some of the figures obtained by Weber in healthy individuals may here be given: The smallest distance at which the two points of a pair of compasses* applied at the same time to the skin may plainly be distinguished from each other is 11 to 15 millimetres on the cheeks, 6 mm. at the tip of the nose, 22 mm. on the forehead, 1·2 mm. at the tip of the tongue, 4 to 5 mm. at the back of the tongue and on the lips, 34 mm. on the neck, 77 mm. on the upper arm, 40 mm. on the forearm, 31 mm. on the backs of the hands, 11 to 16 mm. on the backs of the fingers, 2 to 3 mm. at the tips of the fingers, 55 to 77 mm. on the back, 45 mm. on the chest, 77 mm. on the thigh, 40 mm. on the leg, 40 mm. on the instep; but these figures show certain variations in different individuals, so that they are to be regarded as only average values.

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* There are special "tactile compasses" with blunt ivory points and graduated quadrants.

Testing the sense of locality according to Weber's method takes a great deal of time, and demands much patience and good will on the part of the patient. The influence of practice is manifested in a very remarkable way, since the perceptible difference becomes considerably less if the examinations are often repeated. On the other hand, a single examination, as in testing any form of sensibility, must not be too long protracted, for otherwise the patient may easily become fatigued, and the data obtained will be entirely contradictory. If we test the sense of locality by bringing down the two points not at the same time but one after the other, and vary it by touching the same place twice, or a different place each time, we obtain from the outset, as we have repeatedly proved, smaller numbers than if we touch the skin with the two points of the compasses at the same time. We also obtain some different values for the fineness of the sense of locality if we test the so-called sensations of motion (Leube)—that is, the distinction between a simple circumscribed touch of the skin, and a very short line drawn with a stick on the skin. In this way we can also determine whether the patient can distinguish accurately the direction of transverse and longitudinal lines.

We may also mention here the peculiar symptom termed by Fischer polyæsthesia, which is that certain patients, especially ataxics, when the skin is touched with only one point of the compasses, have a sensation as if they felt two or even more points. The cause of this remarkable anomaly of sensation is not yet adequately explained.

3. Sense of Pressure.—Since E. H. Weber's investigations we know that we estimate the difference in the intensity of our sensations of pressure, not according to the absolute, but according to the relative increase in the pressure. If, for instance, a place in the skin has a weight of nineteen grammes on it, and we perceive the first manifest increase in our sensation of pressure when a weight of one gramme is added to it; when the skin has a weight of one hundred and ninety grammes on it, we first perceive the increase of pressure not when one gramme is added, but when we add ten grammes. If this law, on more accurate testing, is not as simply proved as it seems according to the results of Weber's first investigations, still it is generally a fact that under normal conditions an increase of pressure of about one twentieth to one thirtieth of the original pressure may be plainly perceived in the different parts of the body.

Different methods and instruments, like Eulenburg's "baræsthesiometer," have been devised for testing accurately the sense of pressure in patients, but they have entered but little into practice on account of their elaborate character. We usually content ourselves with testing the sense of pressure by applying different weights, or coins. We must mention here that the part of the body to be tested must be fully supported, that we must also exclude sensations of temperature at the same time by putting something beneath the weights, and that we must apply the separate weights to the same place on the skin at equal intervals of time, which must not be too long after one another. There are cases where the patient does not feel even the doubling or tripling of the weights. We can easily confirm a considerable loss of the sense of pressure by means of simply varying pressure with the hand or any object, such as a pencil applied to the skin.

Partial paralyses of the sense of pressure are by no means rare. We find quite often, especially in spinal diseases, like locomotor ataxia, that the patient feels a light touch on the skin, but that he can not distinguish a marked pressure at all, or only obscurely.

4. SENSE OF TEMPERATURE.—As we have already said (p. 505), we have lately been led by physiological investigations (Goldscheider, etc.) to believe more and more strongly that sensations of heat and cold are to be regarded as due to two wholly distinct functions of the cutaneous nerves. These functions are probably performed by distinct nerve terminations and nerve-fibers. From repeated personal observation we can state that pathological symptoms agree completely with this theory, for we see that changes in the temperature sense in the skin by no means affect the heat sense and the cold sense equally. It is therefore always necessary to test both forms of the temperature sense separately. We often find that while one form of temperature sense is in normal activity, the other is much altered—a pronounced partial anæsthesia to heat or cold. The affected sense may be wholly lost or merely blunted, so that hot water is felt merely as tepid, or ice as cool. If the temperature sense be lost, the application of a hot or cold object will cause only a sensation of touch, and not of temperature.

If anæsthesia to cold be present, patients often speak of having a distinct sensation of warmth when the skin is touched with a bit of ice. This symptom, which we discovered and called "perverted temperature sense," may be explained most readily by supposing that the heat nerves are excited by the severe irritation from cold. The contrary symptom, that heat excites a distinct sensation of cold, is very much less common.

Besides the sensations of temperature we usually test also the patient's capacity to distinguish differences of temperature. Within the moderate degrees of temperature, 80° to 100° (25°–35° C.), differences of a degree Fahrenheit (0·5 C.) are easily distinguishable under normal conditions, and even half a degree (0·2 C.) can be distinguished on the face and fingers, but only about two degrees (1° C.) on the back.

Variations in the temperature sense are very common, especially in spinal diseases. The test of the sensibility to heat and cold should therefore never be omitted in testing the sensibility. It can be done with sufficient accuracy for all ordinary purposes simply by touching the skin with test-tubes filled with ice or hot water. The various thermæsthesiometers that have been devised are too complicated for practical purposes. For a hasty test it is sometimes quite serviceable to try whether the patient can distinguish warm breathing upon a given portion of the skin from cool blowing upon it.

5. SENSATION OF PAIN.—The fact is of great theoretical interest that the cutaneous sensibility for touch and pain do not always, under pathological conditions, run parallel with each other. We sometimes see that a patient does not feel a simple touch on the skin, when sticking a needle into it is immediately painful; while, on the other hand, we often find that a patient feels quite a light touch on the skin, but that the most marked irritation, like pinching or pricking it, does not excite the slightest pain, but is felt only as a simple touch, or at most as a slight pressure on it. This latter condition of sensibility, the loss of cutaneous sensibility to pain with retained tactile sensibility, is termed analgesia. Both in peripheral and in central nervous diseases analgesia is a symptom that may be quite frequently observed.

The test of sensibility to pain may be made most simply by the point of a pin, and also by pinching or severe pressure on the skin, by painful thermal irritants, by strong electrical currents, etc.

6. ELECTRO-CUTANEOUS SENSIBILITY.—The test of cutaneous sensibility by means of the electric current has been proposed from various quarters. The advantage of it is that in this way the intensity of the irritation can be very

easily and accurately graded and expressed in numbers, by the position of the cylinder in using the faradic current, or by the galvanometer in using the constant current. The faradic current is usually sufficient in testing the sensibility, and we designate it by the position of the cylinder when the first sensation is felt and the position when the first pain is felt. In general, the differences of the farado-cutaneous sensibility are not very marked. Pathological deviations are given by comparison with normal portions of the skin (testing, if possible, symmetrical parts) or with healthy people. For practical purposes, the test of electrocutaneous sensibility is unnecessary, since its results are the same as in testing the sensibility to touch and to pain.

7. DELAYED CONDUCTION OF SENSATION AND AFTER-SENSATIONS.—In diseases of the spinal cord, especially in locomotor ataxia (*q. v.*) we quite frequently see a marked delay in sensation after the action of an irritant; this is also seen, but more rarely, in peripheral lesions. This delay of conduction affects chiefly the sensibility to pain. If in such a case we stick a pin into the sole of a patient's foot, several seconds elapse, even ten or twenty, it is said, before the pain is felt. As was first observed by Naunyn and E. Remak in ataxics, and as has often been confirmed since, after sticking a pin into the foot there is first a sensation of touch, and some seconds later the peculiar sensation of pain, so that the patient at once responds to the prick with "Now," and a little later with "Ow!" as an expression of pain.

This latter phenomenon has a certain relation to the abnormal after-sensations which are often observed under pathological conditions. After a simple pin-prick a feeling of burning lasts for an extraordinarily long time, or else the first pain disappears quite soon, and then a new sudden sense of pain appears several times in the same part of the skin, just as if the patient were pricked again. There is also a delayed conduction of the sensations of touch and temperature, but it is rarer, and can be made out only by the aid of more accurate methods of measuring time.

The Sensibility of the Muscles and Joints.—A number of sensations are classed together under the names of "muscular sense" or "muscular sensibility." They are not all wholly of the same value, and, under pathological conditions, they must be tested separately.

Ordinarily we call our power to be informed of the position of any of our limbs without the help of our eyes, and of the extent of any motion made by them, the "muscular sense." If we put a healthy person's arm into a given position when his eyes are closed, and tell him to put the other arm into the same position, he can do it with considerable accuracy. If we make passive movements in any of the joints of the extremities, a healthy person whose eyes are closed can easily and correctly state the form and direction of these movements, even if they be very slight. In nervous patients, however, this power may be diminished or lost, and we often speak of "disturbances of the muscular sense"; but we must note that the judgment as to the position of the limbs and the passive movements executed with them is not chiefly, much less exclusively, due to the sensibility of the muscles. In all probability it depends much more upon the sensibility of the articular surfaces, the ligaments and tendons, and in part even of the skin, all which tissues are relaxed or made tense in the different movements. It would therefore be more correct to speak simply of the sense of position of the limbs and the sense of passive movements instead of the muscular sense. The sense of passive movements can also be conveniently tested by describing in the air different letters or figures with the affected extremities of the patient, and requiring him to recognize them with his eyes closed. Another convenient way to test the muscular sense is to have the patient make a definite movement—pointing to or taking hold of some object—with the eyes open, and then to make him repeat the act with the eyes shut.

We also include in the muscular sense the power of estimating the amount of work done by muscular contraction. This is the so-called "sense of power." In raising weights we can distinguish with considerable accuracy the lighter from the heavier, when the pressure on the skin is excluded as far as possible. In such cases, also, we do not deal with the absolute, but with the relative differences in weight; we can usually tell quite plainly when one fortieth of the original weight has been added or taken away. The sense of power, then, is somewhat finer than the sense of pressure. In order to exclude the latter in the test we have the patient lift the weight, suspended in a towel, with his hand or foot, but in the lower extremities it is scarcely possible to exclude entirely the co-existing sensations of pressure.

We must mention, in conclusion, that muscular contraction is in itself accompanied by a sensation, as may be proved, for example, in irritating the muscles by faradism—electro-muscular sensibility; but we have not yet found any real practical value in testing the feeling of contraction in muscles. We must state, however, that in certain forms of spasm the muscular contraction becomes so strong that it causes a decided pain, which is probably due to irritation of the sensory muscular nerves discovered by C. Sachs.

Disturbances of the muscular sense, or, to speak more correctly, disturbances in the sensibility of the deeper parts, are seen chiefly in locomotor ataxia, sometimes in paralysis of cerebral origin, and quite frequently in severe hysterical affections.

CHAPTER II.

ANÆSTHESIA OF THE SKIN.

Ætiology and Pathogenesis.—In every tract of the conducting path, which runs from the terminal apparatus of the sensory cutaneous nerves to the centers for the perception of sensation in the cerebral cortex, we may have, under pathological conditions, a break in the conduction, and, as a result of it, a complete or partial anæsthesia of the corresponding part of the skin. We speak of a peripheral, spinal, or cerebral anæsthesia, according to the place where this break in the conduction occurs. The precise anatomical course of the sensory fibers is, however, very imperfectly known, so that we can only approximately determine the location of the sensory fibers in the different portions of the nervous system.

We know this, however, of the mixed peripheral nerves before their entrance into the spinal cord, that all their sensory fibers enter the cord through the posterior roots. A part of the posterior root-fibers pass directly into the substance of the posterior gray cornua, while another part enter toward the median line into the external (in the lumbar cord, more correctly the median) portion of the posterior columns—that is, into the region of the "root-zones," or the so-called "elementary bundles of the posterior columns." Since new fibers from the posterior roots constantly enter the posterior columns of the cord, going upward from the lumbar region, the fibers which have entered in the lower portions must gradually be more and more crowded inward toward the median line. Hence it follows that the fibers entering the lumbar region, belonging to the sciatic, the crural, etc., must in the upper dorsal and cervical region occupy that internal portion of the posterior columns which is called the "columns of

Goll" (Fig. 88, G). We can not, however, state definitely at present which of the different prolongations of the posterior roots is the special path for sensory conduction. We are ourselves very much inclined to the opinion that the special sensory fibers of the posterior roots for the most part enter directly into the gray matter of the posterior cornua, and that the elementary bundles of the posterior columns—the continuation of the fibers which enter directly into the posterior columns—accordingly serve chiefly for other functions. We are led to this opinion especially by observations of quite severe disease of the columns of Goll, which occasioned no perceptible disturbance of sensation in the lower extremities during the patients' life. Disease of the posterior cornua of the gray matter, however, is always associated with disturbances of sensibility. It is not probable, according to our present experience, that there is a sensory tract in the lateral columns also in man. The fact is certainly proved, and is of importance, that all, or at least the greater part, of the sensory fibers undergo a decussation after their entrance into the spinal cord, so that the fibers from the right half of the body pass upward in the left half of the cord, and *vice versa*. We know absolutely nothing definite as to the further course of the sensory fibers through the medulla oblongata and the pons, or as to their relations to the gray matter there, but it seems to be certain that the sensory tract continues on to the cerebral hemispheres, not through the crusta, but through the tegmentum. From this point the sensory fibers pass to the internal capsule, and a number of experiments show that they lie in the posterior third of the posterior limb of the internal capsule behind the pyramidal tracts (see Fig. 64), a place where the cutaneous and muscular sensory fibers are probably near the fibers for the impressions for the special senses of vision, hearing, etc. Nothing certain is known as to the central termination of the sensory fibers. Perhaps the posterior central convolution and the portions of the parietal lobe behind it may be regarded as the special places of termination for the sensory tract. We do not know whether tracts of conduction go to the brain which are distinguished from one another according to the different forms of cutaneous sensibility.

Regarding the separate causes of anæsthesia, we see, in the first place, peripheral anæsthesia under conditions where the terminal organs of the sensory cutaneous nerves have lost their direct irritability. After chilling the skin, after the local action of ether and similar substances, after the corrosive action of acids and alkalies, carbolic acid, etc., as well as after the use of certain narcotics, such as cocaine, morphine, or atropine, we see an anæsthesia of the skin, which is due to injury of the terminal sensory organs. We may probably put the frequent anæsthesia of washerwomen in this class, for their hands and forearms are exposed all day to the action of cold, lye, etc. The anæsthesias which develop in circulatory disturbances of the skin also have the same peripheral origin, especially the "spastic anæmia" which sometimes comes in the hands and is due to a spasm of the small arteries.

We distinguish the peripheral anæsthesia of conduction, which may be produced by all forms of lesion of the nerve-trunks, from the peripheral anæsthesia in the strict sense of the term. Traumatic influences, compression from new growths, and inflammation and degeneration of the peripheral nerves, as in neuritis, are the most frequent causes of this form of anæsthesia, which is often limited to the region of distribution of one or more definite nerves.

Spinal anæsthesia is very often seen in the different diseases of the spinal cord, most frequently in locomotor ataxia, because this, as we shall see later, attacks chiefly the posterior roots, the posterior columns, and the posterior cornua of the cord; but spinal anæsthesia is not infrequent in diffuse acute and chronic inflammation of the cord and in compression and new growths. As a rule, it is bilateral, para-anæsthesia. The hypothesis advanced by Schiff is quite widespread, but is by no means certainly proven, that the gray matter of the cord conducts chiefly the impressions of pain, and the white matter of the posterior columns the sensations of touch. According to this, in spinal analgesia we must assume chiefly an impairment of the gray matter.

Cerebral anæsthesia is seen especially in hæmorrhages, foci of softening, and tumors, which affect the posterior portion of the internal capsule; but of course the break may occur in any other part of the tract of sensory conduction in the brain. If the cerebral anæsthesia, as is often the case, affects the half of the body opposite the lesion in the brain, we term it hemianæsthesia. Very extensive cerebral anæsthesia of a marked degree is quite frequently found in severe cases of hysteria. We also know that the anæsthetic action of the remedies termed anæsthetics and narcotics, such as chloroform, morphine, ether, alcohol, bromide of potassium, etc., must be explained by their influence on the central nervous system.

Among other ætiological factors we must also mention that we sometimes see more or less extensive anæsthesia as a result of acute diseases, such as typhoid, diphtheria, and other acute infectious diseases, the origin of the anæsthesia, whether peripheral or spinal, being as yet uncertain. Peculiar insular and, rarely, diffuse anæsthesia, showing itself chiefly on the backs of the hands and the chest, is found in the secondary stages of syphilis, according to Fournier; but there has been no further confirmation of this statement so far as we know.

Symptoms.—In many cases the patient himself notices the existence of anæsthesia. He finds that in certain parts of the body he no longer feels the pressure of his clothing or the bedclothes in the usual way. Anæsthesia of the hands is soonest noticed, because this can affect the patient's occupation in diverse ways, so that, for example, he readily lets fine objects, such as needles, drop from his hands. In other cases, of course, the anæsthesia is first found on a physical examination, which alone can give definite disclosures as to the extent and intensity of the affection. For this purpose the skin must be carefully examined by the methods given in the preceding chapter. It is worthy of note that hysterical anæsthesia especially, even if it be very marked and extensive, may often be wholly overlooked by the patients themselves.

Anæsthesia is very often combined with abnormal subjective sensations, paræsthesiæ, in the affected portions of the skin. The patient has a feeling there of "numbness," or a "furry feeling," or complains of prickling or formication. The anæsthetic parts may even be the seat of very decided pain (*anœsthesia dolorosa*), if there is abnormal irritation of the sensory nerves from the break in the conduction toward the center. The most diverse forms of anomalies of motility and of the reflexes, and vaso-motor disturbances, may of course be present in addition to the anæsthesia. Special mention must be made of trophic disturbances which are often seen in anæsthetic regions. We shall return to the peculiar characteristics of these repeatedly in subsequent chapters; therefore we need state here only that the trophic disturbances have nothing to do with the anæsthesia itself. They depend either upon a co-existing lesion of special trophic or vaso-motor nerves, or upon the fact that external irritants, which act upon any anæsthetic portion of the skin, are not felt normally by the patient, and hence are often not avoided. In anæsthetic regions we often find large external wounds, burns, bed-sores, inflammations, etc., which were not noticed by the patient in time to prevent them, and hence they often attain an unusual extent.

Voluntary motion is not disturbed by anæsthesia in itself, of however great a degree, as long as the motions can be controlled by the eyes, but finer movements are often considerably impaired by cutaneous anæsthesia; thus patients with diminished sensibility in the fingers can usually no longer sew, because they lose the needle every minute. With the eyes shut, however, the motions of the anæsthetic parts become very uncertain, both in anæsthesia of the skin and of the deeper parts, the muscles and joints, since then the patient loses, to a great degree, his power of judging of the extent and of the precise direction of his movements. Very extensive anæsthesia of the skin, associated at the same time with anæsthesia of the organs of special sense, is sometimes not without influence upon consciousness. We have for several years had under observation a very remarkable case of total anæsthesia of the whole body associated with unilateral blindness and deafness. If we entirely exclude this patient from all external impressions of sense by closing his still serviceable eye and ear, we can at once in this way put him into a deep sleep!

We can not here go into details of the different forms and distributions of anæsthesia, since they will be spoken of under the different diseases which lie at the basis of the anæsthesia. The course, the duration, and the prognosis of the affection depend, in the first place, of course, upon the form of the primary disease. We will add here merely a few remarks upon anæsthesia of one nerve, namely, anæsthesia in the distribution of the trigeminus.

Anæsthesia of the trigeminus is observed in tumors, syphilitic new growths, chronic inflammations, and analogous processes at the base of the skull, which compress the trunk, the Gasserian ganglion, or one of the three branches of the trigeminus, or directly involve the nerves. Traumatic lesions of the trigeminus are also not very uncommon. The distribution of the anæsthesia, according as the affection involves the whole trigeminus, or only one branch of it, may be seen in Figs. 57 and 58. In total anæsthesia of the trigeminus, the conjunctiva and cornea, the mucous membrane of the nose, the cavity of the mouth, and the tongue are anæsthetic on the affected side. Hence we often find ulcers on the tongue and the mucous membrane, which come from being bitten. The "neuroparalytic ophthalmia" not infrequently seen in anæsthesia of the trigeminus is of special interest, and has been much studied by physicians and physiologists. This

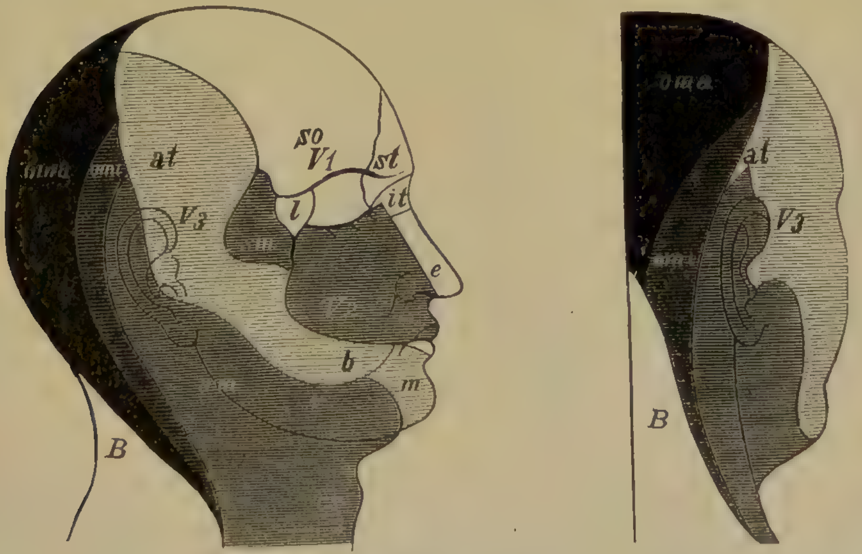

FIG. 57. FIG. 58.

FIGS. 57 and 58.—Distribution of the sensory cutaneous nerves in the head: *oma* and *omi*. Occipitalis major and minor. *am*. Auricularis magnus. *cs*. Superficial cervical. V_1, V_2, V_3. First, second, and third branches of the fifth (V). *so*. Supraorbital. *st*. Supratrochlear. *it*. Infratrochlear. *e*. Ethmoidal. *l*. Lachrymal. *sm*. Subcutaneus malæ, or zygomatic. *at*. Auriculo-temporal. *b*. Buccinator. *m*. Mental. *B*. Posterior branches of the third cervical.

is an ulcerative keratitis, almost always beginning in the lower segment of the cornea, and sometimes passing over into a purulent inflammation of the whole eyeball. This affection is regarded in many quarters as an immediate result of the disturbance of special "trophic" functions, but, after careful experiments (Senftleben), it seems most probable that external traumatic influences always occasion the first trouble, and render the intrusion of inflammatory agents possible. It is still uncertain whether we must also assume a specially diminished power of resistance on the part of the tissues as a result of the nervous lesion.

The skin of the face is often somewhat bloated in anæsthesia of the trigeminus, and it is cyanotic and feels cool. The reflexes are lost in peripheral anæsthesia, and the lachrymal secretion is diminished. The taste is almost always decidedly diminished on the anterior two thirds of the tongue on the affected side, the territory supplied by the lingual nerve.

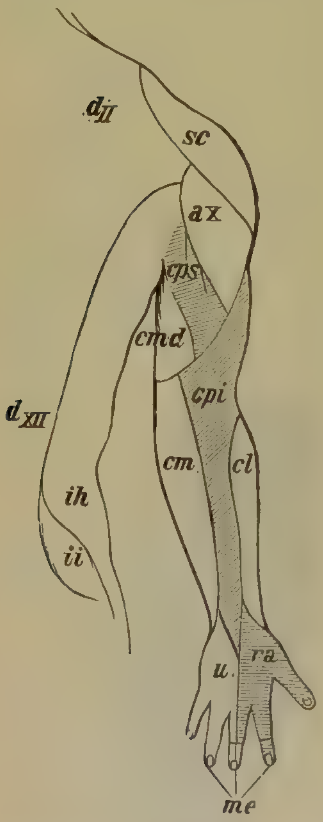

FIG. 59.

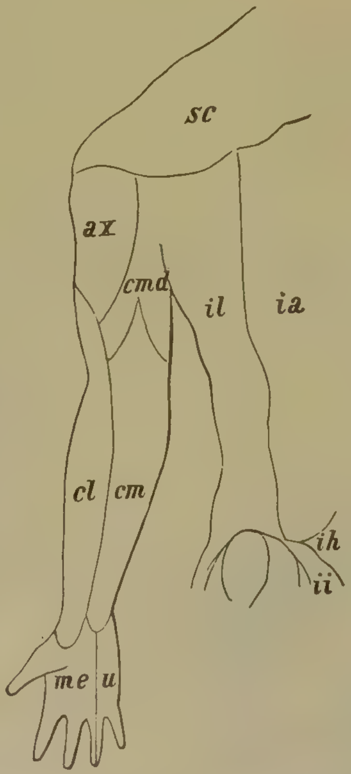

FIG. 60.

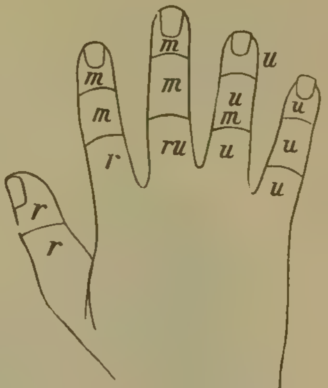

FIG. 61.

FIGS. 59 and 60.*—Distribution of the sensory cutaneous nerves in the trunk and upper extremities: FIG. 59. Posterior aspect. FIG. 60. Anterior aspect. The shaded portion in Fig. 59 designates the territory supplied by the radial nerve. (From HENLE.) *sc*. Supraclavicular nerves (from the cervical plexus). *ax*. Cutaneous branch of the axillary nerve. *cps* and *cpi*. Superior and inferior posterior cutaneous nerves from the radial (*ra*). *cmd*, *cm*, and *cl*. Median cutaneous, median, and lateral nerves. *me*. Median nerve. *u*. Ulnar nerve. d_{II}. Second dorsal nerve. d_{XII}. Twelfth dorsal nerve. *ih*. Ileo-hypogastric nerve. *ii*. Ileo-inguinal nerve. *il*. Lateral perforating branches, and *ia*, anterior perforating branches of the intercostal nerves.

FIG. 61.—Detailed distribution of the nerves to the dorsal surface of the fingers. (From KRAUSE.) *r*. Radial nerve. *m*. Median nerve. *u*. Ulnar nerve.

Treatment.—Since anæsthesia in most cases is only a symptom, treatment, of course, must always first be directed against the underlying disease. Therefore we will mention here only those measures which are to be used symptomatically against anæsthesia, and which must be tried when the special cause can not be discovered, or when it is inaccessible to treatment.

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* Henle's nomenclature of the peripheral nerves has been adopted.—TRANS.

The chief remedy is, without doubt, the electric current. We treat the anæsthetic part of the skin with the faradic current, using ordinary electrodes, or, better still, the wire brush, or with the galvanic current, stroking the skin slowly back and forth with the cathode for three or four minutes. Sometimes we can see a result immediately after the sitting. Hysterical anæsthesia may often be removed in this way in a very short time.

Besides electricity we usually prescribe embrocations to irritate the skin, such as spirits of camphor, spirits of mustard, formic acid, spirits of thyme, etc., and also baths, and cold or hot local douches, combined with rubbing the skin. The action of internal remedies is extremely doubtful. Nux vomica or strychnine, tincture of valerian, etc., have been recommended.

It is very important to protect the anæsthetic part against external injuries. In anæsthesia of the trigeminus, particularly, we guard the eye as far as possible from the development of a neuro-paralytic keratitis, by a carefully applied occlusive bandage.

As an appendix, we will add here some illustrations (Figs. 59 to 63) to represent, in a diagrammatic fashion, the distribution of the sensory cutaneous nerves. These diagrams will be of service, both in the consideration of anæsthesia and in the diagnosis of the neuralgias to be described in the following chapters.

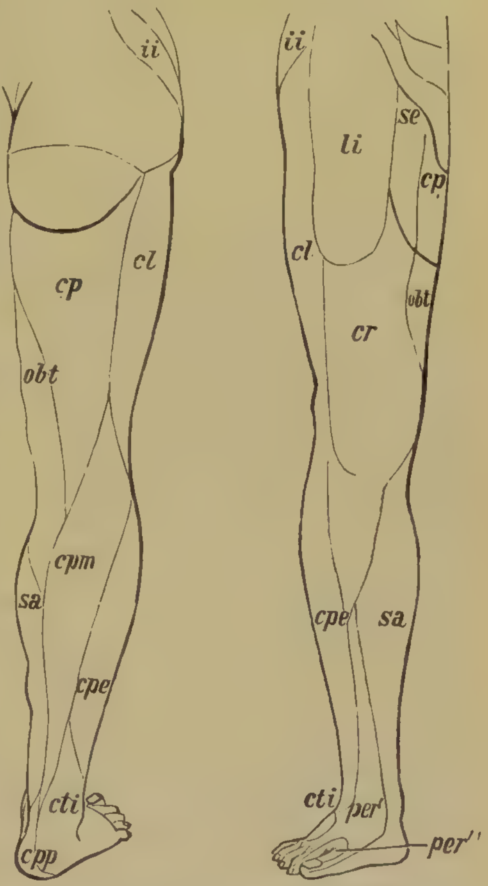

Fig. 62. Fig. 63.

Figs. 62 and 63.—Distribution of the sensory cutaneous nerves to the lower extremities. Fig. 62. Posterior aspect. Fig. 63. Anterior aspect. (From Henle.) *ii.* Ileo-inguinal nerve. *li.* Lumbo-inguinal nerve. *se.* External spermatic. *cp.* Posterior cutaneous. *cl.* Lateral cutaneous. *cr.* Crural. *obt.* Obturator. *sa.* Saphenous. *cpe.* Peroneal communicating nerve. *cti.* Tibial communicating nerve. *per'.* Superficial branch of the peroneal nerve. *per''.* Deep peroneal nerve. *cpm.* Posterior median cutaneous nerve. *cpp.* Cutaneous plantar nerve.

CHAPTER III.

NEURALGIA IN GENERAL.

Although every pain is, of course, excited by abnormal irritation of the nerves, still we are justified in giving a certain special variety of pain the name of neuralgia. The characteristics of this particular "nervous pain" are as follows: 1, it is felt exactly in the course or in the distribution of one or more special nervous trunks or nervous branches; 2, it is usually of very considerable intensity; and, 3, as a rule, it is not present continuously, but it shows manifest remissions and intermissions. It often comes on in single pronounced paroxysms of pain, which are due to definite causes, or which can not be referred to any evident external irritation.

Pathogenesis and Ætiology.—In many cases the cause of neuralgia is entirely unknown, but in other cases we can discover factors which may be regarded either as more or less direct exciting causes, or at least as predisposing causes, for the appearance of neuralgia; but in all these cases the precise form of action, and the peculiar nature of the disturbance produced in the nerves, are almost wholly unknown to us. At best we may suspect that perhaps sometimes we have to do with slight inflammatory changes in the nerve-trunks, with hyperæmia, exudation, œdema, etc.

We may mention the following as predisposing factors, which clinical observation has taught us: 1. Age. Neuralgia comes on most frequently in middle life, but it is also found in older people, and more rarely in children. 2. Sex exerts an influence so far that certain forms, such as trigeminal neuralgia, are seen more frequently in women, and other forms, such as sciatica and brachial neuralgia, are more common in men. Certain phases of sexual life, too, such as puberty, pregnancy, childbed, and the climacteric, favor the disposition to neuralgia. 3. The general neuropathic predisposition, which is hereditary in the majority of cases, is of great significance. Neuralgia often appears in people who suffer from other neuroses, or in whose families other nervous diseases, such as the psychoses, epilepsy, hysteria, or neurasthenia have repeatedly occurred. 4. The physical constitution also seems to be of influence. We often see neuralgia in anæmic people, or in those whose constitution has been impaired by physical and mental strain, by imprudent living, or by mental excitement.

We may mention, as exciting causes of neuralgia: 1. Cold, the action of draughts, wind, or wet—the so-called "rheumatic neuralgias." It is not perfectly clear how the cold acts in such cases. We usually assume that by its action slight anatomical (inflammatory ?) changes arise directly or in a reflex manner in the nerves themselves. 2. Mechanical and traumatic action. Among these are, first, wounds and contusions which directly involve the nerves. Thus, for example, extremely severe neuralgia sometimes arises from the pressure of foreign bodies, such as splinters of wood or of bones in wounds, on the branch of a nerve. We may also mention here the very severe neuralgia sometimes met with after amputations, which develops as a result of the so-called amputation neuromata. To this class, too, belong diseases in the neighborhood of nerves which act as mechanical irritants. Diseases of the bones or periosteum often lead to neuralgia in those nerves which run through bony canals or grooves. Lastly, tumors, aneurisms, herniæ, or the gravid uterus, may lead to neuralgia by pressure on the neighboring nerves; but we must call to mind that every pressure on a nerve does not lead to neuralgia in the same way, so that we must assume a special consequent change in the nerve in "neuralgia from compression."

The relation which certain infections and poisons have to the development of neuralgia is very important. In the first place, it is not impossible that many of the apparently "idiopathic" neuralgias are to be referred to infectious causes—an assumption which may be made, for instance, in intercostal neuralgia associated with an eruption of zoster (*q. v.*), or in many acute trigeminal neuralgias. Many neuralgias, however, also have a definite relation to other infectious diseases. We may mention in particular the malarial neuralgias, which are directly dependent upon the malarial infection, often come on at regular intervals, and are cured by specific treatment, namely, quinine. Neuralgia is often seen during the course, or as a result, of typhoid, small-pox, and similar acute infectious diseases, and in the secondary stage of syphilis. Among toxic substances we may mention especially lead, copper, mercury, and also alcohol and nicotine, as those which have a special relation to the development of neuralgia.

Neuralgia is also found in many constitutional diseases, in gout, and quite often in diabetes mellitus, and seems to bear a direct relation to the general disease; and hence, like many of the other neuralgias mentioned as symptomatic, it may be contrasted with the idiopathic neuralgia due to some direct affection of the nerves themselves. Finally, neuralgia is met with sometimes in distant nerves in diseases of other than nervous organs, like the sexual organs, and is termed "reflex neuralgia"; but we must be very cautious in accepting the theory of reflex neuralgias, since in most of these cases we have to do with other affections, such as hysteria, etc.

General Symptomatology of Neuralgia.—The neuralgic paroxysm begins either quite suddenly, or, more frequently, after certain prodromata, such as cold feelings, prickling, slight painful sensations, etc., have preceded it for some time. The pain during the attack is usually of the greatest severity, and is described as either burning and boring, or as shooting and tearing like lightning. There are frequently short temporary remissions of the pain. The location of the pain usually corresponds precisely to the distribution of the affected nerve, so that the patient can often point out quite definitely the anatomical course of the nerve. At the height of the attack there is often an "irradiation" or shooting of the pain into the territory of neighboring nerves. External irritants, such as cold air, mental excitement, and especially movements of the affected part, often produce an increase of the pain.

On physical examination, we notice, first, certain disturbances of sensibility. The skin in the neuralgic part often shows more or less anæsthesia, which is most apparent in the intervals between the separate attacks and immediately after them. Much more frequently, however, both during the attack and during the time when the patient is free from pain, there is hyperæsthesia of the skin and the parts beneath. There are certain definite points which are often very sensitive and tender even to light pressure. These are called painful points (*points douloureux*). They were first fully described by Valleix, in 1841, for the different forms of neuralgia, and they have considerable diagnostic importance, since they may often be found, not only during the attack itself, but also during the free intervals, although in a lesser degree. The painful points always correspond to certain places in the course of the trunk or the larger branches of the affected nerve, and are found especially where, in marked and deep pressure on the nerve, we can press on some firm part beneath. They are probably always due to an abnormal sensitiveness to pressure in the affected nerve itself. In many cases of neuralgia they may of course be wholly wanting.

Motor symptoms as well as sensory are not infrequent in neuralgia. Co-existing symptoms of paralysis must always be regarded as a complication caused by some coarse lesion of the motor nerves; hence in ordinary idiopathic neuralgia they are entirely absent. The co-existing symptoms of motor irritation, which are often seen, are usually directly dependent upon the neuralgia, however, and hence are to be regarded as reflex contractions, set up in the muscles by the great irritation of the sensory nerves.

Vaso-motor symptoms are often seen in neuralgia. In the face especially, in trigeminal neuralgia, we often see a marked pallor or a decided reddening of the skin and conjunctiva. Abnormal secretions, of tears or sweat, may also be seen during the attack or at the end of it. We do not know whether all these symptoms arise from direct or reflex nervous irritation. Trophic disturbances are noticed in various ways. During the attack we see eruptions of urticaria, or still more frequently of herpes vesicles, along the course of the affected nerves, as in herpes zoster. In severe and protracted neuralgias permanent changes in the tissues have been repeatedly seen in the parts supplied by the affected nerves.

Among these are a falling out or a whitening of the hair, or more rarely an abnormally great growth of hair, thickening or atrophy of the skin, staining or pigmentation of the skin, etc. Lastly we may mention that during the neuralgic attack we sometimes find a diminished frequency of the pulse.

The general nutrition of the body often does not suffer at all in neuralgia, but in many cases, especially when sleeping and eating are constantly disturbed by the attacks of pain, the disease gradually has a noticeable action on the whole constitution. The patient becomes pale and emaciated, and often the persistent and distressing pain is not without influence on his mental condition. He becomes irritable and inclined to melancholy. Patients have, in repeated instances, even committed suicide as a result of severe and incurable neuralgia.

The whole course of neuralgia shows the greatest diversity. As has been said repeatedly, the onset of the disease in separate attacks is the chief characteristic feature. The precise pathogenesis of these attacks is, of course, entirely unknown to us. These attacks usually come on every day or several times a day, or sometimes at greater regular or irregular intervals. They may last only a few minutes or several hours. During the period between the attacks many patients feel quite well, but some have still a certain sensitiveness of the skin. The disease as a whole sometimes lasts only a few days or weeks, but sometimes it persists with manifold variations for years and years, and, in a word, is not capable of improvement; although, on the other hand, there are recoveries after the disease has lasted for years. In many cases, of course, the disease depends upon the presence of some definite anatomical cause, such as a tumor, a disease of the bone, or an aneurism.

Many details as to prognosis and also as to the diagnosis of neuralgia will be spoken of in the following chapter.

General Treatment of Neuralgia.—Prophylaxis of neuralgia is possible in this way, that certain constitutional anomalies, such as anæmia or a general nervous predisposition, favor its appearance, as we have seen; and in attacking these conditions, we may recognize a factor which can prevent to a certain degree any subsequent development of neuralgia. It is still more important, in people who have already suffered from neuralgia, to prevent the return of the affection if we can. For this object we must first consider the strengthening of the whole body, in order to make it better resist the action of any causes of disease. The measures to be chiefly employed for this purpose are proper food, good air, baths, sea-bathing, cold bathing, gymnastics, etc. Of course, we must also particularly guard the part of the body that has once been attacked from any irritation, such as cold, mechanical irritants, or over-exertion.

In treating neuralgia itself we must always look first with great care for some causal factor, which may be accessible to treatment. This fulfillment of the causal indication is often possible in neuralgias which are due to mechanical causes. The extirpation of tumors, the excision of cicatrices, the removal of foreign bodies, the treatment of inflammatory new growths, of syphilitic affections, of aneurisms, etc., is in many cases attended by brilliant success, but, of course, in many other cases the underlying disease is unfortunately not amenable to successful treatment. We should also carry out a causal treatment in the neuralgias which are to be referred to general anæmia, to a general neuropathic constitution, to hysteria, etc. In such cases we must always ascribe great value to the general treatment, such as diet, manner of life, baths, iron, and nervines, as well as to the special treatment directed against the neuralgia; and the same holds true, of course, in the neuralgias occurring in diabetes, gout, and syphilis. Lastly, we may fulfill the causal indication in the malarial neuralgias. If neuralgia comes on at approximately regular intervals in persons who come from a malarial district, and who perhaps have already suffered from other malarial affections, the exhibition of quinine in large doses, twenty to forty-five grains (grm. 1·5–3·0) at once, will usually speedily cut short the attack. In obstinate cases in which quinine does no good, we should try arsenic, as Fowler's solution. In many toxic neuralgias, too, from lead, mercury, or alcohol, our first endeavor in treatment should be to remove the cause of the disease.

In all cases where the causal treatment can not be carried out, or where it alone is not sufficient, we must consider those numerous remedies and methods of treatment which correspond to the *indicatio morbi* and to the symptomatic indications. Starting with the hypothesis of an inflammatory condition of the nerve, we have often tried to exert a favorable influence on the disease by local derivatives, mustard plasters, irritating embrocations, such as spirits of mustard, veratrine ointment (two and a half per cent.), or tincture of iodine, or by blisters, or even by the cautery. The remedies first mentioned are used only in mild cases. Vesicatories, placed along the course of the affected nerve, or behind the ear in facial neuralgia, sometimes act very well in fresh cases, especially those of a "rheumatic" character. We resort to the hot iron chiefly in old and very severe cases, in which, indeed, especially in sciatica, some very favorable results have been thus obtained.

The local electrical treatment of neuralgia is more important and more efficient than the remedies mentioned. Although we do not know certainly how electricity acts, still it is unquestionably often attended with great success in the treatment of neuralgia. We many times secure an improvement in the symptoms, which is of course temporary, even in those cases where the special cause of the disease is not influenced by the electricity, although in idiopathic neuralgias in fresh, and sometimes even in old cases, we can often obtain a complete cure. There are no general rules with regard to the methods to be employed, since different specialists have their own favorite methods. The following forms of application are most in use and are most to be recommended: 1. Stabile action of the anode of a constant current on the affected nerve-trunk over as great an extent as possible, especially on any painful points. We must entirely avoid any great variations in the current or interruptions of it. We gradually increase the intensity of the current up to medium strength. The sittings should last three to six minutes, and sometimes even longer, and must be repeated daily. 2. In neuralgia of the larger nerves we should use a stabile descending (sometimes ascending) constant current, in which the anode is placed on the most central point of the nerve-trunk available, or on the vertebral column, and the cathode on different peripheral points. 3. The faradic current also frequently acts very well. We faradize the nerve either with a moderately strong "increasing" current, or we apply the wire brush to the skin over the affected nerves. The latter method is very painful, but it is often attended with excellent results. 4. Some electro-therapeutists, like Moritz Meyer, lay stress upon the stabile application of the anode of the constant current to any painful points on the vertebral column, such as have been described in many neuralgias by Trousseau.

As a general rule, it is always well to begin with a mild and very cautious use of electricity, and not to go on to the stronger currents until later. Electricity often acts brilliantly at once, during the attack of pain, but sometimes no improvement is seen until after several sittings. If, after two or three weeks, we obtain no result at all, after employing different methods of applying electricity, it is the best plan to give up electrical treatment entirely as not suited to the case.

In the treatment of neuralgia we must consider a number of internal remedies, as well as electricity; some of these act symptomatically, like the narcotics, while others have obtained the reputation of having a specific action. Among the latter, quinine has decidedly the greatest value. In severe cases quinine may do excellent service, not only in malarial neuralgias, although most surely in these, but also in "idiopathic" neuralgias. It is in these cases that the remedy is given in large doses. We begin with fifteen to thirty grains (grm. 1–2) a day, best given in one dose, and in severe cases we may increase to sixty or seventy-five grains (grm. 4–5) or more. We see the best results from quinine in trigeminal neuralgia, while in other forms, like sciatica, it is much less efficient. Some cures have been obtained by salicylate of sodium. Antipyrine, in fifteen to thirty grain (grm. 1–2) doses, antifebrine, in three to seven grain (grm. 0·25–0·5) doses, and phenacetine, in seven to fifteen grain (grm. 0·5–1) doses, have lately been very often used. Next in rank come arsenic and bromide of potassium. The former is given in pill form or as Fowler's solution, five drops three times a day, increasing gradually. Bromide of potassium acts only in large doses, forty-five to seventy-five to a hundred and fifty grains a day (grm. 3–5–10). Among the many other remedies which have been recommended, we may mention here ergotine, internally and subcutaneously, oil of turpentine, oxide of zinc, valerianate of zinc, tincture of gelsemium, aconitine, phosphorus, iodide of potassium, subcutaneous injections of osmic acid, ten to fifteen minims (grm. 0·5–1) of a one-per-cent. solution, etc.

In all severe neuralgias the use of narcotics, especially of morphine, is unavoidable. Morphine is used almost exclusively during the attack, and is best given as a subcutaneous injection of one-twelfth to one-sixth of a grain (grm. 0·005–0·01) in the vicinity of the painful part. The anodyne effect almost invariably follows, but in obstinate and protracted cases the patient gradually becomes accustomed to the remedy. We must then resort to still larger doses, and these, too, finally fail in their effect. Among the victims of the morphine habit we find many patients who have suffered or who still suffer from severe neuralgia, so that we must always be very cautious and conservative in the protracted use of morphine. We should be especially cautious before deciding to put the hypodermic syringe into the patient's own hand. Many physicians ascribe not only a palliative but a permanent benefit to injections of morphine in neuralgia. We sometimes see, in fact, that mild neuralgias recover under the exclusive use of morphine injections; but these are probably cases of spontaneous recovery. The internal use of morphine and opium preparations is decidedly inferior to the subcutaneous administration in certainty and rapidity of action. The external application of narcotic ointments, or embrocations, is much employed in practice, but it appears to be of advantage only in milder cases. We prescribe ointments with extract of opium (one to ten), extract of belladonna (two to ten), extract of opium and veratrine (one part of each to twenty of simple ointment), etc. We may also use chloroform (equal parts of chloroform and oil of hyoscyamus) and ether. Chloral and also paraldehyde are often prescribed in chronic neuralgias for their hypnotic effect. Croton chloral has been especially recommended in neuralgia. Finally, we must add that some physicians have praised the anodyne effect of subcutaneous injections of atropine, gr. $^{1}/_{120}$ to $^{1}/_{60}$ to $^{1}/_{20}$! (grm. 0·0005–0·001–0·003!) at a dose, sometimes even in cases where morphine does not act.

In severe cases the surgical treatment of neuralgia is often of great importance —the section of nerves, neurotomy, or the excision of a portion of the nerve, neurectomy—in order to prevent the union of the divided nerve. Without doubt this operation is often attended with success; but in some cases, of course, it has no effect at all on the disease, or else the neuralgia returns with its old severity after a temporary improvement. We can understand the success of neurotomy in cases where we can assume that the cause of the abnormal sensory irritation is peripheral to the point of section; but observations are reported in literature where the operation has had a favorable influence even on central neuralgias. The operation is to be proposed only in severe cases, where all other remedies have been tried in vain, and the patient can always be promised the possibility, or even the probability, of success, but never the certainty of it. Besides section of the nerves, nerve stretching has lately been frequently tried in neuralgia—sometimes, but not always, with distinctly good results.

The chief question as to the use of baths arises only in the treatment of neuralgias in the domain of the nerves of the extremities, especially in sciatica, and therefore these and massage will be spoken of more in detail in connection with the separate forms of neuralgia.

We see, then, that a large number of remedies are at our command in the treatment of neuralgia, and that the choice among them is not always easy. In any given case we look for the causal indication, and try to fulfill it if possible. In the many cases, however, where we fail to find this, we must first of all try to alleviate the pain, for which purpose, if external derivatives do not suffice, our most effective remedy is morphine. We must then lay out a special plan of treatment. We try electricity, or, if this be not practicable, one of the other remedies mentioned above. We put most trust in quinine, especially in fresh cases; among other remedies, in anæmic persons, arsenic, and, in more robust persons, bromide of potassium are of service. If all these and similar remedies give no aid, in proper cases we may still hope for success from operative interference, or else we must confine ourselves simply to a purely symptomatic treatment with narcotics.

CHAPTER IV.

THE INDIVIDUAL FORMS OF NEURALGIA.

1. Neuralgia of the Trigeminus.

(*Prosopalgia. Tic douloureux. Fothergill's Face-ache.*)

Ætiology.—Trigeminal neuralgia is one of the commonest and most important neuralgias. In its origin many causes and predisposing factors of all sorts play a part, as we have learned in the preceding chapter. Many cases, especially the milder ones, come from taking cold. Sometimes we can not find any definite cause. Malarial neuralgias are very often localized in the region of the trigeminus. The other especial causes which may give rise to trigeminal neuralgia are diseases of the cranial bones and periosteum, very often diseases of the teeth, caries, exostoses, and anomalies in development and position; and also diseases of the nasal and frontal cavities as well as of the middle ear. Romberg found an aneurism of the internal carotid, pressing on the Gasserian ganglion, as a cause of a severe and incurable case. We have since seen a precisely analogous case. Lastly, excessive use of the eyes is not infrequently related to the development of trigeminal neuralgia.

Symptoms and Course.—The attacks of pain in neuralgia of the fifth pair are usually quite intense, and in severe cases they may attain a most distressing and terrible severity. They arise either entirely without cause, or from some slight influence, like washing, taking physical exercise, or mental irritation. The pain is usually limited to the distribution of the different branches of the trigeminus, but it sometimes shoots into the occiput, the back of the neck, the shoulders, etc. We can often perceive reflex twitchings in the face, especially blepharospasm, and twitching of the corners of the mouth. The vaso-motor disturbances are noticed at first as abnormal pallor, but later usually as quite an abnormal redness of the face and conjunctiva. In neuralgia of the upper two branches we often see, during the attack, an unusually great secretion of tears. An excessive flow of saliva and an increased secretion from the nasal mucous membrane are more rare. Sometimes, but still quite rarely, we see herpetic eruptions in the course of the affected nerve, zoster frontalis, herpes of the conjunctiva, etc. In some cases, too, more severe diseases of the eyes, belonging to the category of neuroparalytic ophthalmia, have been observed. In neuralgias that have lasted longer, we often see still further trophic disturbances: changes in the skin and subcutaneous cellular tissue, the hair turning gray or falling out in the frontal region, etc.

Most trigeminal neuralgias are situated, not in the whole distribution of the nerve, but only in one or more of its branches (see Fig. 57, page 513). We accordingly distinguish : 1. *Neuralgia of the first branch* (*ophthalmic neuralgia*) which is especially frequent as supra-orbital or frontal neuralgia. In this we find as a rule, that pressure on the point of exit of the nerve at the supra-orbital foramen is more or less painful. More rarely we find painful points also on the nose, at the inner angle of the eye, or the parietal eminence, etc. 2. *Neuralgia of the second branch* (*supra-maxillary neuralgia*) is most frequent in the distribution of the infra-orbital nerve, infra-orbital neuralgia, with the chief painful point at the infra-orbital foramen, and others on the zygoma, on the upper lip, etc. 3. *Neuralgia of the third branch* (*infra-maxillary neuralgia*), whose most frequent seat is in the territory of the inferior alveolar nerve ; but neuralgia also occurs in the temporal region. in the auriculo-temporal nerve, and in the tongue, in the lingual nerve. The chief painful point is at the mental foramen.

The general course of neuralgia of the fifth pair differs very much in different cases. We see all forms, from the mildest, which rapidly passes off, to the severest and incurable types, which may drive the patient to despair and even to suicide. In general, neuralgia of the first branch usually belongs to the relatively milder forms ; neuralgia of the second, and especially of the third branch to the severer forms. Trousseau has termed a particularly severe form "epileptiform neuralgia," although we can find no definite relation between this disease and genuine epilepsy. We can find no cause for epileptiform neuralgia; the attacks of it come on with the greatest intensity either after brief pauses or after intervals of weeks and months, and they defy all attempts at cure with the greatest obstinacy. It is worthy of note that this form appears in individuals with the neuropathic taint.

Diagnosis.—In all pronounced cases the diagnosis of trigeminal neuralgia is easy. We must regard accurately the distribution of the pain, its paroxysmal onset, and the points of pressure. Otherwise in a superficial examination we may of course confuse it with inflammatory affections of the bones and periosteum, with genuine tooth-ache, with migraine, and with other forms of headache and face-ache.

Prognosis.—The prognosis is never to be made with complete certainty. It is most favorable in fresh cases which have a manifest cause, which may be removed, as a basis ; but if the affection is due to a coarse anatomical cause which can not be removed, or if we are dealing with old cases "which have become habitual," the prognosis, unfortunately, is often utterly unfavorable.

Treatment.—The treatment of trigeminal neuralgia rests entirely upon the principles given in the preceding chapter. In searching for the causal indications we must look chiefly, in neuralgia of the second and third branches, for diseased teeth, and also in all cases for any affections of the nose, of the frontal sinuses, or of the middle ear. Carious teeth, which are painful and which seem to have any relation to the neuralgia, should always be removed, and any of the other affections mentioned are to be treated on special principles.

Of other remedies we use first of all electricity, the anode to the painful points, with the cathode on the back of the neck, or the wire brush, etc.; we also use quinine, Fowler's solution, and in severe cases narcotics. In cases where the neuralgic attacks come on with approximate regularity, quinine especially often does excellent service; we give fifteen to twenty grains (grm. 1–1·5) at first, two or three hours before the expected attack. If quinine does no good, we try arsenic in not too small doses, and if these remedies are unsuccessful we may try one of the other drugs recommended: butyl chloral (croton chloral) in two- to five-grain capsules (grm. 0·1–0·3), or, according to Liebreich's formula :

℞ Croton chloral...................... gr. lxxv– ʒ ijss. (grm. 5–10);
Glycerine................ .... ʒ vj. (grm. 20);
Aquæ destill....................... ℥ iv. (grm. 120). M.
S. One or more tablespoonfuls every five or ten minutes.

We may also give tincture of gelsemium sempervirens, five to twenty drops several times a day; aconitine in pills of $^{1}/_{200}$ to $^{1}/_{120}$ of agrain (grm. 0·0003–0·0005), three to five times a day; nitrite of amyl, ammoniated copper, in powders of one or two thirds of a grain (grm. 0·02–0·04), oil of turpentine, [cannabis indica], etc. Special indications for all these remedies can not be given, so that we are recommended simply to try different ones. In desperate cases Trousseau has tried very large doses of opium, which he gradually increased until he gave two to three drachms (!) (grm. 8–12) a day. Sometimes an attack may be diminished or shortened by compression of the carotid.

If a severe neuralgia persists in spite of rational treatment by electricity or drugs, we should not delay too long in proposing to the patient operative treatment if possible. In frontal and infra-orbital neuralgia especially the section of the nerve is a comparatively slight operation, which—of course with many failures—has many excellent results to show. The full description of the technicalities of the operation, as well as the description of nerve-stretching, and of the ligature of one carotid, which has been done in some desperate cases, must be left to the text-books on surgery. [Unfortunately the only operations that promise permanent benefit are the more serious ones of resection of the nerve at the foramina, at the base of the skull, or excision of the Gasserian ganglion.—K.]

2. Occipital Neuralgia.

Of the neuralgias involving the sensory region of the upper four cervical nerves, neuralgia of the occipitalis major is the most frequent and practically the most important. Besides the factors to be considered in all neuralgias, we must pay particular attention, in regard to ætiology, to diseases of the upper cervical vertebræ—caries and new growths. The painful paroxysms may attain the greatest severity. They are usually located in the two occipital nerves at once, being accordingly bilateral, although often more severe on one side than on the other. Painful points are most frequently found midway between the mastoid process and the upper cervical vertebræ. Vaso-motor disturbances, falling out of the hair, etc., have been often observed.

The prognosis is comparatively favorable in cases which have no severe anatomical disease, such as spondylitis, as a basis. The most efficient remedies are strong cutaneous irritants to the back of the neck, vesicatories in fresh cases, the constant current, and injections of morphine.

Other neuralgias in the distribution of the cervical plexus are rare. They occur in the distribution of the occipitalis minor, which, according to Seeligmüller, is quite frequently due to syphilis, and then is easily cured by iodide of potassium, and in the distribution of the great auricular and the supra-clavicular nerves. A phrenic neuralgia even has been described, in which the pain extends along the course of the phrenic nerve to the insertion of the diaphragm; but this is at all events very rare.

3. NEURALGIA IN THE REGION OF THE BRACHIAL PLEXUS.

(*Cervico-brachial Neuralgia.*)

Brachial neuralgia is, on the whole, rare, and it is hardly ever strictly limited to the distribution of a single nerve. In general, the radial and ulnar nerves are rather more frequently affected than the median. We also see at times neuralgia of the internal cutaneous nerve. In regard to ætiology, we have to mention first wounds and contusions of the nerves, and also cicatrices and foreign bodies. The amputation neuralgias also belong to this class. Severe neuralgia in which the pain may extend over a greater part of the whole arm is sometimes seen after injury of the fingers (crushing, cuts, etc.). In some of these cases we may have to do with an ascending neuritis, starting from injury of a small nerve branch; in other cases there are probably cicatricial contractions, thickenings of the neurilemma, or small neuromata developing after the injury, which excite the pains. The "amputation neuralgias," which are often very severe in amputation stumps, are due to neuromata which form on the cut ends of nerves. Severe neuralgia often arises from pressure on the brachial nerves, as in cases of tumors in the axilla (cancer, etc.), aneurisms of the aorta, etc. Rheumatic neuralgia may also occur. Double brachial neuralgia should always excite suspicion of disease in the neighborhood of the upper posterior nerve roots, especially of cervical pachymeningitis, spondylitis of the lower cervical vertebræ, etc.

We have little to add concerning the special symptomatology of brachial neuralgia. The pain is usually ascribed to the whole course of the nerves without being very exactly localized, as we have stated. Painful points are sometimes found over the brachial plexus, over the radial on the external surface of the upper arm, over the ulnar in the sulcus at the internal condyle, over the median at the inner border of the biceps, and where the cutaneous nerves emerge from the fasciæ. Vaso-motor and trophic disturbances have sometimes been seen, as "glossy fingers," a peculiar, shiny, atrophic condition of the skin of the fingers. In severe neuralgia a pronounced atrophy of the whole arm has also been observed. The diagnosis is usually easy; we must bear in mind only the risk of confusion with articular affections.

The treatment of brachial neuralgia is often no easy task, since we frequently have to do with protracted and obstinate diseases. The causal indications should be met if possible; besides these we should make use chiefly of electricity (the descending galvanic current along the affected nerves). We may also use narcotics, morphine, cocaine, sodic salicylate to alleviate pain, antipyrine, phenacetine, etc.; and lastly local warm peat baths. In severe cases we must consider the possibility of surgical interference, such as nerve-stretching, the extirpation of cicatrices, etc.

4. INTERCOSTAL NEURALGIA.

(*Dorso-intercostal Neuralgia.*)

The neuralgias of this class are almost always pure intercostal neuralgias, since the posterior dorsal branches of the thoracic nerves are only exceptionally involved. The middle intercostal nerves, from the fifth to the ninth, are usually affected, one or more of them being attacked at the same time. The affection is much more frequent on the left side than on the right.

In regard to ætiology, it is important to remember that obstinate intercostal neuralgias are often a symptom, and for a long time the only symptom, of severe organic disease, especially affections of the ribs; diseases of the vertebræ, such as caries and carcinoma; diseases of the cord, such as tabes dorsalis, spinal meningitis, and tumors; and aneurism of the aorta. Genuine idiopathic intercostal neuralgia, however, is often met with, as well as these symptomatic forms, especially in anæmic and nervous women and girls in youth and middle life. Traumatic lesions of the intercostal nerves and taking cold also play a part in the ætiology.

The pain in intercostal neuralgia may attain a remarkable severity, and is usually increased by any considerable movement of the thorax. Hence the patient avoids deep inspirations, coughing, loud talking, etc., as much as possible. We usually find three painful points—one near the vertebral column, one somewhere in the middle of the nerve, and a third near the sternum or over the rectus abdominis. We may mention, among the trophic disturbances, the comparatively frequent occurrence of herpes zoster. In such cases we probably always have an actual neuritis of one or more nerves. The pain precedes the eruption of zoster, or comes on at the same time with it. It often lasts for a long time after the cutaneous affection has healed. The formation of zoster has usually been considered until the present time a "trophic disturbance," but recent careful anatomical investigations (A. Dubler) favor the hypothesis that the formation of vesicles arises simply from a direct extension of the inflammatory process from the terminal branches of the nerves to the skin. It is worthy of note that the attacks of zoster often exhibit a certain epidemic and sometimes even an endemic distribution, so as to suggest an infectious agency. The almost constant swelling of the neighboring lymph-glands, in the axillæ, at the lower border of the pectoral muscle, etc., perhaps supports this view.

The course of intercostal neuralgia depends chiefly upon the ætiology of the affection. Primary neuralgias are often quite obstinate, but on the whole they usually give a favorable prognosis. The differential diagnosis between genuine intercostal neuralgia and rheumatic affections of the muscles, incipient pleurisy, etc., is not always easy. In these cases a careful physical examination, a consideration of the localization of the pain, of the presence of painful points, and of the whole course of the disease, are needed to protect us from errors.

The treatment is governed by the general rules given in the previous chapter. Blisters often act very well in fresh cases. Electricity is given by the faradic brush or the constant current; with the latter, the cathode is placed on the vertebral column, and the anode on the lateral and anterior painful points, using quite a strong stabile current. In severe cases we can not avoid injections of morphine. Herpes zoster heals by simple treatment with salves or by dusting on powders, like one part of zinc oxide to two of starch.

Mastodynia (*Neuralgia of the Mammary Gland*).—Mastodynia ("irritable breast" of Astley Cooper) is to be considered as a special neuralgia in the distribution of the intercostal nerves. It occurs almost solely in women, after the age of puberty, and is a very painful, distressing, and obstinate affection. The pain is either continuous, or it comes on in separate paroxysms, sometimes accompanied by vomiting. The whole breast is extremely sensitive to the touch. We know little that is definite as to its ætiology. Anæmia, hysteria, and traumatic action seem to have some influence on it. We sometimes feel little nodules in the breast, which are very painful (*tubercula dolorosa*, neuromata?), and which may sometimes give rise to the suspicion of the development of carcinoma.

The disease may last for years. Treatment is difficult. Warm packs to the breast, bandaging the breast, and especially narcotics, may afford relief. Electricity may be of distinct service. In the worst cases operative interference has been attempted—amputation of the breast, or extirpation of the painful nodules—but its results are uncertain.

5. NEURALGIA IN THE REGION OF THE LUMBAR PLEXUS.

As the neuralgias of this class are rare, and show few peculiarities, we will content ourselves with a brief account of the most important forms.

Lumbo-abdominal neuralgia causes pain in the lumbar region, which shoots into the buttocks, the hypogastrium, and the genitals. Crural neuralgia is seated, in part, in the region of the external anterior cutaneous nerve of the thigh, and in part in the region of the cutaneous branches of the crural nerve, the internal and middle cutaneous. Its distribution over the cutaneous distribution of the great saphenous nerve, the inner portion of the calves, and the inner border of the foot, is especially characteristic. In obturator neuralgia the pain extends along the inside of the thigh, down to the vicinity of the knee-joint (see Figs. 62 and 63, page 515).

In their individual characteristics, all these neuralgias agree with what has been said in the previous chapter. The diagnosis is not always easy, and we must pay special attention to avoid confusing them with affections of the bones and joints, with lumbago, renal colic, etc.

6. SCIATICA.

(*Ischiatic Neuralgia. Malum Cotunnii.*)

Ætiology.—Next to trigeminal neuralgia, neuralgia of the sciatic nerve is by far the most frequent, and practically the most important form of neuralgia. In contrast to most of the other neuralgias, it is more frequent in men than in women. Cold, wet, and over-exertion of the leg are found to be the most frequent ætiological factors. More rarely venous stasis in the pelvic veins (hæmorrhoids) and habitual constipation give rise to the development of sciatica. We see symptomatic neuralgia in the region of the sciatic nerve in pelvic tumors, caries of the sacrum, and analogous affections. Other traumatic influences, and compression of the nerve, as in constant, uncomfortable sitting, are sometimes evident causes of the disease. Pressure of the gravid uterus on the sciatic plexus may sometimes excite sciatica, and in women it has been seen as a result of delivery by forceps.

Symptoms and Course.—The pain, coming on usually with mild prodromata and gradually increasing to severe paroxysms, generally begins at the posterior surface of the thigh, in the vicinity of the sciatic notch. From this point the lightning-like pains shoot down, usually into the peroneal region, the outer part of the leg, and the outer border and top of the foot, or more rarely into the tibial region, the sole of the foot. They either come on in characteristic neuralgic paroxysms, or they are more continuous, and are then described by the patient as "burning," "boring," and the like. They are often worse at night. In severe cases the leg can scarcely be moved, owing to the pain, so that walking becomes very difficult or almost impossible, and the affected leg is kept quiet in a slightly flexed position. Very often the pain comes on after long standing or sitting. Painful points are often found along the course of the sciatic, over the gluteus maximus, or at its lower border, in the popliteal space (tibial nerve), at the head of the fibula (peroneal nerve), at the malleoli, on the top of the foot, etc.

Besides the pain, we often find other disturbances of sensibility in the affected leg, such as paræsthesia, hyperæsthesia, or slight anæsthesia. Reflex muscular tension, tremors, and even complete clonic spasms have been repeatedly observed in severe cases. A slight stiffness and weakness of the leg are very often found. A slight atrophy of the muscles often develops, but the higher degrees of atrophy indicate that there are serious anatomical changes in the nerve. Eruptions of zoster have been repeatedly observed, but, on the whole, it is rare.

The disease lasts several weeks, although sometimes, in many obstinate cases, it continues for months and even years; but, except in cases which have an incurable anatomical lesion as an underlying cause, the termination is at last usually favorable. Relapses are, of course, quite frequent.

Diagnosis.—The diagnosis of sciatica is easy in the majority of typical cases, but it may sometimes be quite difficult. It is chiefly confused with lumbago, an acute coxitis, nervous coxalgia (*vide infra*), and psoas abscess. We must also bear in mind the occurrence of sciatic pain in the beginning of tabes dorsalis. In doubtful cases we can decide only after the most careful physical examination, embracing every part and function, and by considering the localization of the pain and of the painful points.

Treatment.—Sometimes we can obtain favorable results by fulfilling the causal indication. In regard to this we may mention especially the improvement of many cases of obstinate sciatica, associated with habitual constipation, by methodical treatment with laxatives, especially by the springs at Marienbad, or Kissingen, and also improvement after the removal of tumors, foreign bodies, etc., when they exist.

In regard to the treatment of ordinary sciatica, we must first take care, in all severe cases, to keep the leg perfectly quiet and in a good position during the painful paroxysm. Usually the local application of warmth, warm poultices, or bandages, is grateful to the patient. Sometimes a vapor bath gives real relief. More energetic local derivatives, blisters, or even local blood-letting, are of especial use in cases of "rheumatic origin." If the pain be very severe, a subcutaneous injection of morphine is the only certain remedy, and is sometimes indispensable. Embrocations of narcotics are also frequently prescribed in practice.

Of the other remedies to be considered we may mention, in the first place, electricity and massage. In electrical treatment we usually employ quite strong descending currents with large electrodes, which we let act on the nerve for five or ten minutes a day, while we include one portion of the nerve after another in the current. Where there is much stiffness in the leg, we open and close the current a few times, in order to excite muscular contraction. Many cases are suitable for the use of the faradic current, especially for the wire brush. Besides electricity, massage has often given excellent results in sciatica. Details of the technique to be employed may be found in the special treatises on this important method of treatment.[*]

Besides electricity and massage, baths deserve special consideration in tedious cases. Good results are often obtained from the indifferent thermal baths, such as Teplitz, Wildbad, and Wiesbaden. We would also recommend warm local douches and hot sand baths, like Köstritz, as particularly efficient.

In general, we can promise but slight results from the very many internal remedies recommended against sciatica, but, if there be a suspicion of syphilis, we must try iodide of potassium. Quinine has usually no effect in sciatica. We sometimes see somewhat better results from the exhibition of sodic salicylate, antipyrine, phenacetine, etc. We have also very rarely seen any definite action from oil of turpentine, which is much used, especially in England. Some good results have lately been obtained by injections of osmic acid (see page 520).

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[*] Busch, "Allgemeine Orthopädie, Gymnastik u. Massage," Leipsic, Vogel, 1882; Schreiber, "Praktische Anleitung zur Behandlung durch Massage," Vienna, 1883 [translated by Mendelson, Philadelphia, 1887]; Reibmayr, "Die Massage und ihre Verwerthung in der prakt. Medicin," Vienna, 1883, etc. [Graham, "A Treatise on Massage," New York, 1890].

In very severe and obstinate cases, in which all other remedies have been tried in vain, we are justified in proposing to the patient to try nerve-stretching. This operation has been attended with very good results in some cases—but unfortunately, of course, not in all. Some favorable action in old cases has been ascribed to the use of the hot iron, and finally it may be mentioned as a curiosity that different observers claim to have obtained a recovery from sciatica by cauterization of the lobe of the ear!

[Mention should be made here of a method of treatment advocated by Weir Mitchell, which is occasionally useful in other forms of neuralgia as well as in sciatica. It consists in absolute rest of the leg by application of a splint. With this he combines the application of cold, by an ice-bag over the length of the nerve, which may be continued for a number of hours.—K.]

7. NEURALGIA OF THE GENITALS AND THE RECTAL REGION.

Neuralgic affections of the parts named are not frequent, but still a number of cases have been described by different observers. The pain has its seat either in the external genitals, or in the urethra, or in the anal and perineal region. The most frequent form is spermatic neuralgia ("irritable testis" of Astley Cooper), in which there is the most intense pain in the spermatic cord and the testicles, which is almost always associated with an extreme hyperæsthesia of the affected parts. The treatment of this form of neuralgia by narcotics and electricity is often unsuccessful, so that in severe cases resort has sometimes been had even to castration. In women, genuine uterine and ovarian neuralgia seem to occur especially as one symptom of hysteria.

Coccyodynia is the name of a form of severe pain in the coccygeal region, seen usually in women, which is much increased by walking, defecation, etc. The affection is so distressing that operations have been repeatedly performed on account of it to remove or to cut around the coccyx. We have twice seen this same symptom as a complication of tabes.

CHAPTER V.

NEURALGIA OF THE JOINTS.

(*Articular Neuroses.*)

NEURALGIA of the joints, first described by the English physician Brodie, was first generally known in Germany after Esmarch proved, by publishing many observations, that apparently severe and very painful diseases of the joints are often found, at the basis of which there is no discoverable anatomical disease of the joint, and which we are therefore justified in regarding as nervous affections. Since the localized pain in the joint is the chief symptom in most of the cases of this class, the term "neuralgia of the joints" has been quite fitly chosen, although we do not find here such typical and paroxysmal attacks of pain as in genuine neuralgia, and although, too, a number of other symptoms are usually present, which are not seen in genuine neuralgia.

We see neuralgia of the joints chiefly in nervous hysterical persons, and therefore more frequently in women and girls than in men.

We can very often make out a psychical cause for the origin of the disease. The affection is often caused by injuries which affect the joint, and which would be in themselves without significance, if they were not associated with a decided fright, and did not direct the patient's thoughts to the affected limb. Articular neuralgias do not, therefore, belong to the pure neuralgias, but are symptoms of hysteria (traumatic hysteria). At any rate, the name of articular neurosis is more suitable than that of articular neuralgia.

Either immediately after such an occurrence, or often only some weeks later, the patient begins to complain of pain. The knee- or hip-joint is almost always affected, only rarely the joints of the upper extremity. The pain is continuous, but it is more severe at times, especially on motion, or mental excitement. At other times, especially if the patient's attention be diverted from the trouble, it seems to diminish to a considerable extent. It is mainly localized in the joint, but the whole leg is often painful. Patients are usually very sensitive to pressure, or jarring, and sometimes we may even discover some painful points on pressure over the joints. The patients can not walk at all, or at least walking is very painful, and they limp badly. In severe cases, especially if the excessive sympathy of those about them reduces the patients' power to resist their suffering, they become completely bedridden for weeks or months. There is usually a decided weakness in the affected leg, which is almost always associated with great muscular rigidity and tension. The leg is extended, or flexed and rotated inward, in just the same way as in genuine coxitis.

The diagnosis of articular neurosis is often quite difficult, but it is almost always possible, with long observation of the case. At first, of course, the disease often seems to be a severe affection of the joint, on account of the great pain, the rigid position, and the complete inability to use the leg. The experienced physician, however, is usually struck by the absence of any definite physical changes in the joint, especially of swelling, and also by the changes in the intensity of the complaint, by the influence of mental emotion on the suffering, and finally by the general impression he gets from the patients, the way they behave, and the contrast between their great complaint and their usual (though, of course, not invariable) good appearance, appetite, and undisturbed sleep. In doubtful cases an examination under chloroform is very advisable. With this, contractures apparently the most severe vanish, and the normal character and mobility of the joint become plainly manifest.

As soon as the diagnosis of an articular neurosis is made the treatment has quite definite indications. All embrocations, poultices, bandages, etc., are to be laid aside. The patient is to be brought to the conviction that she can walk, if she will only first learn to will to walk again. We make the patient practice walking methodically ; these attempts at first prove very poor and apparently distressing to the patient, but they often lead to better results very speedily. These exercises are very much aided by electrical treatment of the joint, passing a strong current through it or using the faradic brush, and also by local cold douches and massage. Under some circumstances the use of internal remedies may be indicated, in many cases with regard only to the mental condition. We give iron to anæmic patients, and also the nervines. (See the chapters on hysteria and traumatic neuroses.)

CHAPTER VI.

HABITUAL HEADACHE.

(*Cephalæa. Cephalalgia.*)

In addition to the neuralgias, we must speak here of habitual or "nervous" headache, an affection which is very often met with in practice, but in regard to whose precise causes or whose special nature our knowledge is still in many respects very unsatisfactory.

We do not term the symptomatic headaches, so often observed, "nervous headaches." The former come on in acute febrile infectious diseases, in pronounced general anæmia, in the different anatomical diseases of the brain and its membranes, of the skull, the frontal sinuses, etc. Just as little ought we to confuse habitual headache with other painful and well-characterized affections, especially typical neuralgia in the frontal branch of the trigeminus, or in the occipital nerves, or with genuine migraine or hemicrania (*vide infra*). Those cases, rather, belong to this class in which the headache forms to a certain degree a disease in itself, and is the sole, or at least the chief, symptom of which the patient complains, and for which he seeks aid. We know no definite anatomical basis for these cases. We usually assume disturbances of circulation and of the fine nutrition as the special causes of headache ; but it is only in a very few cases that anything definite as to the form of these changes can be stated. We can also state little that is definite as to the special place where these pains arise. We do not know whether painful irritations may arise in the brain-substance itself. The cerebral meninges, however, especially the dura mater, are decidedly sensitive, and hence they may usually be regarded as the special seat of headache.

The manifold character of the circumstances under which headaches arise renders it probable that the causes of headache are very different in different cases. We have to do either with persons who seem perfectly healthy in other respects, or with weak and anæmic people, or again with robust, very well nourished, "full-blooded" persons with red faces. Hence we look for the cause of the pain, according to the general constitution of the patient, either in an abnormal hyperæmia or anæmia of the brain and its membranes—a hyperæmic or anæmic cephalalgia. We very often find headache, too, as the chief symptom in nervous, neurasthenic people—neurasthenic cephalalgia. To this class belong especially people who are overworked physically and mentally—scholars, officials, students before an examination, etc. If we believe that we can make out "rheumatic" influences, such as taking cold, or toxic influences, such as alcohol, nicotine, or chronic lead poisoning, we speak of a rheumatic or a toxic cephalalgia. Patients with habitual headache often suffer at the same time from chronic gastric disturbances or habitual constipation, so that the latter disorders, in many cases, stand perhaps in a causal relation to the headaches. Finally, it is a very important point that headache is sometimes associated with chronic diseases of neighboring organs, especially of the nose, the naso-pharynx, and the ear. In very many cases, however, we can find no definite cause at all for the affection, so that we have to do with a genuine idiopathic disease. It is worthy of mention, merely, that in not very rare cases a pronounced hereditary predisposition to habitual headache seems to exist. [One of the frequent causes of persistent headache is eye-strain due to the persistent unconscious effort of the muscles of accommodation to overcome some error of refraction, such as astigmatism or hypermetropia. In some cases this eye-strain may be associated with insufficiency of some of the external muscles of the eye. This condition may be associated with normal visual power, so that the patient often maintains that there can be no trouble with the eyes ; this is especially true where the refractive error is slight, less than one dioptric. It is therefore essential in every case of persistent headache, to have a thorough examination of the eyes, in order tó determine whether there is astigmatism or hypermetropia. The adjustment of proper glasses, or, in rare cases, tenotomy of some of the external muscles, will often give marked relief, although even in these cases attention must also be paid to the general condition.—K.]

Habitual headache is a chronic disease. It may last for months or years, or even through the whole life, either being present continually, or, what is more frequent, coming on in separate attacks and lasting several hours or days. These attacks sometimes come without any evident cause, but they may often be referred to definite influences, to mental excitement, physical exertion, errors of diet, etc. The pain is felt either in the forehead or in the occiput, or sometimes over the whole head. It is sometimes limited to a definite and quite sharply defined part of the head. The precise form of the pain is described in different ways, either as boring, or tearing, or as if the head were pressed together from without, or as if it would split open. In many cases the intensity of the pain is not great; there is merely a dizziness and a feeling of "pressure" in the head, but in other cases the pain is very severe. In such cases there is also at times a pronounced hyperæsthesia of the skin of the head, so that it may cause pain even to touch the hair.

The general health is almost always disturbed in headache. The patient can not work, he is often ill-tempered and irritable, and he loses his appetite. We sometimes see more marked gastric symptoms, especially nausea and vomiting, and sometimes copious perspiration. Severe cases of the disease are of great importance, since by an attack the patient is rendered almost wholly unable to attend to his business.

The treatment of headache is always a difficult task. In the first place, of course, we should try to adapt our treatment to the ætiology of the disease if it is evident. We should therefore never neglect to make a thorough examination of all the organs which are to be considered (nose, ear, stomach, heart, kidneys [eyes], etc.). In such a case the existing primary disease requires special treatment. If there is any suspicion of syphilis, which must especially be considered if the pain is worse at night, we must first of all try iodide of potassium. For anæmic patients we prescribe iron, arsenic, a country residence, strengthening diet, etc. We order full-blooded persons, especially if they also suffer from indigestion, to drink bitter waters, or we send them to health resorts like Marienbad or Carlsbad. Nervous headaches in hysterical and neurasthenic patients require a rational general treatment: electricity, general faradization, galvanism to the head, or to the sympathetic, cold-water cures, etc. For persons who have overworked, we most urgently advise complete physical and mental rest. We send them to the country or to try sea-bathing.

The number of symptomatic remedies recommended to relieve headache is very considerable. In most of the tedious cases the patient himself has learned to know his disease perfectly well. Many know that there is no remedy for "their old headaches," and they merely desire to rest, waiting until the pain ceases of itself. Others have become accustomed to employ certain domestic remedies: they put poultices on the head, take a cold or a hot foot-bath, put a mustard plaster to the back of the neck, bathe the forehead with cologne-water, bind a towel tightly about the head, drink strong tea, smell ammonia or "smelling-salts," etc. We sometimes see good results, although frequently we do not, from the internal remedies. These may be used, either during the attack or for a longer period, to prevent the return of the pain. There are no special indications for the different remedies, so that we must try to see which one is the most efficacious. Antipyrine is most used of late in doses of seven to twenty grains (grm. 0·5–1·5). Its efficacy in migraine is undoubted, but it sometimes gives distinct relief in other forms of headache. We may also use antifebrine, in doses of three to seven grains (grm. 0·25–0·5), phenacetine, seven to fifteen grains (grm. 0·5–1), and quinine, six to ten grains (grm. 0·3–0·75). In headaches which come on after exposure to cold, draughts, etc., we may try sodic salicylate, half a drachm to a drachm (grm. 2–4). Of other remedies we may mention paullinia serbilis (guarana, which contains caffeine), in seven to thirty grain powders (grm. 0·5–2), three to six one-grain ergotine pills (grm. 0·05) a day in hyperæmic headache, potassic bromide half a drachm to a drachm (grm. 2–4), arsenic, [cannabis indica], etc.

Electrical treatment (*vide supra*) has given decidedly good results in many cases, but we must always begin it with great caution, and try first what method is best borne. Cold-water cures, too, are sometimes beneficial, or residence in the country, at the seashore, or in the mountains.

We can sometimes do the patient good service with the remedies mentioned, but in other cases the evil obstinately defies all attempts at cure. Then, however, the patient has still the encouragement left that the disease often ceases at last spontaneously in advanced age, after lasting years and years.

CHAPTER VII.

ANOMALIES OF THE SENSE OF SMELL.

Anomalies of smell, which depend upon a disease of the olfactory nerve itself, or of its terminal apparatus, or of its central termination, are not infrequently observed, but they have no great practical interest. It is well known that only the upper two turbinated bones and the upper part of the septum of the nares, the olfactory region, are supplied by fibers of the olfactory nerve. The branches of the olfactory nerve pass into the cranial cavity through the openings of the lamina cribrosa and form the trunk of the nerve. Nothing certain is known as to their further central course. The hemianosmia, in affections of the posterior portion of the internal capsule, is worthy of notice, and also the anosmia of the left nasal cavity which has sometimes been claimed to be observed in cases where there was also right hemiplegia and aphasia.

In testing the sense of smell, we use substances which do not at the same time irritate the sensory fibers of the trigeminus in the nasal cavity. The best substances to use are cologne-water, ethereal oils, such as oil of cloves or oil of bergamot, oil of turpentine, camphor, musk, valerian, asafœtida, etc.

Hyperæsthesia of the sense of smell (hyperosmia) makes itself manifest either by a remarkably fine perception of odors, or by an abnormal sensitiveness to them. The latter symptom is often noticed, especially in the hysterical. Patients have headaches, or attacks of fainting, from slight odors, which healthy persons notice but little. Subjective sensations of smell (hallucinations of smell) are quite frequent among the insane, and sometimes during the aura of an epileptic attack.

A diminution of the power of smell (olfactory anæsthesia, anosmia) is also not infrequent. We see it in the different diseases of the nose—coryza, etc.; also in affections of the base of the skull, such as tumors, and acute and chronic meningitis, which involve the trunk of the olfactory sympathetically, and also in cerebral disease, tumors, etc., and most frequently in severe hysteria. In far-advanced tabes dorsalis we have sometimes found pronounced anosmia, dependent, perhaps, upon an atrophy of the olfactory nerve. It is important to state that in every marked enfeeblement of the smell the "taste" for many forms of food suffers, since it is well known that their "aroma," as in roast meats, wines, and the different sorts of cheese, is due chiefly to the co-existing sensations of smell.

The treatment of anomalies of smell almost always coincides with the treatment of the primary disease. In case the disturbance of smell makes special interference desirable, we can try electrization of the nasal mucous membrane, or painting it with a one-per-cent. solution of nitrate of strychnine in olive-oil.

CHAPTER VIII.

ANOMALIES OF THE SENSE OF TASTE.

Sensations of taste are obtained from two nerves—the glosso-pharyngeal, and the lingual nerve from the third branch of the trigeminus. The glosso-pharyngeal is the nerve of taste for the posterior third of the tongue and the palate, the lingual for the anterior two thirds of the tongue. The gustatory fibers of the lingual, all, or at least a great part of them, pass over to the chorda tympani, and reach with this the trunk of the facial; they do not remain in the facial, however, as many pathological experiences have most plainly shown, but they finally come back to the trigeminus, and, probably chiefly by the great superficial petrosal nerve and the Vidian nerve to the spheno-palatine ganglia, in this way they pass to the second branch of the trigeminus. There may be some other channels, however, by which the gustatory fibers finally unite again with the trigeminus, and enter the brain with its trunk. We know nothing definite as to their further course and their central termination.

Hyperæsthesia of taste is rare, and it has been seen almost exclusively in the hysterical. Paræsthesia of taste is sometimes found in patients with facial paralysis, who complain of an abnormal taste in their mouths. In the insane, too, subjective sensations of taste (hallucinations of taste) may occur. Anæsthesia of the gustatory nerves (gustatory anæsthesia, ageusia) is, however, quite frequent. As follows from what has gone before, this may be seen: (1) In affections of the peripheral terminal organs of the gustatory nerves, as in disease of the mucous membrane of the tongue; (2) in affections of the glosso-pharyngeal, such as compression; (3) in affections of the lingual nerve, and of the trigeminus within the cranial cavity; (4) in affections of the chorda tympani, from diseases of the middle ear; (5) in affections of the facial nerve between the entrance of the chorda tympani and the geniculate ganglion; but we know from experience that any obstacle to conduction in this nerve, above or below the points named, causes no disturbance of the sense of taste. Central disturbances of taste have been observed in affections of the posterior portion of the internal capsule. Disturbances of taste are very common in hysteria and allied conditions (traumatic neuroses, etc.).

The test of the sense of taste must be performed separately for all the different varieties of the sensation of taste, since partial paralyses of taste are not infrequent. The test is performed by putting small amounts of the substance to be tasted in solution on the tongue with a glass rod or a brush. The anterior and posterior parts are to be tested separately. A solution of quinine or tincture of nux vomica serves as a test for bitter, a solution of sugar for sweet, vinegar or dilute muriatic acid for sour, and a solution of common salt for salt. We may also use as a test for taste the well-known galvanic taste, which is strongest at the anode, but is also detected at the cathode in even very weak currents, and hence is so often noticed from by-currents in galvanizing the head, neck, etc.

An accurate diagnosis as to the seat and cause of disturbances of taste can be made only by considering the other symptoms which are also present. Direct treatment may best be employed by the aid of electricity

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SECTION II.

DISEASES OF THE MOTOR NERVES.

CHAPTER I.

GENERAL REMARKS UPON THE DISTURBANCES OF MOTILITY.

1. PARALYSIS.

General Classification of Paralyses.—By "paralysis" we mean the loss of voluntary motion in the muscles of the body controlled by the will. We usually distinguish between the complete loss of the power of active motion (paralysis) and the mere weakening of it (paresis). In complete paralysis of any part of the body, or of a single muscle, the slightest voluntary motion can not be produced in it; while in paresis in a diseased part certain movements are still possible, but they are more or less below the normal in strength, extent, and duration.

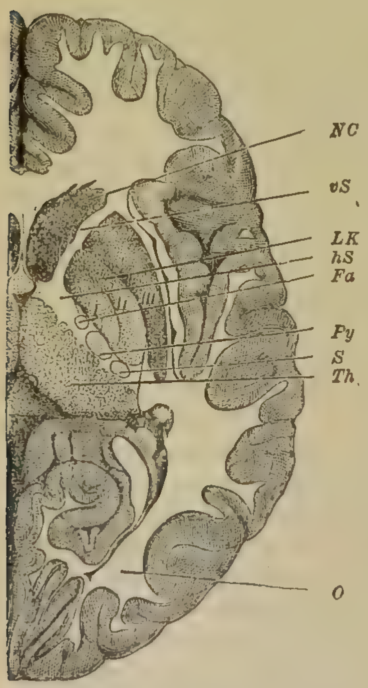

FIG. 64.—Horizontal section through the right cerebral hemisphere. *NC.* Caudate nucleus. *Th.* Optic thalamus. *LK.* Lenticular nucleus (first, second, and third divisions). *vS.* Anterior limb of the internal capsule. *hS.* Posterior limb of the internal capsule. *Fa.* Fibers belonging to the facial nerve. *Py.* Pyramidal tract (motor). *S.* Sensory tract (probably cutaneous nerves and those of special sense). *O.* Occipital lobes.

In every portion of the tract that leads from the motor portions of the cortical gray matter of the brain to the muscles—that is, in every part of the so-called great "cortico-muscular conduction-path" or "pyramidal tract"—disease may lead to paralysis if it takes away from the part affected its power to conduct voluntary motor irritations. Every destruction or inhibition of function of the motor centers in the cerebral cortex, with whose integrity the initiation of voluntary innervation is associated, must also lead to a paralysis in the corresponding muscular region. Finally, it is conceivable, at least *a priori*, that diseases of the muscles may also lead to a paralysis, since the muscles may either have their contractile substance injured, or lose their power to respond by a contraction to any nervous irritation that reaches them; but an absolute confirmation of such "myopathic paralyses" is associated with great difficulties, because diseases of the special muscular substance can scarcely be separated from diseases of the terminal branches and terminal apparatus of the motor nerves.

If we represent to ourselves briefly the precise course of the chief tract for exciting voluntary movements, as far as it is now known, we must put the beginning of this tract, according to all recent experiments, in the region of the central convolutions of the cerebrum and of the paracentral lobule. Here we find the so-called psycho-motor centers (see the details in the chapter on cerebral localization), from which the motor fibers in the corona radiata converge and pass downward. The latter, after they have united into quite a compact bundle, enter the internal capsule, which they traverse obliquely. As we see in a horizontal section through the cerebral hemispheres (see Fig. 64), the internal capsule consists of two limbs —an anterior, between the lenticular and the caudate nuclei, and a posterior, between the lenticular nucleus and the optic thalamus. The two limbs form an obtuse angle, opening outward, whose top—that is, the junction of the anterior and posterior limbs of the internal capsule—is termed the "knee of the capsule." The motor tract (*Py*) lies in the posterior limb of the internal capsule about the posterior end of its middle third. In this it runs downward rather obliquely, so that in the upper part of the internal capsule it lies somewhat farther forward than in the lower. From the internal capsule the pyramidal tract enters the crusta. It lies first in the third quarter, counting from the inside, then farther down in the middle third of the crusta (see Fig. 65), and from this point it passes into the anterior half of the pons. In the pons the fibers of the pyramidal tract are somewhat separated, but they come together below it into the compact bundle of the pyramids on the anterior surface of the medulla. At the lower end of the pyramids the decussation of the (lower) motor pyramids takes place—that is, the motor fibers of each pyramid pass over, for the most part, into the lateral column of the opposite half of the spinal cord, and here form the distinct bundle of the lateral pyramidal tract (*PyS*, see Figs. 66 and 67). A small part of the pyramids, which sometimes seems to be entirely wanting, remains uncrossed, and passes downward in the anterior column of the cord on the same side as the so-called anterior pyramidal tract (*PyV*, Fig. 66). From the lateral column, or the anterior column of the cord, the motor fibers pass into the anterior gray columns of the cord, and are here directly connected with the large motor ganglion-cells of the anterior cornua. The anterior root-

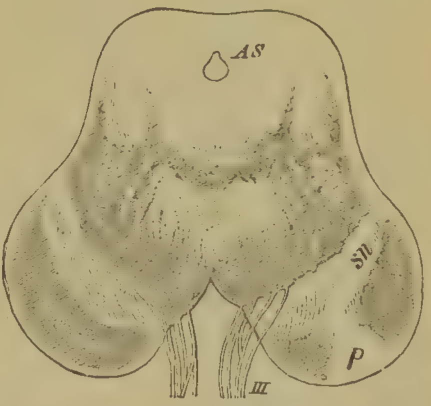

FIG. 65.—Transverse section through the crura cerebri in secondary degeneration of the right pyramidal tract. (From CHARCOT.) *sn.* Substantia nigra. *p.* The degenerated and therefore translucent pyramidal tract. *III.* Oculo-motor nerves. *AS.* Aqueduct of Sylvius.

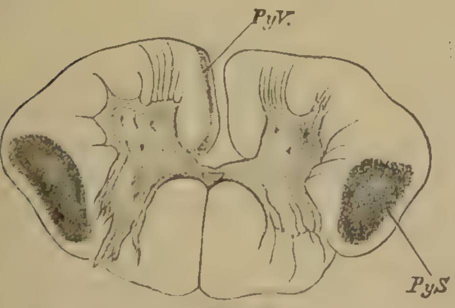

FIG. 66.

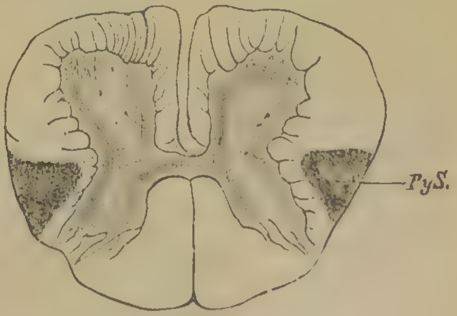

FIG. 67.

FIG. 66.—Transverse section through the cervical enlargement of the spinal cord. *PyS.* Lateral pyramidal tract. *PyV.* Anterior pyramidal tract (in this case present only on one side).
FIG. 67.—Transverse section through the lumbar enlargement. *PyS.* Lateral pyramidal tract. (The anterior tract is no longer present in the lumbar portion of the cord.)

fibers pass out from these ganglion-cells, as is well known, and become the anterior spinal roots of the peripheral nerves. Through the latter the motor impulses, coming from the cerebrum, finally reach the special motor apparatus, the voluntary muscles.

The long motor tract, just described, the cortico-muscular path or the pyramidal tract, has been quite accurately determined in its details by the results of pathological observations (Türck, Charcot), and investigations into the history of its development (Flechsig). It forms, at all events, the chief path for the conduction of voluntary innervation. It is possible that there are other motor paths of conduction besides this tract, but we know nothing definite concerning them.

If we pay attention to the course of the motor tracts described, we shall easily understand certain peculiarities in the distribution of motor paralyses, which are of great importance in diagnosis. Since the motor centers for separate parts of the body, such as the face, the arm, or the leg, are separated from one another in the cerebral cortex, and are distributed over a comparatively large surface, as we shall see more fully later on, it is easily explained why affections of the cortex, if they are not very extensive, may lead to paralysis of only a single part of the body. We call such an isolated paralysis of one part of the body monoplegia, and thus we speak of a cortical facial or brachial monoplegia. Farther downward in the brain, in the internal capsule and the crus cerebri, however, as we have seen, all the motor fibers are collected into one bundle, whose diameter takes up comparatively little space. Hence we comprehend that any disease of the brain, which is situated in this part of the motor tract, may easily make this tract incapable of conduction throughout its whole extent, or at least throughout the greatest part of it. The result must be, then, a more or less complete paralysis of the facial muscles, the arm, and the leg at the same time—that is, of the entire half of the body—a form of paralysis which is termed hemiplegia or unilateral paralysis. We may note here that, as a result of the passage of the motor fibers to the other half of the cord in the decussation of the pyramids, the paralysis must develop on the side of the body opposite to the focus of disease in the brain. Farther down in the medulla and the cord the fibers coming from both hemispheres, and belonging to the two sides of the body, lie quite near each other. Since many diseases of the cord have a tendency to affect the two halves of the cord at once, or gradually to extend over the whole transverse section of the cord, a simultaneous paralysis of the corresponding portion of the two sides of the body will readily arise as a result of this. This form of paralysis we call paraplegia. Diseases in the cervical cord may have as a result a paralysis of all four extremities or of the two arms—cervical, brachial, or superior paraplegia; diseases in the dorsal and lumbar cord a paraplegia of the two legs—inferior paraplegia, often called simply "paraplegia," without further prefix. In affections of the peripheral nerves we have, of course, again a limitation of the paralysis to the region of the affected nerve. The paralysis may be quite extensive in diseases of a nervous plexus—paralysis of a peripheral plexus; or it may confine itself wholly to the region of a single nerve, or even of a single branch of the nerve—paralysis of a peripheral nerve.

We will have to add many more details to what has just been said, but as a fundamental principle we may now note that hemiplegia is the chief form of cerebral paralysis, while paraplegia is the chief form of spinal paralysis. Monoplegias are usually either cortical cerebral paralyses, or peripheral paralyses.

General Ætiology of Paralysis.—The kind of lesion which leads to paralysis may, in the different cases, be very diverse. From easily understood reasons we can very rarely decide as to the kind of lesion from the intensity and extent of the paralysis, but only from the manifest ætiological factors, from the development and course of the paralysis, from other morbid symptoms that are also present, etc. In general, we may divide the paralyses into two groups, according to the nature of their cause: into paralyses from causes that can be discovered anatomically, and into the so-called functional paralyses, in which no anatomical cause for the paralysis can be found; but since our anatomical, and especially our histological, methods of investigation have become better developed and are more employed, the domain of functional paralyses has become gradually more and more restricted, and a definite anatomical cause has now been found for many paralyses which were once regarded as functional.

All the diseases of the nervous system may be anatomical causes of paralysis, if they lie in a spot where they damage or destroy the paths of motor conduction. Inflammation, degeneration, new growths, hæmorrhages, and severe disturbances of circulation, with their results, especially embolic and thrombotic softening, are found in the brain, the cord, and the peripheral nerves, and under some circumstances give rise to the appearance of paralysis. Mechanical lesions of the nervous system also play a great part in the pathogenesis of paralysis, especially traumatic injuries, and compression of the brain, the cord, and the peripheral nerves by swellings, new growths, and other diseases in their vicinity.

We also know certain toxic substances which produce paralysis from their persistent action on the organism. Of these toxic paralyses, lead paralysis is the most important in its clinical relations; but other poisonous substances, such as copper, arsenic, and certain vegetable alkaloids, may cause paralysis. In regard to lead paralysis (*vide infra*), which was once considered a purely functional paralysis, we now know that anatomical changes form its basis. These can be plainly made out, especially in the peripheral nerves.

We may group a large number of paralyses together under the term of "paralysis after acute diseases." Since in these cases we always have to do with acute infectious diseases, we may assume, as their most probable cause, certain changes in the nervous system, sometimes in the brain, but more frequently in the cord or the peripheral nerves, which stand in direct relation to the specific infectious material. There seem to be chemical poisons ("ptomaïnes") which form in the body in infectious diseases, and cause degeneration of certain nerve fibers, in the same way, for example, as lead. We most frequently see paralysis appear after diphtheria, diphtheritic paralysis (*vide infra*), or more rarely after typhoid, small-pox, dysentery, the acute exanthemata, etc. The paralyses which may arise in certain chronic infectious diseases, especially in syphilis and tuberculosis, usually have another origin. Here we usually have to do with the specific morbid product itself (gumma, tubercle), which may invade various parts of the nervous system.

Those paralyses which come on most manifestly because of exposure to cold are termed paralyses from exposure to cold, or "refrigeratory," or frequently "rheumatic" paralyses. Although many spinal diseases, like myelitis, may perhaps be referred to exposure to wet or cold, we usually reckon among rheumatic paralyses only certain peripheral paralyses, like that in the region of the facial nerve. The functional disturbance of the nerves in these cases probably depends upon mild inflammatory changes in them produced by cold, and is accordingly of an anatomical and not merely of a functional nature.

There are, however, quite a comprehensive group of paralyses which we must to-day still term functional paralyses. To this class belong the hysterical paralyses, the paralyses from psychical causes, like paralysis from fright, the "paralyses from imagination," etc. We shall learn to recognize these more fully in the chapter on hysteria.

In conclusion, we must bear in mind the "reflex paralyses," whose ætiology is not yet fully explained—that is, paralyses which come on in the course of diseases of certain internal organs, especially of the intestine, and of the urinary and sexual organs. An attempt has been made to explain their origin, from analogy with well-known physiological experiments, by the idea that a "reflex inhibition" is excited in certain motor regions by a sensory irritation arising in the diseased organs, a theory which has not yet been fully confirmed. Leyden's hypothesis is somewhat more probable, but it is by no means proved beyond a doubt. According to it the paralyses of this class are explained by an ascending neuritis, arising from the organs originally affected (see the chapter on neuritis). Generally speaking, the whole conception of "reflex paralyses" is still far from clear, and we will do well to be extremely guarded in their diagnosis. Lépine has also regarded the paralysis of the arm of the affected side, seen in some cases of empyema, especially after operative interference, as a "reflex paralysis," an explanation which may be proper in some cases, but in regard to which we should be the more cautious, as metastatic abscesses of the brain are not very infrequently found in empyema (see the chapters on purulent meningitis and cerebral abscess).

General Symptomatology of Paralyses.—We can recognize the existence of a paralysis, except from the patient's statements as to the impossibility of performing certain motions and functions, only by a careful and thorough physical examination of the power of voluntary motion. This examination in patients with nervous disease must extend to all parts of the body, and demands an accurate knowledge of all the movements that can normally be executed by the different joints, and of the muscles and nerves requisite to produce them. In the description of the different single forms of paralysis we will go more into detail in regard to the anomalies of motion to be observed.

In each individual case of paralysis some other symptoms must be considered besides immobility—first the condition of the paralyzed muscles, and then certain accompanying symptoms that are often present with the paralysis.

In regard to the first point, the trophic condition of the paralyzed muscle is of the greatest diagnostic and practical importance. In comparing a large number of paralyses, a very evident difference in this respect strikes us at once. We see on the one hand paralyses where the paralyzed muscles retain their normal volume and their normal state of nutrition entirely, or almost entirely, for years, and on the other hand we see cases in which there is a considerable atrophy in the paralyzed muscles in a few weeks or months. This difference is so effectual that all the last-named paralyses have been classed together under the name of "atrophic paralyses." Since muscular atrophy does not occur in every case of paralysis, it can not be simply the result of the rest and inactivity of the muscles, but it must have its special cause.

If we once more represent to ourselves the whole course of the motor tracts, from the cerebral cortex to the voluntary muscles, we shall remember that the nerve-fibers through this long route undergo a single interruption—namely, by the interposition of the large ganglion-cells of the anterior cornua of the gray matter of the spinal cord. Clinical and anatomical experience teaches us that, in all those paralyses where the cause, that is, the break in conduction of the motor fibers, lies in the first portion, that is, between the cortex and the cells in the anterior cornua, there is, as a rule, no atrophy, or only a slight amount of atrophy, in the paralyzed muscles, while in those paralyses where the cause is situated in these ganglion-cells themselves, or in the portion of the motor tract peripheral to them, a pronounced muscular atrophy rapidly develops. The only possible interpretation of this fact is, that the large motor ganglion-cells of the anterior cornua have, as we express it, a trophic influence on the muscles. If these cells are intact, and the conduction from them to the muscles is not interrupted, the muscles keep approximately their normal condition of nutrition, even if they are paralyzed, while an affection of the ganglion-cells themselves, or an interruption of conduction in the peripheral nerves, rendering the transmission of the trophic influences from the cells to the muscle impossible, necessarily results in an atrophy of the muscles. This atrophy is not confined merely to the muscles separated from their "trophic center," that is, from the ganglion-cells in the anterior cornua of the cord, as we must note here, but the nerves proceeding downward from the point of the lesion also take part in the atrophy. Since this atrophy, both of nerve and muscle, is associated with a destruction of tissue to be more fully described later—a genuine "degeneration" of the fibers—we speak of a "degenerative atrophy" of the nerves and muscles, in contrast to the simple muscular atrophy which occurs in almost all severe diseases, in starvation, etc. The degeneration of the nerves is, of course, not evident to our sight and touch during life, but it is proved, as we shall soon see, by certain changes in their electrical excitability.

From the above statements the following extremely important points in the anatomical diagnosis of paralysis are at once evident: that in cerebral paralyses there is never a degenerative atrophy in the paralyzed muscles; that atrophy is present in spinal paralyses only when the large ganglion-cells in the anterior cornua of the cord, belonging to the muscles, are destroyed or injured in their functions by the cause of the paralysis; but that in all long-continued peripheral paralyses a degenerative atrophy of the paralyzed muscles and nerves must inevitably develop. These fundamental principles may suffice for the present; further deductions from them must be postponed until the special chapters on the various forms of paralysis.

We observe a further distinction in the condition of the paralyzed muscles if we perform passive motion in the paralyzed parts. There are paralyses where we can perform passive motion in the paralyzed parts at any joint with perfect ease and freedom, and without perceiving the slightest resistance. We term such paralyses "flaccid paralyses," but there are also paralyses where passive motion meets with considerable muscular resistance, so that it can be performed only with a certain greater or less amount of exertion, or only within certain limits, or not at all. This difficulty in performing passive motion may have different causes. It is most frequently caused by the development of persistent shortening in the paralyzed muscles themselves, or in their antagonists (the so-called contractures), which prevent the free performance of passive motion. In other cases there is no special contracture, but the paralyzed muscles exhibit a peculiar rigidity. There are all sorts of muscular contractions, which either are to be regarded as symptoms of direct motor irritation (*vide infra*), or have a reflex origin. Paralyses in which the performance of passive motion is hindered by such muscular contractions are termed "spastic paralyses." The details of all these symptoms will be spoken of in the special chapters.

Finally, in every case of paralysis, we must consider the other nervous symptoms which accompany it, since these may also be of great importance in judging of the cause of the paralysis. We must first of all investigate the condition of the reflexes (*vide infra*) in the paralyzed parts, from which many conclusions can be drawn as to the seat of the lesion which causes the paralysis. We must also test the state of the sensibility, both in the skin and in the muscles themselves. Certain attendant trophic and vaso-motor symptoms are also to be regarded. The skin over the paralyzed parts sometimes appears cyanotic, or marble-like; it feels cool, is œdematous, and sometimes is peculiarly dry, hard, and scaly.

2. Symptoms of Motor Irritation.

As we have termed the symptoms of motor deficiency "paralysis," we group the symptoms of motor irritation in general together under the name of "spasm." We mean by this all the morbid movements occurring in the muscles involuntarily and even against the will. Although we may find spasm in the smooth muscles, which generally are not controlled by the will, as in spasm of the bronchial muscles, spasm of the vessels, etc., we will concern ourselves here only with the spasmodic movements in the voluntary muscles. We must look for the cause of these in abnormal irritation exerted in some way on the motor tracts, but we know very little of the precise nature and character of this irritation in most cases. The abnormal irritation often acts directly on the motor nervous region, as in the frequent spasms in affections in the neighborhood of the cortical motor centers; but the motor irritations often seem to be excited secondarily through some reflex channel—reflex spasms.

For a long time two kinds of spasm have been distinguished symptomatically. We term those spasms clonic where the abnormal muscular contraction lasts only a short time, and then is interrupted by a short period of relaxation, to come on again afresh. The affected parts of the body are thus put in a constant convulsive motion. In distinction from this we term those abnormal muscular contractions tonic spasms where the muscle remains spasmodically contracted for a longer time—minutes, hours, or days. The affected part of the body is thus kept motionless in some abnormal position. Both forms of spasm, however, show many transitions and combinations, so that we must often speak of "tonic-clonic" spasms.

A more careful examination of the symptoms of motor irritation, however, gives a still greater number of different forms. We will here group together the most important varieties of morbid involuntary movements without giving a completely exhaustive review of the manifold forms of spasm.

1. *Epileptiform convulsions* are severe, and usually clonic spasms, at times tonic-clonic, spread over the whole body or limited to one half or one portion of the body. By them the whole body or the part affected is put into violent motion, usually thrashing and shaking movements. The pure epileptic spasm in epilepsy is the type of this form of spasm, but precisely analogous spasms, "epileptiform" spasms, are seen in organic diseases of the brain, in hysteria, etc.

2. *Rhythmical contractions* in single groups of muscles are sometimes seen in certain cerebral diseases, such as apoplexy and sclerosis, and also, as we have lately observed, after acute myelitis. In these the part of the body affected is put in motion by continuous, separate, weaker or stronger thrusts, which follow one another in a regular rhythm. Rhythmical contractions are also seen as precursors or at the end of epileptiform spasms.

3. *Trembling motions*, or *tremor*, as we say in ordinary parlance, are moderate motions, rapidly following one another, with a not very marked excursion. If the tremor is more pronounced, we term it "shaking." Tremor is an important symptom, almost pathognomonic in many nervous diseases, such as paralysis agitans, but we know almost nothing in regard to the more intimate manner of its origin. We often see pronounced tremor, especially in the hands in exophthalmic goitre. We know that tremor is often present in old people—senile tremor; and in alcoholic subjects—alcoholic tremor. Tremor sometimes appears in muscles at rest, that is, not innervated by the will, and sometimes only in those which are moved voluntarily. This latter form of tremor, which is seen most frequently in multiple sclerosis (*vide infra*), is termed "intention tremor." Very marked intention tremor, which is increased by any mental excitement, is seen as a symptom in chronic mercurial poisoning (mercurial tremor), particularly in mirror-makers, etc.

We may mention here the so-called essential tremor, that is, a condition where the tremor, which is most marked in the hands, is the only morbid symptom and can be referred to no known cause. This form of tremor is sometimes seen in comparatively young people, and even in children. A distinct hereditary predisposition is often present, so that several "tremblers" are found in the same family.

We very often find trembling in "nervous" persons, which is at once increased by any emotion. There is also a pure hysterical tremor.

4. *Single contractions*, either sudden twitchings, or in the form of a slow contraction of the muscle, are seen with especial frequency in diseases of the cord. They are either single, or frequent and persistent. Their mode of origin is not always plain. They may depend on direct motor irritation or they may have a reflex origin.

5. *Fibrillary muscular contractions* are little contractions in the separate muscular bundles, which may be seen on a close examination of the muscles, but which do not have any special influence on motion. If the fibrillary contractions in a muscle are very pronounced, there may develop an actual "wave" in the muscular substance. We see this symptom especially in atrophied muscles, particularly in spinal progressive muscular atrophy (*vide infra*).

6. *Choreic movements* are either slight contractions or quite complicated and extensive movements, which usually appear in the face, in one limb, or sometimes over the whole body, without regard to rule. In severe cases they are almost continuous, but in milder cases they are interrupted by shorter or longer pauses. They form the chief symptom of chorea proper, but they are also present in other cerebral affections, such as symptomatic chorea, post-hemiplegic chorea, etc.

7. *Movements of athetosis* is the name we give to peculiar involuntary and usually quite slow movements, which are seen especially in the arm and hands, but also in the head, the trunk, etc. The fingers make slow but often very extensive movements, are extended, spread apart, flexed, and moved over and around one another in the most remarkable way. This form of motor irritation occurs as a special disease, "*athetosis*," or as a symptom in certain central nervous diseases, especially the cerebral paralysis of children (*vide infra*).

8. *Constant* or *co-ordinated spasms* are symptoms of motor irritation in which the patient performs complicated movements by compulsion—forced movements. Among these are classed the compulsory going forward or moving in a circle, the turning about the axis of the body (forced attitudes), and also certain peculiar complicated forms of spasm, such as spasms of jumping, laughing, screaming, with all sorts of spasms of the respiratory, pharyngeal, and laryngeal muscles, associated with hiccoughing and belching sounds, etc. They are seen most frequently in severe cases of hysteria, but epilepsy may also occur exceptionally in the form of co-ordinated spasms. The forced movements and attitudes mentioned above are seen chiefly in affections of the cerebellum and the cerebellar peduncles.

9. *Tonic spasm*, as has been said, is the name for all morbid muscular contractions that continue for a long time. Tonic spasm in the muscles of mastication, the masseters, is termed trismus. Tonic spasm in the muscles of the back and neck, by which the whole body is drawn backward, and the vertebral column is bent into an arch with the convexity in front, is called opisthotonos. Tonic rigidity of the whole body is termed tetanus. Tonic spasms are seen in tetanus, tetany, hysteria, etc.

10. *Cataleptic rigidity* is the name of that tonic condition of the muscles in which the limbs are deprived of the influence of the will, but are held in position by the muscles in any position given to them passively. It is seen chiefly in certain cases of hysteria, but cataleptic states are also present in other cerebral diseases, such as meningitis (see the chapter on catalepsy).

11. *Associated movements* are abnormal movements which appear, while mak-

ing voluntary movements, in muscles which have no connection with the movement willed. Thus in hemiplegia associated movements sometimes take place in the arm when the patient wills to move the leg alone. In spinal cases, as we have seen, the movement of one leg is accompanied also by an unintentional movement of the other leg. Associated movements are commonest in the muscles of the same limb. Thus we often see in hemiplegia or in spastic spinal paralysis that patients can not draw the leg up on the body without at the same time producing a marked dorsal extension of the foot as an associated movement. In old peripheral facial paralysis (*q. v.*) we very often see associated movements in the facial muscles.

Besides the conditions of motor irritation, other attendant nervous symptoms often occur at the same time. Symptoms of motor paralysis and irritation are very often combined with each other, since the different forms of spasm may appear not only in groups of muscles whose motion is otherwise normal, but also in paretic or paralyzed muscles. In general convulsions the state of the consciousness deserves special attention. Genuine epileptic attacks are usually associated with complete loss of consciousness, but in most of the other forms of spasm the consciousness is unaffected. Finally, it is worthy of mention that tonic spasms especially are attended by a feeling of decided pain, which is probably due to an irritation of the intra-muscular sensory nerves. Such painful tonic muscular contractions are termed cramps. Among them are the well-known painful spasms in the calves after physical exertion.

3. Ataxia.

In executing all normal complicated movements we need the simultaneous action of several muscles. Consider the numerous muscles which must be put in activity in walking, in grasping, and in all the manifold employments of the hands. Hence, in order to perform such movements correctly, it is not only necessary that all the muscles concerned should be innervated by the will—that is, that they be not paralyzed—but that we should also be able so to modify the innervation of each individual muscle that its contraction corresponds precisely to the special part of the work belonging to it. A prescribed voluntary motion can take place only when, first, all the muscles requisite for it come into no less but also no greater action; second, when each individual muscle contracts only so far and so much as its special task requires; and, third, when the conditions in the time of innervation take their normal course—that is, when all the muscles involved contract at the same time or in the proper order after one another. A movement which is executed in such a prescribed manner we call a co-ordinated movement, and the process of modifying properly the innervation of the different muscles necessary for a complicated movement we call the co-ordination of motion. We must especially bear in mind that the simultaneous action of several muscles is so far necessary, even for what seem to be the simplest movements, that muscles antagonistic to those moved must also come into activity. Only by the aid of the ever-ready antagonistic muscles can we grade our movements as finely, or check or hasten them as rapidly, as is demanded for the execution of almost all complicated movements.

Nervous pathology is rich in facts which can make the idea and the necessity of the co-ordination of motion clear to us. We often see disturbances of motility which make the patient incapable of any fine motor acts, and yet which do not depend at all upon any motor weakness or paralysis, but only upon a disturbance in the co-ordination of motion. Such a disturbance we call ataxia, and we speak of an ataxia of the arms, of the legs, etc., when the parts named can perform all the motions and retain their full strength, but these movements show, usually at once, a striking, disordered, uncertain "ataxic" character.

Many theories have been advanced as to the precise cause of ataxia, upon which we must enter in the special chapters. We can remark here only that ataxia is seen both in cerebral diseases, especially in affections of the cerebellum (cerebellar ataxia), and in diseases of the spinal cord (spinal ataxia). Among the latter, degeneration of the posterior columns especially (locomotor ataxia, tabes dorsalis) has ataxia as one of its chief symptoms. Therefore we will discuss the character of the symptoms and the cause of ataxia more fully in describing this disease.

4. GENERAL REMARKS UPON TESTING THE REFLEXES AND UPON THE CONDITION OF THEM.

In testing the reflexes, which should never be omitted in any case of nervous disease on account of its frequent great diagnostic importance, we must distinguish the two chief groups of reflexes from each other: the cutaneous reflexes, and the "tendon reflexes."

Cutaneous Reflexes.—We term the muscular contractions, excited reflexly by irritation of the sensory centripetal cutaneous nerves, cutaneous reflexes. These are usually present only to a slight degree in the upper extremities; but we can sometimes excite reflexes even here by pricking or pinching the skin, especially that of the fingers. The very marked reflex in many people caused by tickling the axilla is well known. The test of the cutaneous reflexes in the lower extremities is much more important. The soles of the feet are the most sensitive parts for exciting a reflex. Simply tickling the soles with the finger is a sufficient irritation (the tickling reflex), and so is the prick of a pin (the prick reflex), or striking the skin hard with a blunt object, usually the handle of a percussion-hammer (the blow reflex). Thermal irritants are also very suitable for exciting a reflex, especially bits of ice held to the skin (cold reflex). It is often advisable to try all these methods, since with diminished reflex irritability a reflex contraction in the leg can often be excited only by some one of them. We should also examine the reflex irritability of the rest of the skin, as well as the soles of the feet, by a pin-prick, by pinching a fold of the skin, etc. We should especially remember that in nervous diseases there is often a delay in the reflex, so that the reflex contraction appears only when the irritation has lasted for a certain time. Thus in many diseases of the cord, as we have repeatedly seen, the reflex follows only after we have pinched a fold of skin continuously for several (ten or fifteen) seconds, a delay which has a connection with the fact of the "summation of reflex irritation" known from physiology. The fact also deserves mention that in many patients the reflexes are easily excited in certain parts of the skin, but in other parts with difficulty or not at all—the "place of easiest reflex irritability."

In general, the reflex contractions are confined to the limb irritated. On pricking the sole of the foot, a dorsal extension of the toes or of the foot follows, or a greater or less flexion of the leg. The reflex rarely involves the rest of the body, but under pathological conditions there is such an increased reflex irritability that, on irritating the soles of the feet, both legs, or even the whole body, fall into contraction. Such a condition is sometimes seen in hysteria, in tetanus, in hydrophobia, in strychnine poisoning, etc.

We must also mention two special forms of cutaneous reflex which are often examined: the abdominal reflex, consisting of a contraction of the abdominal muscles on the same side, when we stroke the skin of the abdomen with the finger or the handle of the percussion-hammer; and the cremaster reflex—that is, the reflex retraction of the testicle, when we stroke the internal surface of the thigh, or exert a marked pressure a hand's breadth above the internal condyle. The cremaster reflex appears first on the side irritated, but not very infrequently on both sides at once. Other cutaneous reflexes, such as the gluteal reflex, the mammillary reflex, etc., have less significance and are often absent.

Judgment in regard to any pathological condition of the cutaneous reflexes is rendered difficult by the fact that their intensity varies considerably even under normal conditions. Many healthy people have much livelier reflexes than others. Hence we may judge most accurately of a patient if, in unilateral affections, we can compare the reflex symptoms in the two halves of the body with each other. The precise condition of the reflexes in the different forms of disease will be spoken of in the special chapters. We can state here only that a diminution or a complete absence of the cutaneous reflexes must, of course, be seen where the reflex conduction—through the centripetal nerve, the gray matter, the special anterior cornu, and the motor nerve—is interrupted at any point, as may be the case both in diseases of the peripheral nerves and of the spinal cord. On the other hand, however, the cutaneous reflexes may lose their intensity, or even disappear entirely, if the reflex centers lose their irritability from an irritation of the reflex-inhibitory centers or fibers. We see an abnormal increase of the cutaneous reflexes when either the irritability of the parts that aid in producing the reflex is increased, as in many cases of cutaneous hyperæsthesia, in strychnine poisoning, and in many general neuroses; or when the inhibitory processes which normally act upon the reflex centers are abolished, as in certain diseases of the brain and spinal cord. The increase of the cutaneous reflexes is shown partly by the fact that the reflex movements are particularly lively, and appear with a comparatively slight irritation of the skin, and partly by their extension to more distant groups of muscles than usual.

Tendon Reflexes.—Of almost greater practical importance than the investigation of the cutaneous reflexes is the test of the phenomena classed under the name of the "tendon reflexes," and first carefully investigated and described by Erb and Westphal in the year 1875. We understand by these those muscular contractions which arise from the mechanical irritation of the tendons and analogous parts, such as the periosteum and fasciæ. By this the sensory nerves of the tendon are irritated, and excite a reflex muscular contraction by means of the spinal cord. If we give a quick blow to the ligamentum patellæ (the tendon of the quadriceps extensor) with the ulnar side of the hand, or, better, with a percussion-hammer, while the leg hangs down laxly, or, if the person examined be in bed, while the leg is in a passive position of slight flexion, it is followed almost invariably in healthy persons by a more or less vigorous contraction of the quadriceps, by which the leg is extended. This is termed "patellar reflex," "knee phenomenon" (Westphal) [or "knee-jerk"]. In order to produce it, it is especially necessary for the person examined to avoid all active muscular tension in the leg, especially in the extensor. If we examine patients who are not in bed, we can test the patellar reflex by making the patient cross the leg to be tested over the other, and by striking the patellar tendon as the leg hangs loose; but it seems to us more convenient to direct the patient to extend the leg until it forms an obtuse angle with the thigh with the opening of the angle below. If the sole be set fully on the floor the extensor cruris is made tense in this position, and we can very easily and plainly excite the contraction of the quadriceps by striking the patellar tendon. If the patellar reflex be weak and hard to detect, we should try the proceeding advised by Jendrássik, which consists in testing the reflex while the patient grips the two hands together, and tries with all his strength to pull them apart. By this ingenious tension of the muscles of the upper extremities the muscles of the legs are probably greatly relaxed, while any voluntary innervation is avoided, and thus is explained the increase of the reflex, which is often pronounced. If by the help of this "Jendrássik's test" no reflex is to be elicited, we may regard it as absent.

The second important tendon reflex to be provoked in the lower extremity is the Achilles' tendon reflex. If we give to the foot of the person examined a passive position of slight dorsal extension, so that the tendo Achillis is a little tense, and then strike the tendon a quick blow, a marked contraction of the gastrocnemius follows. Under normal conditions this reflex is often absent. Where the tendon reflexes are abnormally increased, however, it is very vigorous, and then we can very often produce it in the following especially characteristic manner. If we make a sudden, short, vigorous, passive, dorsal extension of the foot, the tendo Achillis is suddenly made tense, and thus is irritated mechanically. As a result of this, there is a reflex plantar flexion of the foot. If now, by persistent passive dorsal extension of the foot, the tendo Achillis is again made tense, there follow by turns new plantar and dorsal movements of the foot, so that the foot is thus put into a vigorous tremor. This symptom, which can only exceptionally be provoked in healthy persons, is termed ankle clonus (foot clonus), or "foot phenomenon" (Westphal). Where there is a very great increase of the tendon reflexes the tremor is sometimes not confined to the foot, but the whole leg falls into a vigorous clonus, a symptom which was once given the unsuitable name of spinal epilepsy. We can also obtain the patellar reflex in the form of a clonus if we pull the patella firmly down with the finger and force it downward by a sudden blow on the finger.

The two symptoms described—the patellar reflex, and the Achilles' tendon reflex or foot phenomenon—are practically the most important, and are the most often tested, but they are by no means the only reflexes in the lower extremity. Besides the reflexes from the special tendons, we also frequently obtain muscular contractions by striking the periosteum and the fasciæ, which have been termed the periosteal and fascia reflexes. Thus a contraction in the quadriceps often follows a blow on the anterior surface of the tibia. We also see contractions frequently in the adductors of the thigh on striking the internal condyle of the femur, contractions in the muscles of the posterior aspect of the thigh in striking the calves, etc.

Under normal conditions the tendon reflexes in the upper extremities are often insignificant or entirely absent, but where the irritability is abnormally increased we see even here the most various and vigorous reflexes. The most important and most constant are the periosteal reflexes in the supinator longus, biceps, and deltoid, from a blow on the lower end of the radius and ulnar, and also the tendon reflex in the biceps from a blow on the biceps tendon at the elbow-joint, or in the triceps from a blow on the triceps tendon above the olecranon. A persistent clonus in the hand upon passive volar flexion sometimes occurs, but it is rare.

In many places in the special chapters we shall go more fully into the exact conditions and diagnostic significance of the tendon reflexes. We shall see that the absence of tendon reflex is characteristic of certain spinal diseases, such as poliomyelitis and tabes dorsalis, and also of most peripheral paralyses (traumatic paralyses and neuritis). We see an abnormal increase of tendon reflex in many diseases of the cord, especially in that form of spinal paralysis which is termed spastic spinal paralysis, and very often, too, in cerebral paralyses. The increase of the reflex in these cases is probably always due to a disappearance of certain influences which normally inhibit the reflex.

Although we have so far tacitly assumed the reflex nature of the symptom termed "tendon reflex" as certain—a theory first propounded by Erb, and now shared by most nervous pathologists on the ground of many clinical and experimental facts—we can not conceal the fact that the reflex nature of the phenomena in question is not recognized by another party, headed by Westphal. Westphal holds the "tendon phenomena" to be the result of a direct mechanical irritation of the muscle provoked by the jarring or stretching of the muscle. Since, however, careful experimental investigations, repeatedly performed, have of late almost without exception decided in favor of the reflex nature of the phenomena in question, and since many clinical facts, such as the occurrence of contractions in distant muscles, crossed contractions, etc., can be explained only in this way, we will hold in the sequel to the term "tendon reflex."

Mechanical Muscular Irritability and Paradoxical Contraction.—In addition to the description of tendon reflexes we must make brief mention here of two symptoms which must also be considered in the examination of nervous patients. "Direct mechanical irritability of the muscles" is shown by the occurrence of contractions from a direct blow on the belly of the muscle, in which, of course, we can not definitely separate the direct muscular irritation from some mechanical irritation of the muscular nerves. Sometimes the muscular contraction is perhaps also a reflex, arising from the mechanical irritation of the fascia drawn over the muscle; but it is worthy of mention that in cases where the tendon reflex has wholly disappeared, as in tabes dorsalis, the direct mechanical muscular irritability is usually retained. The so-called idio-muscular contractions must be especially distinguished. We see these most plainly if we give a vigorous blow with the ulnar side of the hand to the belly of a muscle, like the biceps. A circumscribed muscular swelling forms at the point struck, and gradually disappears again. The test for mechanical muscular irritability has not yet attained any special practical importance.

"Paradoxical contraction" is the name which Westphal has given to a symptom seen especially in the tibialis anticus, and rarely also in the flexors of the leg and forearm. It is when the foot, after being put in passive dorsal extension, remains in this position even after the expiration of a considerable time (several minutes), and a marked prominence of the tendon of the tibialis anticus is usually visible. We can not at the present time give an explanation of this phenomenon, which so far has been observed in different spinal and cerebral diseases, multiple sclerosis, paralysis agitans, etc.

5. General Remarks upon the Changes of Electrical Excitability in the Motor Nerves and Muscles.*

Electricity, since the investigations of Duchenne, Remak, Benedikt, Moritz Meyer, von Ziemssen, Brenner, Erb, and others, has become not only one of the most prominent therapeutic aids in the treatment of nervous diseases, but it also plays an extremely important part in the examination of nervous patients, since the test of the electrical excitability of diseased nerves and muscles gives us a large amount of valuable information in regard to diagnosis and prognosis.

Every complete electrical examination must be made with both currents—the faradic or induction current (usually the secondary current), and the galvanic or constant current. One "indifferent" pole is usually put on the sternum or the back of the neck, and the other "testing" pole on the nerve or muscle to be tested. The excitement of the muscle from the nerve is called indirect; the excitement from placing the electrode on the muscle itself (where, of course, the excitement of the intra-muscular nerves can not be excluded) is called direct. Those points on the human body where the different nerves and muscles are most easily accessible to the electrical excitement are to be found in Figs. 68 to 73, taken from Erb's hand-book.

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* In regard to all the details of electrical diagnosis and electro-therapeutics we would refer to Erb's "Handbuch der Elektrotherapie." Leipsic, Vogel, 1882. [Translated by De Watteville. New York: Wm. Wood & Co., 1887.]

In faradic examination the rule is that we can provoke marked muscular contractions both from the nerves and from direct excitement of the muscles at the points generally accessible to excitement. We designate the strength of the current required by the distance between the two coils of the induction apparatus (coil distance, *Rollenabstand*) at which the first minimal contraction of the muscle occurs. On increasing the current, the minimal contraction gradually passes into a vigorous tetanic contraction of the muscles.

Galvanic examination is performed by the aid of a "current reverser," by which the testing pole can be made either the negative pole (the cathode or zinc pole) or the positive pole (the anode or copper or carbon pole). By this "polar method of investigation" (Brenner) the following law of contraction is obtained, which holds equally for the normal motor nerves and for the muscles.

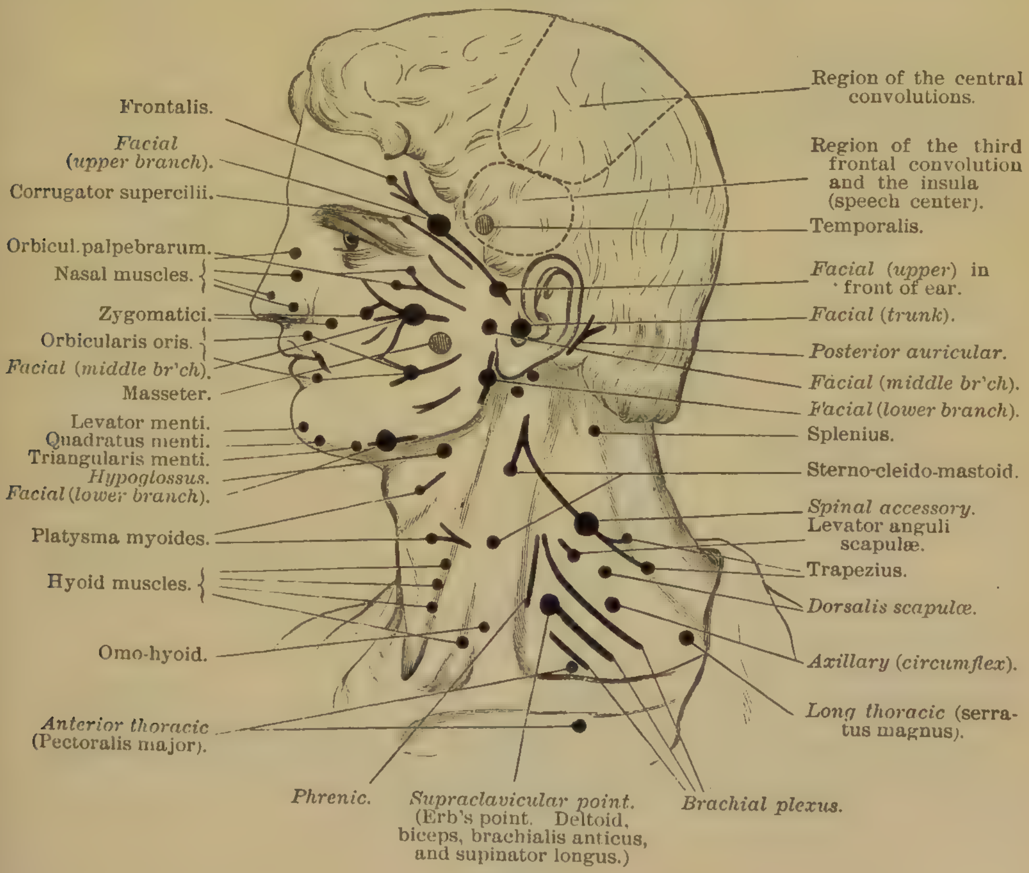

FIG. 68.

With a weak current no noticeable excitement takes place. If we gradually increase the strength of the current, the first weak contraction of the muscle occurs at the closure of the cathode—that is, when the current is closed so that the cathode is made the testing pole. On opening the cathode, or on closing or opening the anode, nothing follows. If we increase the strength of the current still more, the cathodic closure contractions become stronger, and the anodic closure and anodic opening contractions appear, now the one being earlier and stronger, and now the other. Opening of the cathode has still no effect. Only with a very strong current, in which the cathodic closure contractions have already become tetanic—that is, they still persist after the closure of the current—can we provoke weak cathodic opening contractions. Expressed in the abbreviations now in general use in electrical diagnosis, the law of contraction for normal muscles and nerves in man is as follows:*

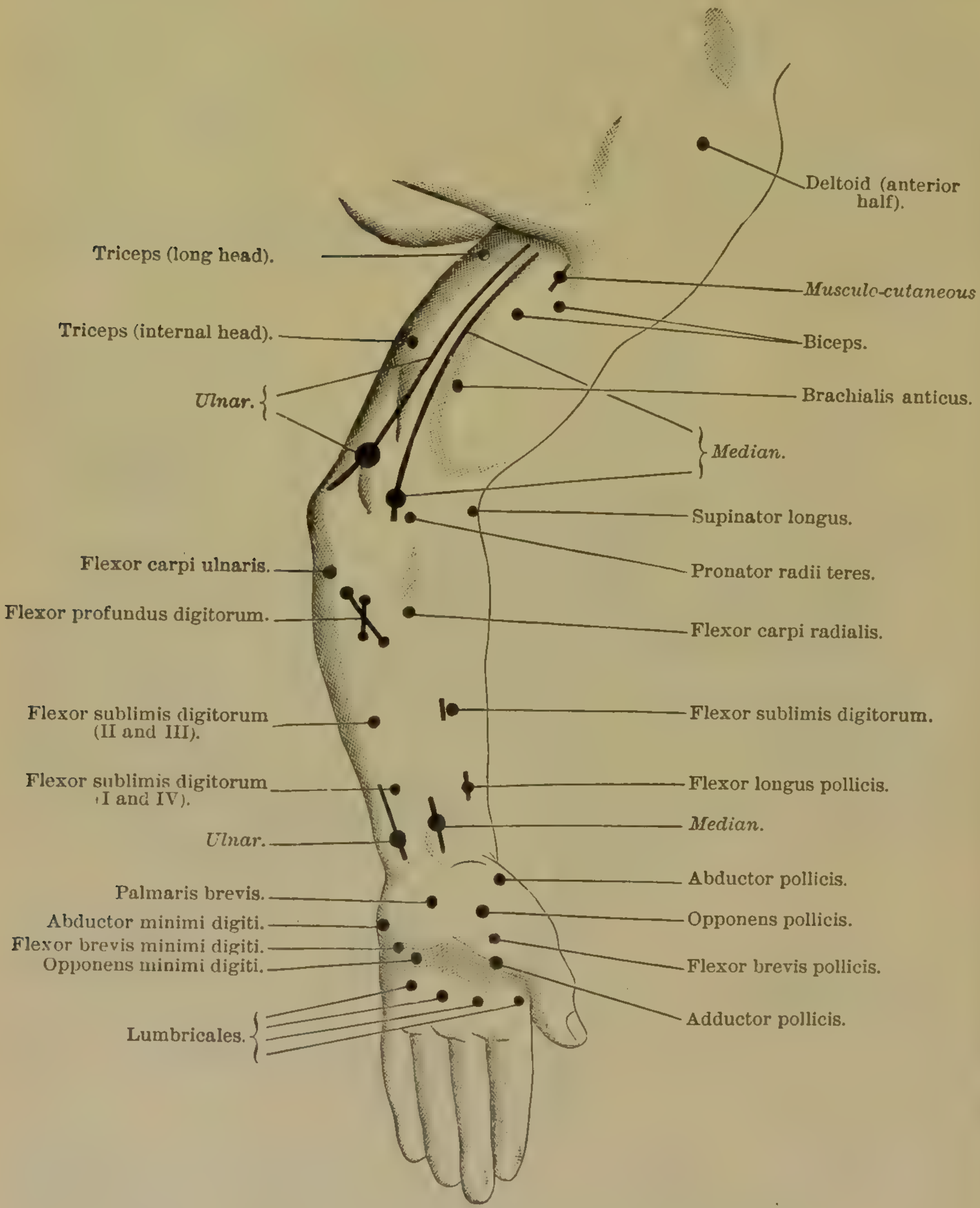

Fig. 69.

1. Lowest degree with weak currents: **KaSz**, KaO—, AnS—, AnO—.
2. Middle degree with stronger currents: **KaSZ**, KaO—, **AnSz**, **AnOz**.
3. Highest degree with very strong currents: **KaSTe**, **KaOz**, **AnSZ**, **AnOZ**.

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*Ka signifies cathode, An = anode, S = closure, O = opening, z (Zuckung) = weak contraction, Z = stronger contraction, Te = tetanus. Sometimes the increasing strength of the contractions is

The variations from the normal state seen under pathological conditions consist of quantitative and also of qualitative changes in the law of contraction. We term the simple increase or diminution of the electrical excitability in nerves or muscles, without simultaneous changes in the quality and order of the occurrence of muscular contractions, quantitative changes. The discovery of increased or diminished irritability of nerve and muscle can be made most easily in unilateral diseases, where we can compare the strengths of current required to obtain the minimal contraction on the diseased and healthy sides with each other. If we are dealing with bilateral or general diseases this is much harder to make out. We must then draw our comparisons from the conditions of excitability in normal individuals, where the different obstacles to conduction can be carefully estimated by the aid of a galvanometer, or by comparing the excitability of the nerve-trunks in the different parts of the body with one another. For this purpose we are

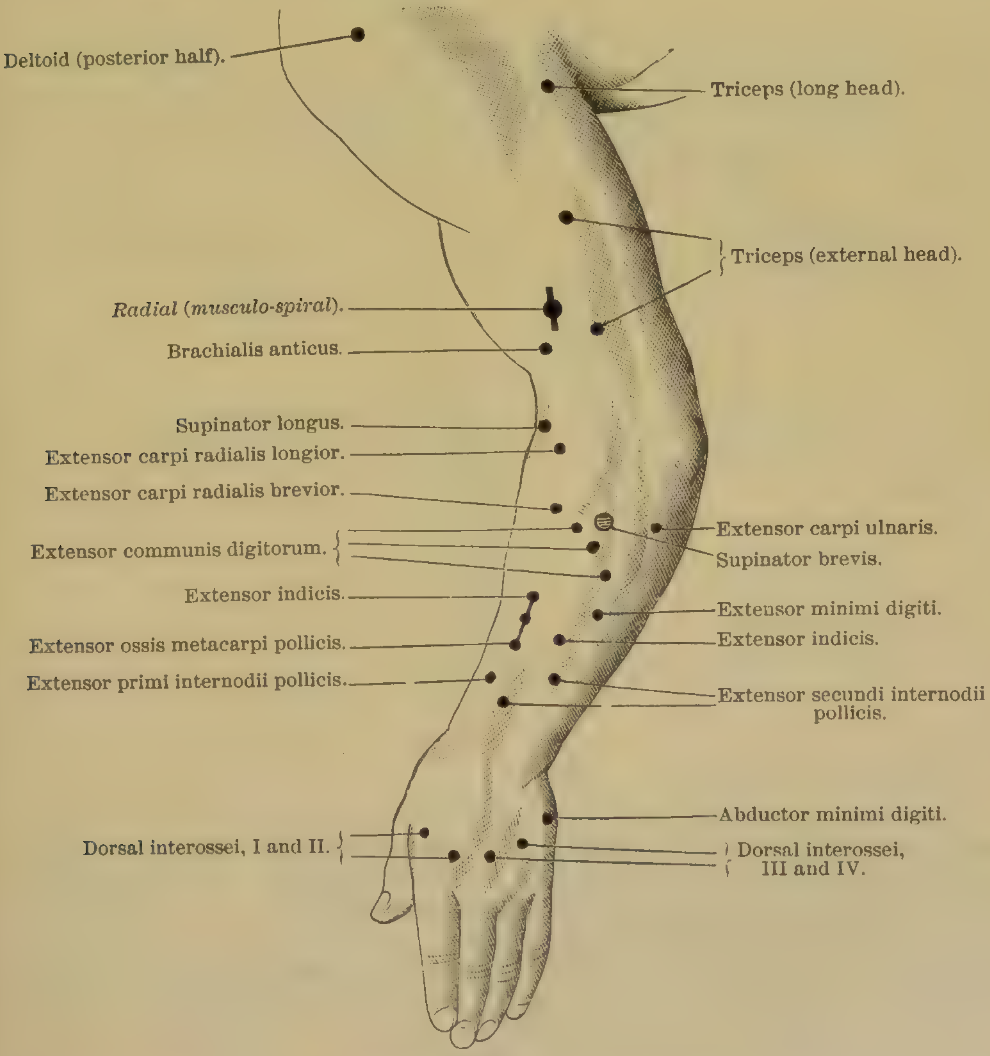
Deltoid (posterior half).
Triceps (long head).
Triceps (external head).
Radial (*musculo-spiral*).
Brachialis anticus.
Supinator longus.
Extensor carpi radialis longior.
Extensor carpi radialis brevior.
Extensor carpi ulnaris.
Supinator brevis.
Extensor communis digitorum.
Extensor indicis.
Extensor minimi digiti.
Extensor indicis.
Extensor ossis metacarpi pollicis.
Extensor primi internodii pollicis.
Extensor secundi internodii pollicis.
Abductor minimi digiti.
Dorsal interossei, I and II.
Dorsal interossei, III and IV.

FIG. 70.

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abbreviated by the signs Z, Z′ and Z″. [Many English and American writers on electricity use letters derived from the English names. Thus C stands for cathode, closure, and contraction. A and O have the same meaning. It seems to us clearer and conducive to greater harmony to retain the German abbreviations, which are simple and definite.—TRANS.]

usually content (following Erb's example) with comparing the superficial nerves, such as the frontal, accessory, ulnar, and peroneal, which are easily excited. An increase of electrical excitability is found in many fresh peripheral paralyses, and also in tetany. A diminution of electrical excitability is found quite frequently in bulbar and spinal paralyses, in progressive muscular atrophy, etc.

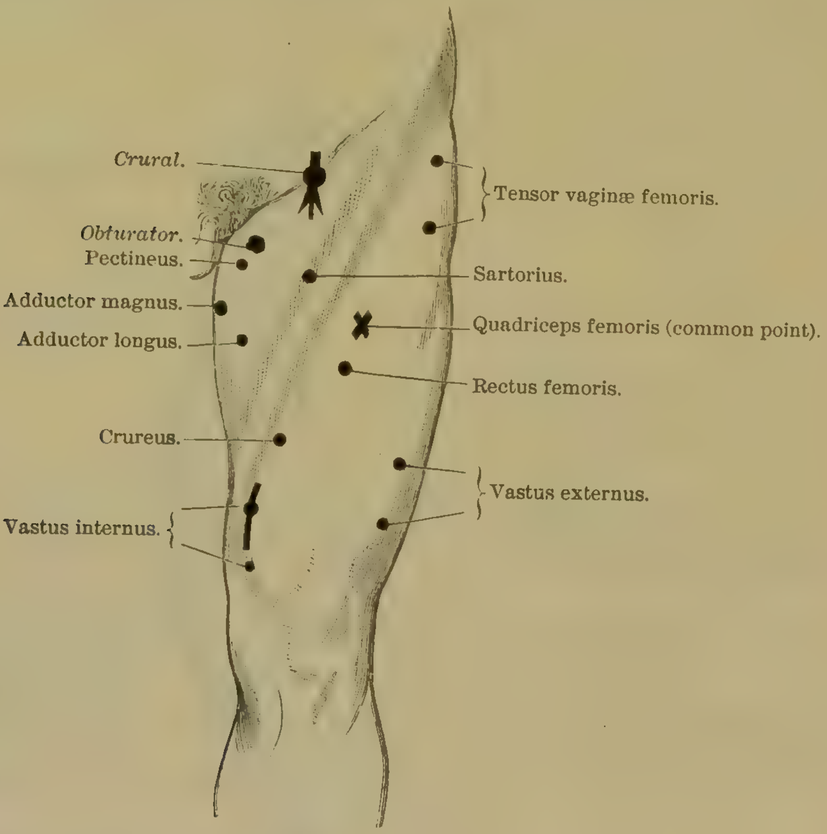

Fig. 71.

A significant advance in the investigation of the quantitative conditions of excitability has been rendered possible by the introduction of the "absolute galvanometer" (Hirschmann, Edelmann, etc.), by which the strength of the current can be read off directly in milliampères. By extensive investigations on healthy persons Stintzing has found certain limits within which the normal excitability of the different nerves varies. Deviations from these figures above or below accordingly permit us to recognize an increase or a diminution of the galvanic excitability. These limits are, for instance, for the frontal nerve, 1–2·5 milliampères; for the accessory nerve, 0·1–0·44 ma.; for the ulnar nerve (at a point one or two inches above the olecranon), 0·2–0·9 ma.; for the peroneal nerve, 0·2–2·0 ma. In these cases it is necessary, in order to obtain results that may be compared, always to use electrodes of the same area ("unit electrode" of 3 sq. cm).* An accurate measure of the absolute strength of the faradic current has not yet been found. We therefore content ourselves with the statement of the position of the outer coil [of the DuBois-Reymond apparatus] over the inner (coil distance, *Rollenabstand*), at which the first perceptible contraction occurs. Stintzing found as an average position for the frontal nerve, 128·5 mm.; for the accessory nerve, 137 mm.; for

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[* This is Stintzing's unit. Erb has proposed a larger one, a "normal electrode" of 10 sq. cm.—K.]

the ulnar nerve, 130 mm.; and for the peroneal nerve, 115 mm. We must refer to the special works for further details.

Much more important, however, than the simple quantitative changes of electrical excitability are those not merely quantitative but also qualitative deviations from the normal law of contraction, which were first discovered in certain forms of paralysis by Baierlacher in 1859, and were soon generally confirmed. Erb has given these the name of the "reaction of degeneration," because they are closely connected with the progress of certain anatomical changes in the paralyzed muscles and nerves.

In order to make the relations of the reaction of degeneration clear, let us select as an example any fresh peripheral paralysis and follow the changes in excitability to the two currents in the nerves and muscles. In a short time (two or three days) after the onset of the paralysis a gradually increasing decline in the faradic and galvanic excitability in the nerve begins. After one or two weeks the excitability is completely lost, so that from the nerve we can no longer provoke any trace of muscular contraction with the strongest faradic or constant current. During this time the excitability of the paralyzed muscles to the faradic current has also rapidly diminished, and finally has wholly disappeared. The case is quite different with direct galvanic excitement of the mus-

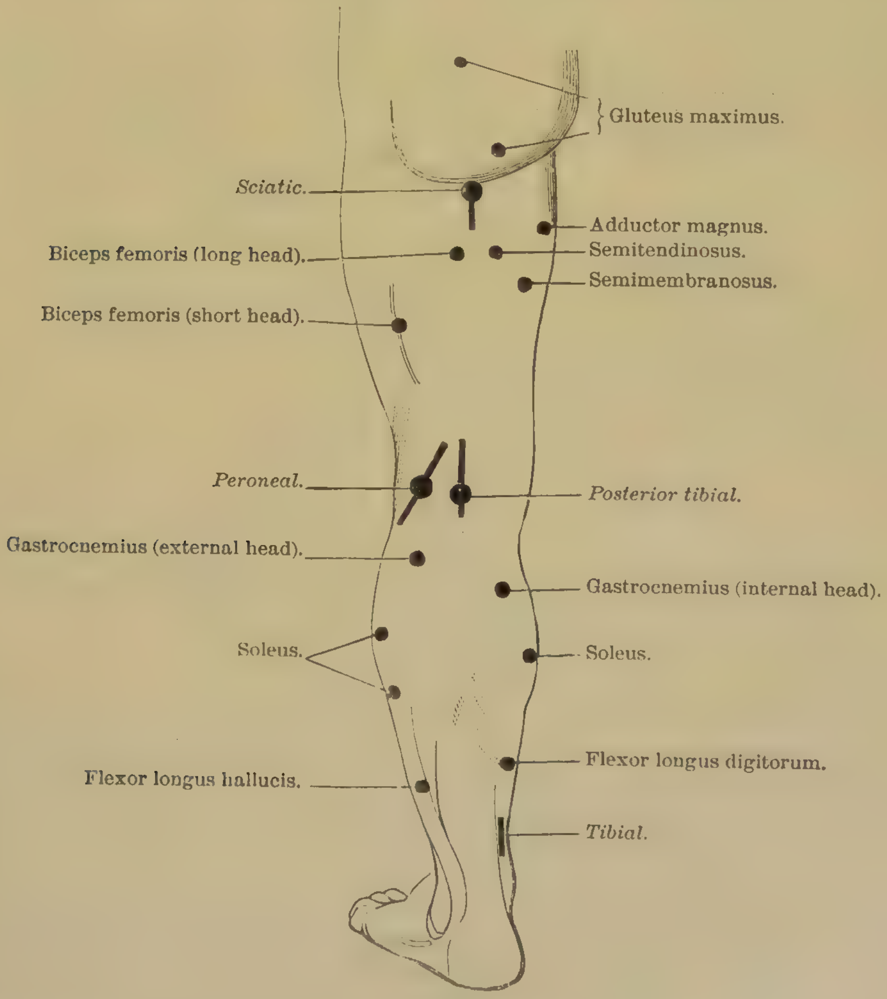

FIG. 72.

cles. Here we find at first a slight diminution, which in the second week passes to a decided increase of the galvanic muscular excitability. We now obtain marked muscular contractions with relatively very weak currents. Besides that, two other very important peculiarities are to be noted: 1. The muscular contractions are not short and lightning-like, as under normal conditions, but they seem quite sluggish, protracted, "worm-like," and often persist during the whole duration of the closure of the current. 2. The muscular contractions occur not only chiefly at cathodic closure (KaS), as under normal conditions, but the anodic closure contractions are as strong as the cathodic closure contractions (KaSZ), or even plainly exceed them. The cathodic opening contraction (KaOZ) is also frequently stronger. 3. It may also be mentioned here that the mechanical irritability of the muscles in such cases is usually increased.

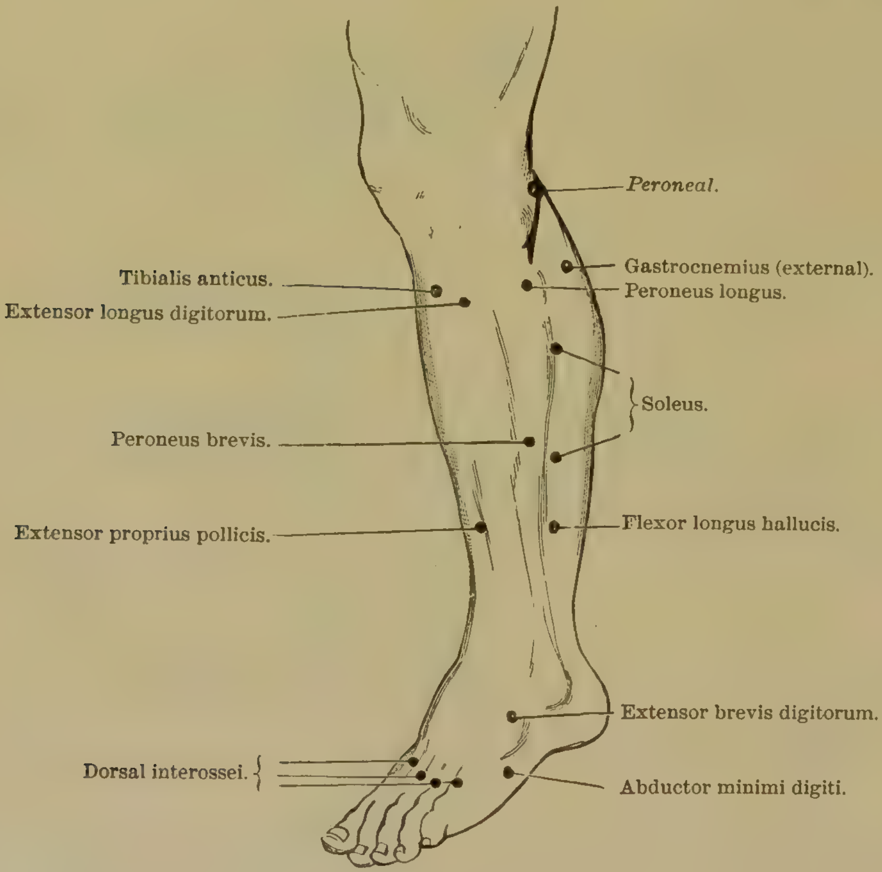

FIG. 73.

This second degree of the reaction of degeneration lasts from four to eight weeks. If the paralysis be severe and long continued, or incurable, at the end of this period comes a decline of the galvanic muscular excitability. The contractions become weaker, the strength of current necessary to produce them greater, and, finally, in incurable cases, even with the strongest currents, we can obtain only a little slow anodic closure contraction, or none at all. It is different, however, in the milder, curable cases. In these the passage to the normal condition gradually follows either the increase of the galvanic muscular excitability, or, in more protracted cases, its secondary decline. The contractions become more vigorous and shorter, the cathodic closure contraction (KaSZ) again predominates, the faradic muscular excitability and the faradic and galvanic excitability of the nerves finally return, and with them the old normal conditions are restored. A fact to be observed in these cases is of great interest, namely, that the voluntary motion in such cases often returns decidedly earlier than the electrical excitability of the peripheral nerves. We see, then, that a diseased nerve may be capable of conducting irritations coming from the brain, while the taking up of irritation, its direct excitability, is still completely lost.* In such cases we can obtain a muscular contraction by electrical irritation of the nerve above the point of lesion.

Besides the complete reaction of degeneration just described, there is also a so-called partial reaction of degeneration, which is not infrequent in milder cases. This may show itself in several forms, but it is chiefly when the diminution of the faradic and galvanic excitability in the nerves and the diminution of faradic excitability in the muscles is only of a slight degree, while the characteristic changes in the direct galvanic excitement of the muscles—increased excitability, slow contractions, and predominance of anodic closure contractions—are fully developed. In some cases the occurrence of slow contractions on faradic excitement of nerves and muscles has lately been observed—"faradic reaction of degeneration." In the course of atrophic paralyses we often see that the different varieties of reaction of degeneration pass into one another as the process advances or improves (Stintzing).

Anatomical Changes of the Nerves and Muscles in the Reaction of Degeneration; its Significance in Diagnosis and Prognosis.—As we have seen on page 538, all paralyses may be divided into two great groups—into atrophic paralyses, and paralyses without marked atrophy of the affected muscles. We have learned to recognize the necessary hypothesis of the "trophic" influence of the ganglion-cells in the anterior cornua of the spinal cord as the foundation of this distinction. In all cases where the disease affects these ganglion-cells, or is situated in the peripheral nerves, so that the trophic influence of the ganglion-cells on the muscles can no longer be of influence, we have a degenerative atrophy of the peripheral portion of the nerve, and of the muscle belonging to it. This degenerative atrophy is the anatomical cause of the symptoms of the electrical reaction of degeneration.

If we have to do with a peripheral paralysis, such as a traumatic lesion of a nerve-trunk, the portion of the nerve peripheral to the point of lesion is separated from its "trophic center" in the cord, and begins to undergo secondary degeneration. The first anatomical sign of the degeneration is a breaking down of the medullary sheath into large and small flakes and drops. The axis cylinder is also soon destroyed, so that the sheath of Schwann finally incloses only homogeneous fluid contents, which are in great part rapidly absorbed. At the same time there is an increase of the nuclei in the sheath of Schwann, and this increase, when the process is long continued, leads to a decided increase of the interstitial connective tissue in the nerve. The diminution and final loss of electrical irritability in the nerve are perfectly parallel to these anatomical changes, as we can easily understand.

The degeneration of the nerve involves its finest terminal branches in the muscles; but the muscle itself does not remain unchanged. The muscular fibers undergo a marked atrophy. They become much smaller, their transverse striation is less distinct, and they show in part a fatty and "granular" degeneration of their contents. The motor end-plates of the nerves in the muscles are spared for a comparatively long time, and they disappear only when the degeneration of the muscles has reached the highest degree. In regeneration, however, the end-plates are already completely restored at a time when the nerve-fibers themselves are still found destroyed (Gessler). Some fibers show that peculiar yellow homogeneous character which we call "waxy degeneration." In addition to this, there is a considerable increase of the muscular nuclei, and in the later stages a great new growth of interstitial connective tissue, often associated with a marked deposit of fat. These muscles, thus altered, now react only to the galvanic current, and in the manner above described. The particular cause of this remarkable fact is, of course, still completely unknown to us.

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* This is also connected with the repeated observations by Erb, Bernhardt, and others, that in lesions of peripheral nerves, and perhaps also in spinal diseases, reaction of degeneration can sometimes be made out even in those muscles which show no essential limitation of their voluntary mobility. In these cases the electrical examination points to finer anatomical disturbances which have not led to the loss of voluntary excitability.

In the incurable cases the processes of degeneration just described gradually advance, but, in the cases that recover, a number of processes of regeneration begin sooner or later. We can not here go into the finer details, which are still, in many respects, the subject of controversy; but it is certain that new nervous and muscular fibers are formed, and that, hand in hand with the anatomical processes of regeneration, first the voluntary motion, and later the electrical excitability of the paralyzed parts, gradually return again.

The same anatomical changes, which we have just described as a secondary degeneration in lesions of the peripheral motor nerves, also develop, if the primary disease has its seat in the anterior cornua of the gray matter of the spinal cord—that is, in the trophic centers themselves. In these cases, of course, the form of the disease has nothing to do with it. Both in the different forms of inflammation and of primary atrophy, and also in new growths, which affect the anterior gray matter of the cord, a secondary degeneration, with pronounced reaction of degeneration, develops from the anterior roots of that portion of the cord affected to the ends of the peripheral nerves, and even to the corresponding muscles. We shall also learn to recognize a number of primary degenerations of the peripheral nerves, such as primary neuritis, diphtheritic and toxic paralyses, etc., which likewise show the same anatomical changes, and likewise give, as a result of these, electrical reaction of degeneration. In all cerebral paralyses, however, and in those spinal paralyses where the cause of the paralysis is situated above the part of the anterior gray cornua concerned, the degenerative atrophy, and also the reaction of degeneration, are entirely wanting.

We thus see that the reaction of degeneration, in regard to diagnosis, at once permits us to decide that the disease is situated in the gray matter of the cord, or in the peripheral nerves. It does not permit any further distinction. In regard to prognosis, it teaches us that anatomical changes have taken place in the nerves and muscles, from which a restoration is still very possible, but at all events it can take place only after the lapse of a longer time, at least two or three months. We will soon learn to recognize a number of mild peripheral paralyses in which there is generally no reaction of degeneration. From the absence of reaction of degeneration we can then draw the conclusion, with certainty, that no coarse anatomical changes are present in the nerve, and that we may expect after the trouble a much more rapid recovery, perhaps in three or four weeks. The partial reaction of degeneration, above mentioned, is also an important symptom in regard to prognosis. It shows that severe anatomical changes have taken place in the muscles but not in the nerves, and hence it always permits a more favorable prognosis as to time than in the cases with complete reaction of degeneration.

CHAPTER II.

THE DIFFERENT FORMS OF PERIPHERAL PARALYSIS.

1. PARALYSIS OF THE OCULAR MUSCLES.

Ætiology.—The largest part of all the ocular paralyses arise from affections which involve either the peripheral nerves or their nuclei in the brain-stem. We accordingly make a distinction between peripheral and nuclear paralyses of the ocular muscles. As we shall take up the latter more fully in the description of chronic bulbar paralysis, we have here to mention only the most important and most frequent causes of the peripheral ocular paralyses. These are as follows:

1. Traumatic injuries, which directly affect the nerve-trunks or their branches: blows on the eye, knife-stabs, fractures of the skull involving the orbit or the base of the skull, and the like.

2. Compression of the nerves from morbid processes in their neighborhood. Tumors of the base of the skull, especially, very often lead to ocular paralyses. Periostitis at the base of the skull or in the orbit may also cause similar symptoms, and so do syphilitic diseases of the nerves and their surroundings, the meninges or periosteum, aneurisms of the basilar artery, acute or chronic meningitis in its different forms, etc. In all these cases we usually have to do with a pure mechanical compression of the affected nerves by the morbid new growths in their immediate vicinity. More rarely the pathological process directly invades the nerves themselves.

3. The so-called rheumatic ocular paralyses are quite common. These arise after some decided exposure to cold, such as a draught from an open window, and are in all probability very largely of a peripheral nature. They depend, as is supposed, upon an acute neuritis of the affected nerve, and hence are to be regarded as completely analogous to the other rheumatic paralyses, like rheumatic facial paralysis. Among the "rheumatic paralyses" we usually class the paralyses which apparently come on spontaneously and completely recover, for which no other special cause can be made out.

4. The ocular paralyses that sometimes arise after certain acute diseases are also of a peripheral nature, and are due to a degenerative neuritis of the affected nerves. They are most frequent as a result of diphtheria, and are much more rare in typhoid, acute rheumatism, etc. Of chronic diseases, diabetes mellitus may sometimes give rise to ocular paralyses, especially to paralysis of accommodation.

A fuller account of the very important ocular paralyses in tabes dorsalis will be found in the description of that disease.

Symptoms.—Since we must refer to the text-books of ophthalmology in regard to the more precise symptomatology, and the more special methods of ophthalmic investigation, we will here give only a brief review of the chief symptoms of ocular paralyses which are important in nervous pathology.

The disturbance in the mobility of one eye is noticed by the patient himself from the appearance of double images—double vision, or diplopia. These arise because, on looking to one side, the eye on the paralyzed side can not be brought into the corresponding position, and consequently the retinal images no longer fall upon the same spots. In pathological convergence of the visual axes homologous double images arise, in pathological divergence crossed images—that is, in the first case, on closing one eye, the image disappears on the same side, in the second case it disappears on the opposite side. By alternately fixing the gaze on one or the other of two fingers held in line with each other, and by regarding the disappearance, on closing one eye, of the double image of the finger not fixed, we can easily demonstrate this on ourselves. If, then, crossed double images arise, for example, on looking to the right, we must have a divergent strabismus—that is, an imperfect function of the left internus; but if there are homonymous double images, there must be a convergent strabismus, and consequently a weakness in the right abducens. It makes it much easier to test the double images if we put a colored glass before one of the patient's eyes. False projections of the visual field arise, as a result of the double images and of the abnormal strength of innervation which the patient exerts, so that the patient's judgment of the position of external objects is uncertain. In the more extensive ocular paralyses this often leads to a pronounced feeling of dizziness. In order to avoid this unpleasantness many patients confine themselves to monocular vision, close the affected eye, or put their heads in a position to avoid the double images.

Physical examination gives the following results, according to the extent of the paralysis:

In complete paralysis of one **oculo-motor nerve** (the levator palpebræ superioris, the superior, inferior, and internal recti, the inferior oblique, the sphincter of the iris, and the ciliary muscle) the first thing that is noticed, besides the disturbance in the movements of the eye, is the more or less complete drooping of the upper lid—ptosis. If we ask the patient to follow with his eyes the motion of any object, like the finger, held before him, keeping his head still, we notice at once that the affected eye does not move upward, downward, or inward. The pupil is dilated (mydriasis) and no longer contracts to light. Accommodation is lost, and distinct vision for near objects is impossible. As a rule, the whole eye seems rather prominent (paralytic exophthalmus), because the backward traction of the recti is very largely absent. In old oculo-motor paralysis there is often a secondary contracture in the unparalyzed external rectus (and superior oblique), by which the eye is persistently drawn outward. Partial oculo-motor paralyses are not infrequent, especially isolated ptosis, isolated paralysis of the internal, inferior, or superior rectus, or isolated paralysis of accommodation, and they may usually be easily recognized from what has been said.

Paralysis of the **abducens** is characterized by the inability to move the external rectus. The eye can no longer be moved, or it can be moved only imperfectly, outward beyond the median line. In old paralysis the eye is drawn inward from a secondary contracture of the internal rectus, and convergent strabismus arises. Paralysis of the abducens may be isolated, bilateral, or combined with other ocular paralyses.

Paralysis of the **trochlear nerve**, the superior oblique muscle, is not perfectly easy to recognize; but it is rarely of special practical importance. The action of the superior oblique coincides with that of the inferior rectus. The paralysis of the former, therefore, is soonest recognized by the retardation of the eye in movements downward and also inward, and sometimes by the failure of the eye to revolve, which rotation normally takes place on looking downward, and is due to the superior oblique muscle. This latter movement takes place in each eye inward, toward the nose, about a sagittal axis, in such a way that the left eye is turned from the left and up to the left and downward, and the right eye from the right and up to the right and downward. In regard to diagnosis it is also characteristic that the double images in trochlear paralysis appear only in the lower half of the field of vision, and especially on looking downward. Hence it happens that the disturbance of vision is especially manifest in going up or down stairs.

Finally, we must mention a symptom to be observed in almost every ocular paralysis—the so-called secondary deviation of the healthy eye. If, after the sound eye has been covered, we have the paretic eye fix itself upon a point which it can not reach at all, or which it can reach only after the utmost exertion, we see, when the covering hand is taken away from the sound eye, that the latter has been moved much too far in the corresponding direction. The abnormal exertions of innervation with the affected eye, somewhat after the analogy of certain associated movements, pass over to the associated muscle of the healthy side and cause in it too extensive a contraction.

The following must be added in regard to the separate forms of ocular paralysis: Rheumatic ocular paralysis affects the abducens most frequently, and not infrequently the oculo-motor or one of its branches, as in isolated ptosis. A case observed by us a short time ago is, at any rate, rare, in which exposure to severe cold had been followed by a complete paralysis of all the muscles of the right eye, with complete ptosis, and almost absolute immobility of the eye in any direction. The rheumatic ocular paralyses are almost always acute in their onset, and they are often during the first period associated with sensations of pain about the eye and in the head. Vomiting (of reflex origin ?) is also not rare at the beginning of the affection. The course of most cases is favorable, since they usually completely recover in a few weeks, though sometimes not for months. In some cases the paralysis may remain stationary. The diphtheritic ocular paralyses usually appear, like the other diphtheritic paralyses, a week or two after the termination of the disease. They most frequently affect the muscles of accommodation, so that the patient complains chiefly of indistinct vision for near objects; but we sometimes see paralyses of the external ocular muscles also, the abducens or the internal rectus. The prognosis of diphtheritic paralyses is almost invariably favorable. Finally, we must mention here the "periodical oculo-motor paralysis," to which Möbius and others have lately called attention, and whose nature is still almost wholly inexplicable. In the cases of this sort repeated paralyses of one oculo-motor nerve, often associated with headache and vomiting, just as in migraine, come on at longer or shorter intervals (in women sometimes at the menstrual period) in the same individual, who has often had them since childhood. The individual attack sometimes lasts only a few days, but often some weeks. The attacks usually become gradually more severe later on.

Nothing generally applicable can be said as to the course and prognosis of the other forms of ocular paralyses, since in them everything depends upon the form of the underlying disease.

Treatment.—In regard to the fulfillment of any possible causal indication we must remember especially that a syphilitic origin of ocular paralysis is not very rare. Iodide of potassium and energetic mercurial inunction may sometimes give very good results in such cases. Hence these remedies must also be tried in doubtful cases.

Of other remedies galvanic treatment gives the speediest relief. We pass weak currents transversely through the temples, or, what is usually better, put the anode to the back of the neck and apply the labile cathode to the closed eye, especially to the region corresponding to the paralyzed muscles. Great caution, weak currents, and the avoidance of any great variations in the currents are of course necessary. We may also try preparations of strychnine internally, or, better, subcutaneously in the vicinity of the eye. We must refer to special treatises with regard to a correction of the double images by prismatic spectacles, or in regard to operations, such as tenotomy, that are sometimes performed.

2. PARALYSIS OF THE MOTOR BRANCH OF THE TRIGEMINUS.

(*Paralysis of the Muscles of Mastication.*)

Paralysis of the muscles of mastication, the masseters and temporals, supplied by the third branch of the trigeminus, is a rare affection. It is most frequently seen in diseases of the base of the skull which compress the motor branch of the fifth. We shall also learn later on to recognize paralysis of the muscles of mastication as a rare symptom of chronic bulbar affections.

The chief symptom of motor paralysis of the trigeminus is the difficulty or impossibility of chewing. In unilateral paralysis the patient can chew only on the healthy side; in bilateral paralysis he can no longer chew at all. The lower jaw hangs loosely down and can also no longer be moved sideways, from the co-existing paralysis of the pterygoids. There are often sensory disturbances in the distribution of the trigeminus at the same time.

The prognosis and treatment depend upon the primary disease. Local faradization or galvanization of the paralyzed muscles is to be tried.

3. FACIAL PARALYSIS.

(*Mimetic Facial Paralysis. Bell's Palsy.*)

Ætiology.—Facial paralysis is one of the commonest peripheral paralyses, as we can understand from the exposed position of the nerve and its course through the narrow Fallopian canal. The most important causes of it are: 1. Exposure to cold, draughts, sleeping by an open window, riding in the cars with the window open, etc. The paralyses arising in this way are termed "rheumatic," and we also include in this class those peripheral paralyses which are apparently spontaneous—that is, those without any marked exposure to cold that can be discovered. In all these cases we probably have to do with a neuritis of the nerve-trunk, which comes on in a way as yet unknown. [In a recent autopsy of a case of so-called "rheumatic" facial paralysis, Minkowski found a simple degenerative parenchymatous neuritis, without any swelling of the nerve sheath or increase of connective tissue.—K.] 2. Diseases of the middle ear and caries of the petrous bone. As the facial passes through the Fallopian canal, which is in the immediate vicinity of the tympanic cavity, we can easily understand how, in caries of the petrous bone and in purulent affections of the middle ear, inflammation may often invade the trunk of the facial, or how the facial may be compressed by inflammatory exudations, etc. 3. In rare cases a pressure paralysis of the facial nerve arises in swelling of the parotid gland or tumors in its vicinity. 4. Diseases of the base of the skull or brain, tumors, syphilitic new growths, and acute or chronic inflammations, often give rise to the development of a facial paralysis by invasion of the trunk of the facial or compression of it. 5. We shall have to speak repeatedly of the frequent implication of the facial nerve in diseases of the brain and medulla in the following sections.

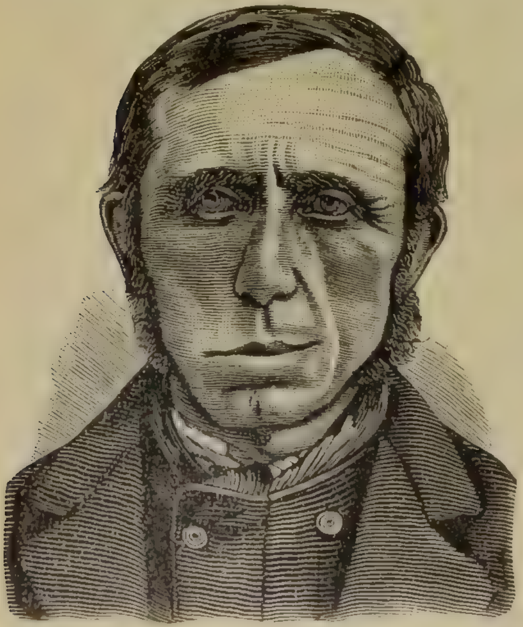

FIG. 74.—Right facial paralysis (after SEELIGMÜLLER). The folds are smoothed out and in part entirely absent on the paralyzed side, while they are strongly marked on the left. The mouth and nose are drawn to the left.

Symptoms and Course.—The manifold character and different functions of the nerves, which unite in the trunk of the facial, are the cause of the quite large array of symptoms in facial paralysis. The paralysis of the facial muscles of expression is the most striking and characteristic (see Fig. 74). The paralyzed half of the face is lax and expressionless, the wrinkles in the forehead are obliterated, the eye is abnormally wide open and waters (epiphora), the naso-labial fold is obliterated, the corner of the mouth droops, and saliva frequently flows from it. The paralysis becomes still more marked on any movement of the face, on wrinkling the forehead, turning up the nose, laughing, talking, whistling, or inflating the cheeks. The eye can be only partly closed. On attempting it, the upper lid sinks down from its weight (a weakening of the levator palpebræ superioris), the eye is turned upward, so that the pupil is covered, but quite a wide space is left between the eyelids (lagophthalmus). This defective closure of the lids facilitates the entrance of dust, etc., into the eye, and sometimes gives rise to conjunctivitis, or even to severer inflammation of the eye. Speech is rendered difficult and indistinct from defective movements of the lips, and mastication becomes impaired from the imperfect movement of the cheeks. In many cases we also find a paresis of the soft palate on the affected side; the fibers from the facial pass through the superficial petrosal nerve to the spheno-palatine ganglion, and thence to the soft palate. It droops more, and on phonation the soft palate is raised obliquely to the healthy side. No general rule can be given as to the position of the uvula, as this varies very much even under normal conditions.

Disturbances of taste in the anterior two thirds of the tongue have been repeatedly found on the paralyzed side, but they usually attain only a slight degree. They are explained by an affection of the fibers of the chorda tympani, which run for some distance in the facial, as has been described on page 533. At the beginning of the paralysis many patients complain of subjective sensations of taste. Later on the dullness of taste may often be discovered by careful testing. Tactile sensibility in the tongue is only exceptionally diminished (sensory fibers in the chorda ?). There is sometimes a diminished secretion of saliva (fibers in the chorda), which produces an abnormal feeling of dryness in the patient's mouth on the paralyzed side. Disturbances of hearing are frequent, but they are usually due to some complicating aural trouble (*vide supra*), or to a co-existing affection of the acoustic nerve. Paralysis of the stapedius muscle, however, sometimes seems to cause symptoms, including a marked sensitiveness to all loud sounds, and even an abnormal acuteness of hearing, especially for low notes (hyperacusis oxyokoia). This symptom is due to the fact that in paralysis of the stapedius its antagonist, the tensor tympani, causes a greater tension of the membrana tympani. Reflex movements, winking, etc., are, of course, lost in complete peripheral facial paralysis. For the special reflexes, which are often seen in the later stages of facial paralysis, *vide infra.*

By testing all the symptoms described, in most cases we can decide with accuracy upon the place where the break in conduction in the facial must occur. If we examine the accompanying plan of the facial (Fig. 75), devised by Erb, we can easily understand the following chief symptomatic forms of facial paralysis:

1. Paralysis of the facial muscles; but taste, secretion of saliva, hearing, and soft palate normal; seat of the affection in the portion between 1 and 2, usually the trunk of the facial below the Fallopian canal.

2. Paralysis of the facial muscles, disturbance of taste, and eventually diminished secretion of saliva; but hearing and soft palate normal; seat of the affection within the Fallopian canal between 2 and 3.

3. Paralysis of the facial muscles, disturbance of taste, diminished secretion of saliva, abnormal acuteness of hearing; but soft palate normal; seat between 3 and 4.

4. Paralysis of the facial muscles, disturbance of taste, diminished secretion of saliva, abnormal acuteness of hearing, and paresis of the soft palate; seat in the geniculate ganglion between 4 and 5.

5. Paralysis of the facial muscles, diminished secretion of saliva, abnormal acuteness of hearing, paresis of the soft palate, but no disturbance of taste; seat above the geniculate ganglion between 5 and 6. [Gowers doubts whether there be paralysis of the soft palate in lesions of the facial nerve. If it exist at all it is very rare. Consequently the conclusions just given can hardly be accepted in full.—K.]

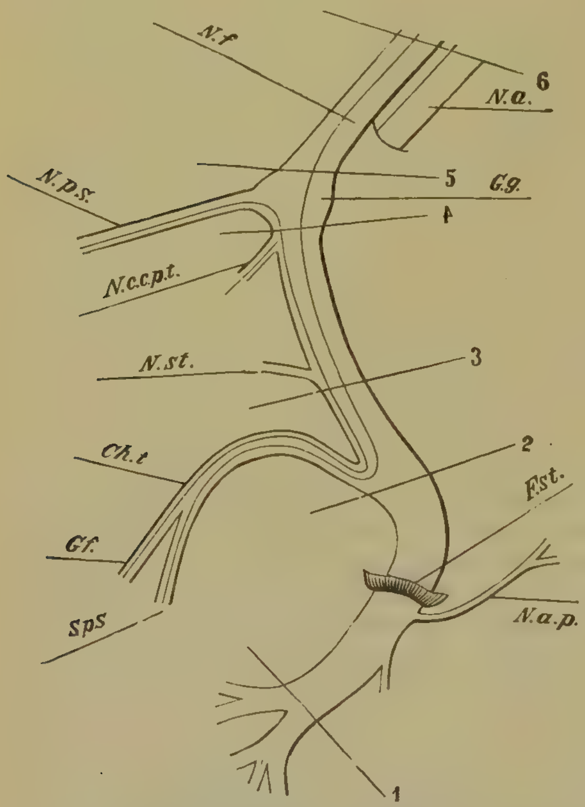

FIG. 75.—Schematic representation of the trunk of the facial from the base of the skull to the pes anserinus. Different localizations of the lesion in paralysis. *N.f.* Facial nerve. *N.p.s.* Great superficial petrosal. *N.c.c.p.t.* Nerve communicating with the tympanic plexus. *N.st.* Stapedius. *Ch.t.* Chorda tympani. *Gf.* Fibers of taste. *Sps.* Nerve governing the secretion of saliva. *N.a.* Acoustic nerve. *G.g.* Geniculate ganglion. *F.st.* Stylomastoid foramen. *N.a.p.* Posterior auricular nerve.

The changes in electrical excitability, and some other changes, may be best described in connection with the course of facial paralysis. The paralysis usually begins quite suddenly; less frequently it is more gradual. Sometimes there are, for a short time, subjective prodromata, such as abnormal sensations of taste, slight ringing in the ears, and above all painful sensations in the ear and face, which symptoms may be referred to the beginning of acute inflammatory processes in the nerve. In a few cases the occurrence of herpes vesicles has been observed in the distribution of the affected facial, a condition which may be soonest explained, in accordance with what was said on page 525, by reference to the numerous anastomoses between the branches of the facial and those of the trigeminus.

In regard to the further course we distinguish the three following forms:

1. *The mild form of facial paralysis*, to which especially many rheumatic paralyses belong. The affection is usually referred only to the facial muscles, disturbances of taste, etc., being wholly absent. Electrical excitability in the facial and the paralyzed muscles remains entirely normal. Recovery is rapid, usually in two or three weeks. In these cases we may certainly suppose that there are no severe and deep-seated anatomical changes in the nervous or muscular fibers.

2. *The middle form of facial paralysis* (Erb). In this there is no complete reaction of degeneration, but only a partial one. The excitability of the nerve is somewhat diminished, but it is not lost. In the muscles, however, in about two or three weeks, there appears a decided increase of galvanic excitability to direct excitement. The anodic closure contraction (AnSZ) is also greater than the cathodic closure contraction (KaSZ), and the contractions are slow. In regard to prognosis we may decide from this that the recovery will still be quite rapid. It usually follows in from four to six weeks.

3. *The severe form of facial paralysis* is that in which there is a complete reaction of degeneration in the nerve and muscles, the details of which we have

learned in the previous chapter, loss of faradic and galvanic excitability of the nerve, loss of faradic excitability of the muscles, and quantitative and qualitative changes in the galvanic excitability of the muscles. In this form there are always coarse processes of degeneration in the nerve and muscles, so that recovery follows, if at all, only after two to six months, or even later, because the processes of regeneration require at least as much time for their accomplishment. We often see in the later stages of these cases special symptoms of motor irritation (Hitzig). These consist, first, of a more or less marked tonic contracture of the paralyzed muscles, which is sometimes very striking; second, of single spasmodic contractions of the muscles; third, of special associated movements—if the patient closes his eyes, winks, etc., there always follows a marked distortion of the corner of the mouth, which can not be suppressed; fourth, of an increased reflex irritability—on pricking the skin, or blowing on it, vigorous muscular contractions follow. We have often ourselves seen contractions in the affected facial muscles following a blow on the bridge of the nose, on the nasal bone, or on the forehead on the healthy side. These reflexes come from the skin, or perhaps in part from the periosteum and the fasciæ also. All these symptoms may last for a very long time—for years in incurable or in imperfectly cured cases. The above-described associated movement in the angle of the mouth on shutting the eyes we have repeatedly seen in students who have had an injury of a peripheral branch of the facial in a duel.

Prognosis.—The prognosis of facial paralysis depends, of course, in the first place, upon the primary disease, if any exists. Paralysis in tumors of the base of the brain, caries of the petrous bone, etc., is almost always incurable. The course of the paralysis in affections of the middle ear depends upon the curability of the latter disease. Very important data for the accurate prognosis of rheumatic paralysis are given by the electrical examination, as has been carefully described above. Of course, we can never form a definite judgment from this at the beginning of the paralysis, but only at the end of the first week. If, at the end of the first week or fortnight, the electrical excitability of the nerve still remains normal, we can almost certainly prophesy a rapid and favorable course. If reaction of degeneration appears, we can not count upon recovery in the most favorable cases before two or three months. As a rule, relapses do not occur, but we saw a man of about thirty who had a peripheral facial paralysis four times within a few years, which disappeared each time after a few weeks—a condition which possibly is to be regarded as analogous to the "periodical oculo-motor paralysis" (see page 557).

Diagnosis.—The symptoms of facial paralysis are so pronounced that the paralysis itself can always be easily recognized. In regard to the precise form of the paralysis and its cause, we can often decide only by considering the ætiological factors, such as injuries, exposure to cold, or aural affections. In distinguishing between peripheral and central (bulbar or cerebral) paralyses, the other co-existing bulbar or cerebral symptoms must also be considered. We shall learn to recognize more accurately later the different modes in which, in these cases, facial paralysis may be combined with paralysis of the other cerebral nerves, or the nerves of the extremities. In doubtful cases electrical examination is often of decisive value. Reaction of degeneration can be present only in peripheral paralysis, or in such bulbar paralyses as affect the fibers of the facial below the facial nucleus or affect the nucleus itself. In all the genuine cerebral paralyses the electrical excitability is perfectly retained. It may also be mentioned here briefly that in all cerebral facial paralyses the frontal portion of the facial is usually unaffected, while in peripheral paralysis it also is paralyzed. The power to close the eye is usually not affected in cerebral facial paralysis.

Treatment.—The treatment of the underlying disease is of the greatest importance in all cases where any aural affection, any removable compressing swelling, as of the parotid, or syphilis, lies at the bottom of the trouble. The methods of treatment indicated in such cases are self-evident. In other cases electricity is the only remedy which can give sure results, although we must not overestimate its efficiency. In fresh facial paralysis we may recommend the stabile conduction of a weak constant current through the auriculo-mastoid fossæ, four to six times a week for two or three minutes, at first the anode, then the cathode, to the affected side. Later on the chief treatment is peripheral galvanization, or eventually faradization of the muscles. We place the anode in the auricular fossa and slowly stroke the different nerve-branches and the muscles with the cathode. We can often confirm the fact, immediately after each sitting, that the eye closes better after galvanization of the orbicularis. Faradization perhaps excites a reflex irritation of the nerves from irritation of the skin, and hence is of service.

Of other remedies we may mention subcutaneous injections of strychnine, seven to fifteen minims of a one-per-cent. solution of the sulphate three or four times a week, although it is only rarely of use. In secondary contractures we may obtain favorable results by methodical stretching of the muscles by wooden wedges inserted under the cheeks and by massage.

4. PARALYSES IN THE REGION OF THE MUSCLES OF THE SHOULDER.

Isolated paralyses of these muscles are rare, with the exception of the practically important paralysis of the serratus. Disturbances in their functions are more frequent, as one symptom in complicated paralytic states, especially in progressive muscular atrophy. The diagnosis of these paralyses in detail is often very difficult.

PARALYSIS OF THE STERNO-CLEIDO-MASTOID (*spinal accessory nerve*).—The chin is somewhat raised and turned toward the affected side in consequence of the antagonistic contracture of the other sterno-mastoid. Motion in the opposite direction is difficult. In bilateral paralysis of this muscle it is very difficult to turn the head with the chin raised, and it can be done only imperfectly.

PARALYSIS OF THE TRAPEZIUS (*spinal accessory nerve*).—The shoulder sinks downward and forward so that the supra-clavicular fossa becomes deeper. The median border of the scapula is not parallel to the vertebral column, as under normal conditions, but it runs obliquely from below and inward, upward and outward. Voluntary raising of the shoulder, "shrugging the shoulder," is impaired, and it can be done only by the levator scapulæ. The drawing back of the shoulder, approximating it to the vertebral column, is difficult, and can be done only by the rhomboidei. Raising the arm above the horizontal is also affected, from the impaired fixation of the scapula.

PARALYSIS OF THE PECTORALIS MAJOR AND MINOR (*anterior thoracic nerves*).—Abduction of the upper arm is difficult or impossible. The hand can no longer be placed on the shoulder of the healthy side.

PARALYSIS OF THE RHOMBOIDEI AND THE LEVATOR ANGULI SCAPULÆ (*dorsalis scapulæ nerve*) can be certainly recognized only when the trapezius is also paralyzed. Then the approximation of the scapula to the vertebral column (rhomboidei) and the raising of the scapula (levator anguli scapulæ) are completely abolished.

PARALYSES OF THE LATISSIMUS DORSI (*subscapular nerves*).—There is no deformity when at rest, but the arm can not be strongly adducted, and the hand can not be placed on the sacrum.

PARALYSIS OF THE ROTATORS OF THE HUMERUS INWARD AND OUTWARD.—In paralysis of the inward rotators, the teres major and subscapular, innervated by the subscapular nerves, the arm, when rotated outward, can not be brought back again to its normal position. All manipulations, too, which the paralyzed arm tries to make on the opposite side of the body, are considerably impaired. In paralysis of the outward rotators—the infra-spinatus, innervated by the supra-scapular nerve, and the teres minor, innervated by the axillary—rotation of the arm outward is abolished. In writing and sewing (using the needle), the paralysis causes very marked disturbance.

PARALYSIS OF THE SERRATUS ANTICUS MAJOR (*paralysis of the long thoracic nerve*).—This paralysis is quite common, and therefore is of practical importance. Its most frequent cause is traumatic action on the nerve; therefore it is especially common in porters, soldiers, etc. Besides this, serratus paralysis sometimes arises from exposure to cold, "rheumatic serratus paralysis," and also as a result of infectious diseases, such as typhoid, and as one symptom of progressive muscular atrophy, especially in the juvenile form.

As the arm hangs down quietly, the scapula on the paralyzed side stands out a little from the chest-wall, from the action of the antagonists (the rhomboidei, the levator anguli scapulæ, and the trapezius), its lower angle is a little approximated to the vertebral column, and therefore its median edge runs obliquely upward and outward. If the patient tries to raise his arm, he can raise it only to the horizontal position, and we fail to see the projection of the tense indentations of the serratus on the lateral wall of the chest; but as soon as we seize the scapula firmly and push it forward—that is, supply the missing action of the serratus—the patient can at once raise the arm. If the arm is raised upward to the horizontal

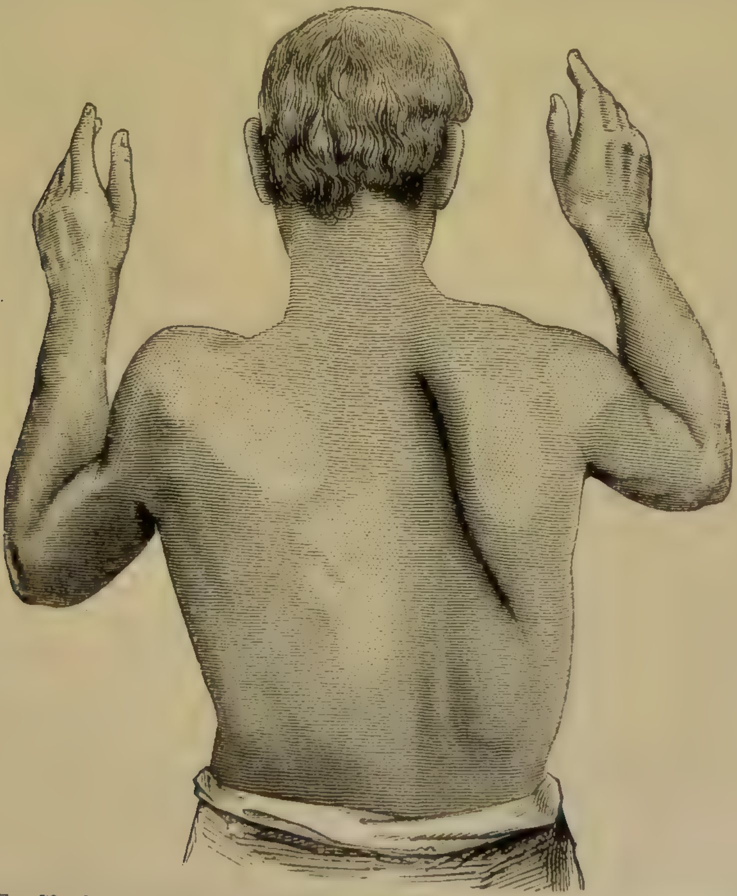

FIG. 76.—Paralysis of the right serratus. (From a photograph by DUCHENNE.)

line, the scapula approaches the vertebral column; if it is raised forward, there appears a very characteristic wing-like projection of the inner border of the scapula, so that we can touch with the hand the inner surface of the scapula (see Fig. 76). Adduction of the arm and laying the hand on the other shoulder are also disturbed. The cutaneous sensibility of the chest is, as a rule, normal.

The course of serratus paralysis is usually tedious. Recovery does not take place for several months, as a rule. Many cases are incurable. Treatment consists chiefly in the application of electricity to the paralyzed nerve and muscles.

5. Paralyses of the Muscles of the Back.

Of the paralyses of the muscles of the back, which are rarely seen except as a complication of more extensive paralyses, paralysis of the extensors of the spine in the lumbar region (the erector spinæ and its divisions, the sacro-lumbalis and longissimus dorsi) is the only one that has a practical interest. This is seen comparatively often in the muscular atrophy or pseudo-hypertrophy of children (*vide infra*), and causes a remarkably characteristic and easily recognizable picture. If we make the little patient stand up, the peculiar carriage of the body strikes us at once. The lumbar vertebræ are arched forward in lordosis, the belly is very prominent, and the upper part of the body is bent backward. The trunk is balanced on the hips and the gait is waddling. The paralysis of the erectors appears most plainly if the child has stooped to get any object and tries to straighten up again. He can bring the upper part of his body into the erect posture only by supporting himself with his hands on his knees, and slowly climbing up his thighs.

6. Paralyses in the Region of the Upper Extremity.

Paralysis of the Deltoid Muscle (*axillary* [*circumflex* *] *nerve*).—Deltoid paralysis occurs either as one symptom of complicated paralyses arising from the brachial plexus, or as an isolated traumatic and rheumatic paralysis; that is, neuritic, beginning with pains in the region of the shoulder. It may be recognized by the impossibility of raising the upper arm at all. We can easily distinguish it from an anchylosis of the shoulder-joint by passive motion. If the paralysis lasts a long time there is a very marked atrophy of the muscle, and there is the electrical reaction of degeneration in it. Paralysis of the teres minor, which is also innervated by the axillary, can not be diagnosticated with certainty.

Paralysis of the Biceps and Brachialis Anticus (*musculo-cutaneous nerve*) is only exceptionally isolated, but is quite often seen combined with other paralyses. The forearm, when in supination, can not be flexed, but in pronation the supinator longus it can still display its action of flexion. The action of supination by the biceps, which it exerts when the forearm is flexed, is also absent. We sometimes see at the same time a disturbance of sensibility on the radial side of the forearm from an affection of a cutaneous branch of the musculo-cutaneous nerve.

Radial [Musculo-spiral] Paralysis.—The anatomical course of the radial nerve causes pressure paralysis of this nerve to be among the commonest peripheral paralyses. It is seen especially when the nerve is pressed against the humerus during sleep by the body or head lying on it, in drunkenness, sleeping with the arm hanging over the arm of a chair, etc. The paralysis is usually noticed immediately on waking. Other traumatic influences, direct injuries of the nerve, compression in dislocation of the shoulder, in fractures of the humerus, in pressure from crutches, in bandaging the arm, etc., are also frequent causes of radial paralysis. Exposure to cold, rheumatic radial paralysis, plays a very subordinate part. For lead paralysis, which is localized chiefly in the distribution of the radial, *vide infra.*

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* We have followed Henle's nomenclature for the peripheral nerves.—Trans.

The radial innervates the triceps and the muscles on the extensor side of the forearm. Paralysis of the triceps is present only in the cases where the point of lesion is situated quite high up, as in crutch paralyses, dislocation paralyses, plexus paralyses, etc., but it is absent, or at least it is only faintly manifest, in most of the ordinary pressure paralyses, in which the place where the radial turns about the humerus is the point of compression. Triceps paralysis is readily recognized by the impossibility of extending the forearm, but we must always make the experiment with the upper arm raised, so as to exclude the action of gravity in extending the forearm.

Paralysis of the muscles on the extensor side of the forearm may at once be recognized, since the hand hangs down relaxed in a flexed position (see Fig. 77). Any dorsal extension by the extensor carpi ulnaris and the extensor carpi radialis longus and brevis is impossible, and the lateral movements of the hand in abduction and adduction are rendered difficult. The fingers are flexed, the first phalanx can not be extended by the extensor communis digitorum, extensor indicis, and extensor minimi digiti; but if the first phalanges be extended passively and supported, the extension of the terminal phalanges is perfectly normal, from the action of the interossei which are supplied by the ulnar nerve. The thumb is flexed and adducted, and can neither be abducted nor extended actively (extensores ossis metacarpi et primi et secundi internodii pollicis). If the forearm be extended and pronated it can not be supinated (supinator brevis), but the flexed forearm can be supinated by the biceps. Flexion of the forearm in supination, which is done by the biceps and brachialis anticus, is retained, but flexion when half pronated (" middle position ") is weakened, from the paralysis of the supinator longus. If we have the patient make short and rapid movements of flexion of the forearm in this position we do not see the characteristic normal prominence of the tense supinator longus. The very characteristic prominence of this muscle is also wanting if the patient tries to hold his pronated and semi-flexed forearm firm against forcible attempts at extension.

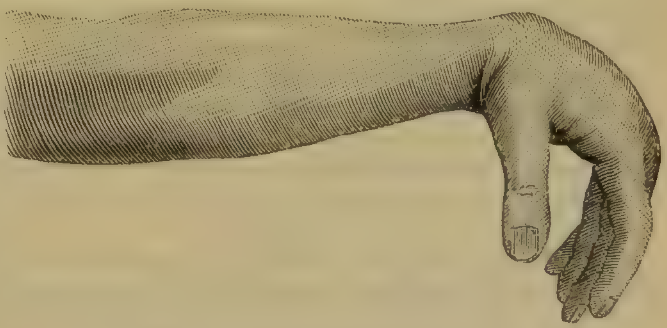

FIG. 77.—Position of the hand in paralysis of the radial nerve. (From SEELIGMÜLLER.)

The functional disturbance of the hand in radial paralysis is very considerable. The action of the flexors is also weakened, since their points of insertion are approximated, on account of the constant drooping of the hand. We often see, too, sensory disturbances as well as motor in the distribution of the radial, but these are usually slight. The chief seat is on the radial half of the back of the hand and the dorsal surface of the thumb, and index and middle fingers (compare Fig. 61). The electrical excitability of the paralyzed parts corresponds to the laws that generally obtain. At the onset, and in mild cases, it is normal; at a later period, in severe cases, there are pronounced atrophy and reaction of degeneration. It is worthy of note that in all forms of radial paralysis, especially in lead paralysis, we very often find a peculiar chronic thickening and swelling of the tendons on the back of the hand, the chief cause of which is probably the mechanical tension of the tendons.

Ulnar Paralysis.—Except from the frequent implication of the muscles supplied by the ulnar nerve in extensive paralyses and atrophies, especially in progressive muscular atrophy, ulnar paralysis arises chiefly from traumatic influences, pressure, wounds, fractures of the humerus, dislocations of the shoulder-joint, etc. Neuritic paralyses are more rare.

Flexion of the hand, and especially its lateral movement to the ulnar side, is disturbed (flexor carpi ulnaris). Flexion of the last three fingers is imperfect from partial paresis of the flexor profundus digitorum, and the little finger can not be moved at all (hypothenar muscles). Paralysis of the interossei is most striking, by which flexion of the primary phalanges and extension of the terminal phalanges of the fingers becomes impossible. Spreading the fingers, and still more bringing them together again, is also much impaired (interossei, lumbricales). The thumb can not be firmly adducted against the metacarpal bone of the index-finger (adductor pollicis).

In almost all old cases of ulnar paralysis a very characteristic position of the hand is developed, besides the muscular atrophy which is especially noticeable in the interosseal furrows of the back of the hand. By the contraction of the muscles antagonistic to the paralyzed interossei (extensor and flexor communis digitorum), the first phalanges are put in marked dorsal extension, but the terminal phalanges are completely flexed, so that the hand assumes an actual clawing position—"claw-like hand," *main en griffe* (see Fig. 78).

The disturbance of sensibility, if it be present at all, affects the volar surface of the last two fingers, the dorsal surface of the last three fingers, and a portion of the back of the hand (see Figs. 59 and 60).

Median Paralysis.—Median paralysis is seen chiefly as a traumatic paralysis. It is often, too, one symptom of more extensive paralyses, in progressive muscular atrophy, etc.

The disturbances of motion are very striking. Pronation of the forearm is almost wholly abolished (pronator radii teres and quadratus). The hand can be flexed only toward the ulnar side by the flexor carpi ulnaris (paralysis of the flexor carpi radialis). The terminal phalanges of the fingers can no longer be flexed (flexor sublimis digitorum and a part of the profundus), but flexion of the primary phalanges is normal by means of the interossei. The patient can grasp an object only by the last three fingers, which can still be partly flexed by the flexor profundus digitorum (ulnar nerve). The thumb can no longer be flexed or opposed (flexor longus pollicis et brevis, opponens), and usually lies on the hand.

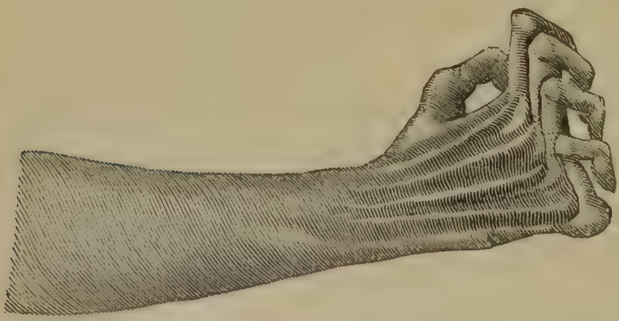

FIG. 78.—Claw-shaped hand, *main en griffe*. (From DUCHENNE.)

If there is any disturbance of sensibility, it is found on the volar surface of the thumb and the two adjacent fingers, and also on the dorsal surface of the terminal and middle phalanges of the index and middle fingers, and the radial side of the ring-finger (see Figs. 60 and 61). We quite frequently see in severe cases trophic disturbances, vesicles on the fingers, a shining atrophic skin, and changes in the nails.

Combined Paralyses of the Arm.—Combined paralyses, in which the affected muscles belong to the distribution of several nerves, occur in various forms, especially as a result of injuries which affect the brachial plexus in the neck—plexus paralyses. To this class belong also a great part of the paralyses following dislocation of the humerus—dislocation paralyses.

A combined plexus paralysis, first described by Erb, and since then repeatedly observed, deserves special mention. In this the deltoid, biceps, brachialis anticus, and supinator longus (muscles whose nerves all rise from the roots of the fifth and sixth cervical nerves) are paralyzed at the same time. The arm hangs down relaxed, and can not be raised at all, the forearm can not be flexed at all, but the hand and fingers have their normal mobility. The cause of the paralysis must have its seat at the point where the nerve-fibers for the muscles mentioned lie near one another (see Fig. 68). Sometimes the infra-spinatus is also paralyzed, so that when the arm is rotated inward it can not be rotated outward.

This same combination of paralyzed muscles is found in a part of the delivery paralyses first described by Duchenne. These are sometimes seen in infants after hard labor, and are the result of traumatic injuries of the brachial plexus in turning, in the Prague method, in extracting the child by the shoulders, etc.

In some cases of complicated paralyses of the brachial plexus, which are usually traumatic (Seeligmüller and others), co-existing symptoms on the part of the sympathetic have been observed, consisting of contraction of the pupil, narrowing of the opening of the lids, and a retraction of the eyeball on the paralyzed side. These symptoms, pointing to a paralysis of sympathetic nerves (*vide infra*, page 591), probably depend, as follows from clinical and experimental investigations (Klumpke), always upon a lesion of the ramus communicans of the first dorsal nerve. Vaso-motor symptoms in the face are usually absent, but we sometimes find a peculiar flattening of the cheeks, which has not yet been correctly explained.

General Prognosis and Treatment of the Peripheral Paralyses of the Upper Extremity.—In the prognosis of the peripheral paralyses of the arm the same points hold good that have been spoken of in the prognosis of facial paralysis. In this, too, there are mild and severe cases, the latter having complete reaction of degeneration, and a course that lasts at least several months before recovery. A number of traumatic paralyses can be cured only up to a certain point, or they may be even entirely incurable.

The treatment can fulfill a causal indication only in comparatively rare cases, when we can succeed in removing by operation any compressing tumors, cicatrices, splinters of bone, formations of callus, etc.

In other cases the electrical treatment of paralyses promises the best success. We use the galvanic current chiefly, although we generally employ the faradic current at the same time. In regard to the method of application, we may employ the stabile action of the constant current on the very point of the lesion, especially in fresh cases, but the chief method is the electrical irritation of the paralyzed nerves and muscles. We treat the nerve above the point of the lesion in order to act in some degree against the hindrance to conduction from above and to overcome it. The muscles are irritated by galvanism by stroking the cathode over the separate paralyzed muscles. If there is reaction of degeneration, with anodic contractions predominating or exclusively present, we use the anode for the testing pole. The other pole is placed on the back of the neck or on the seat of the lesion. Faradization of the muscles may also be of service, especially if the muscles react to faradism; but, even if this is not the case, the sensory faradic irritation has perhaps a favorable influence, since it produces a reflex irritation of the motor nerves. The single sittings should last five or ten minutes, and should take place daily or three or four times a week. The fresher the paralysis, the more favorable, comparatively, is the prognosis, but even in old and severe cases we may sometimes obtain noticeable results by patience and perseverance. The treatment must in such cases be kept up for months, and even longer, with occasional interruptions.

Embrocations with spirits and with similar substances must often be prescribed in practice, but they act favorably only when associated with methodical massage of the paralyzed muscles. We sometimes see a certain advantage, too, from local warm bathing, or from the use of the baths in Teplitz, Wiesbaden, Wildbad, etc.

7. PARALYSES OF THE DIAPHRAGM.

Isolated paralysis of the diaphragm occurs but rarely, in wounds of the phrenic nerve in the neck, as a "rheumatic" paralysis, and finally in hysteria. Muscular paresis of the diaphragm seems to develop sometimes as a result of inflammation of the serous layer of the diaphragm. The paralysis of the diaphragm which comes on as one symptom in more extensive paralyses, is more frequent and practically more important. In diseases of the upper cervical cord, in ascending myelitis, in progressive muscular atrophy, in multiple neuritis, etc., the development of paralysis of the diaphragm is the cause of the rapidly fatal termination which follows the appearance of disturbance of respiration.

The symptoms of paralysis of the diaphragm are readily recognized, especially in the ordinary bilateral affection. We detect the modification of the respiratory movements at the first glance. While we are struck by the marked upper thoracic respiration, which becomes very labored on the slightest cause, the visible and palpable protrusion of the epigastrium on inspiration is entirely absent. Instead of this there is usually an inspiratory retraction in the epigastric region. The respiration is but little accelerated in simple paralysis of the diaphragm as long as the patient is perfectly quiet; but in other cases the development of a severe bronchitis, from the defective respiration in the lower lobes of the lungs, causes constant dyspnœa. The cause of the bronchitis may be found in the fact that the action of abdominal pressure is very much diminished in the constant high position of the diaphragm, which may be made out by percussion, and consequently the cough and the expectoration of secretion are very imperfect.

The prognosis is favorable only in hysterical and rheumatic paralyses; otherwise it is usually very unfavorable. In regard to treatment, the only thing that can be tried is to excite the diaphragm from the phrenic in the neck by faradism or galvanism, while the other pole is placed on the region of the insertion of the diaphragm in the thorax. A transverse conduction of the constant current through the diaphragm, associated with changes of the current, may also have a favorable influence.

8. PARALYSES IN THE REGION OF THE LOWER EXTREMITY.

PARALYSIS OF THE CRURAL NERVE.—Crural paralysis is but rarely isolated. It is seen after injuries, after compression of the nerve by tumors of the pelvis or thigh, in disease of the vertebræ, psoas abscess, etc.

The symptoms are readily recognized. The thigh can not be flexed on the trunk, and the trunk can not be raised from the recumbent position (ilio-psoas muscle). The leg when flexed can not be extended (quadriceps extensor). Walking and standing are very difficult or almost impossible. Paralysis of the sartorius and pectineus causes no special symptoms. If there is any disturbance of sensibility it is found in the lower half of the anterior surface of the thigh and on the inner side of the leg down to the great toe (saphenous nerve, see Figs. 62 and 63).

PARALYSIS OF THE OBTURATOR NERVE is very rarely seen as an isolated phenomenon. The chief symptom is the defective adduction of the thigh (the adductor magnus, longus, and brevis, and the gracilis), and the impossibility of crossing one leg over the other. Rotation of the thigh outward is also disturbed (obturator externus). Some disturbance of sensibility may be found on the inner side of the thigh.

Paralyses in the Distribution of the Gluteal Nerves are not uncommon. We have seen them in progressive muscular atrophy and in multiple neuritis. Paralysis of the glutæus maximus is rendered noticeable by the fact that this muscle extends the thigh on the pelvis. It is therefore especially called into play in going up-stairs, climbing mountains, and rising from a sitting position. All these movements are rendered nearly impossible when the muscles are paralyzed. The glutæus medius and glutæus minimus are abductors of the thigh, and also fix the thigh on the pelvis. If they are paralyzed the patient has a very characteristic waddling gait. On account of the predominance of the adductors the feet are then advanced near each other or even one over the other. Rotation of the thigh inward is also due in part to fibers of the glutæus medius, but chiefly to the obturator internus.

Paralyses in the Region of the Sciatic are quite frequently seen. They come from traumatic lesions, from compression of the separate nerve-branches in diseases of the vertebræ, in pelvic tumors, in hard labors, rarely from rheumatic influences, sciatic neuritis etc.

PARALYSIS OF THE PERONEAL NERVE, which is also frequently isolated, may at once be recognized by the flaccid drooping of the foot. On walking, this becomes very marked, and the tip of the foot often sticks to the floor. The patient, therefore, has to raise the thigh higher, and to put the foot down awkwardly, toe first. Dorsal extension of the foot (tibialis anticus) and of the toes (extensor communis digitorum longus and extensor hallucis longus), and also abduction of the foot and raising its outer border (the peronei), are almost impossible. In old cases a permanent toe-drop (talipes equinus or varo-equinus) develops, usually as a result of secondary contracture of the muscles of the calf.

PARALYSIS OF THE TIBIAL NERVE makes plantar flexion of the foot impossible (gastrocnemius and soleus). The patient can no longer rise on his toes. Adduction of the foot (tibialis posticus) and plantar flexion of the toes (flexor communis digitorum and flexor hallucis longus) are also abolished. As a result of secondary contractures, talipes calcaneus sometimes develops, and also a claw-like position of the toes, with dorsal extension of the first and plantar flexion of the last phalanges from paralysis of the interossei.

In PARALYSES OF THE TRUNK OF THE SCIATIC there is added to the symptoms mentioned the inability to flex the leg backward on the thigh (to be tested when the patient is lying on his side or standing), which is due to a paralysis of the biceps, semi-membranosus, and semi-tendinosus. In unilateral paralysis of the sciatic, walking is still possible, since the leg is fixed at the knee by the quadriceps extensor and is rigid like a wooden leg.

The distribution of the disturbance of sensibility on the posterior surface of the whole leg is given in Fig 63. Vaso-motor and trophic disturbances, cyanosis and coldness of the skin, and atrophy of the muscles, are often present.

Treatment follows the same rules as are given for the management of peripheral paralyses in the upper extremity.

9. TOXIC PARALYSES.

Lead Paralysis.—Of all the toxic paralyses, that from lead poisoning is practically the most important. It is a common symptom of chronic lead poisoning, and is seen chiefly in those people whose occupation gives rise to a long-continued taking of small amounts of lead into the system, especially, therefore, in type-setters, type-cutters, and type-founders; in artists and house-painters, from lead colors; in potters, from lead glaze, etc.

As to the special anatomical causes of lead paralysis, we have not yet reached a complete harmony in our theories. While some seek the starting-point of the paralysis in the muscles themselves, most authors at present assume, as a cause of the paralysis, an affection of the nervous system excited by the toxic action of the lead. Since lead paralysis belongs to the genuine atrophic paralyses (*vide supra*), as we shall soon see, we have to do only with a disease of the anterior gray cornua in the cord, or with a degeneration of the peripheral motor nerves. The positive lesions found at present do not fully agree, but there can be scarcely a doubt, after the discoveries of Leyden, Zunker, and others, that, at least in most cases, the degenerative atrophy of the peripheral motor nerve-fibers is primary, and that the degenerative atrophy of muscles supplied by the nerves follows secondarily in the ordinary way. In many cases, however, there is perhaps, besides the peripheral degeneration, an affection of the cord, especially in the anterior gray cornua, caused by the toxic action of the lead.

Lead paralysis shows an extremely typical localization in the great majority of cases, and it affects by far the most frequently a part of the radial distribution. A paralysis of the extensor communis digitorum comes on rapidly or slowly. Extension of the primary phalanx of the middle and ring, and later of the index and little fingers, becomes impossible, but the extension of the terminal phalanges by the interossei remains normal. There often follows later a paralysis of the extensor longus pollicis and the extensor brevis, and of the abductor pollicis and the extensors of the wrist, while the supinator longus and the triceps almost always remain free in a remarkable way. In much rarer cases lead paralysis affects the deltoid, biceps, brachialis anticus, and supinators. Paralysis of the lower extremities is also very rare.

Lead paralysis is usually bilateral. In all severe cases a pronounced atrophy and electrical reaction of degeneration develop in the paralyzed muscles. It is an interesting point that the latter may sometimes be made out in muscles which can be perfectly well moved by the will (see page 553). The sensibility is almost invariably perfectly normal, so that the sensory nerves are manifestly unaffected by the lead.

Lead paralysis permits a favorable prognosis in the cases where the patient can be removed from the injurious influence of the poison. Recovery takes place after several weeks, or in severe cases after some months. Relapses and complications with other morbid results of chronic lead poisoning, however, are, of course, frequent.

The treatment is the same as in all other peripheral paralyses. Electricity is first to be considered. Local sulphur baths and the internal use of iodide of potassium are also recommended.

[It is well to mention here that chronic lead poisoning may give rise to symptoms closely resembling those of almost every chronic disease of the nervous system. It is often so difficult to detect the source of the lead that it is well in all obscure nervous diseases to test the urine for the metal after iodide of potassium has been administered in five- to ten-grain doses thrice daily for a week. For the precautions to be observed in carrying out this test the reader is referred to works on medical chemistry. If large doses of the iodide are administered there is danger lest the lead be liberated too rapidly and cerebral symptoms supervene. J. J. Putnam has shown, however, that lead is often found in the urine of persons in apparent good health. Lead may also cause simple anæmia, and it is occasionally found in the urine of patients with degenerative diseases of the spinal cord, especially combined sclerosis, ataxic paraplegia, and chronic myelitis.]

Arsenical Paralysis.—Arsenical paralysis is much rarer than that from lead. In distinction from that it comes on especially after acute poisoning with arsenic, and usually, though not always, it follows immediately the other symptoms of poisoning. The localization of the paralysis is not as typical as in lead paralysis. The paralysis is sometimes very extensive over the arms and legs, but it usually chiefly affects only the lower extremities. The paralyzed muscles rapidly atrophy. It has not yet been certainly decided whether reaction of degeneration occurs. The accompanying disturbances of sensibility are very characteristic, either anæsthesia, or especially paræsthesia and severe pains in the sacrum and legs. Trophic disturbances in the nails, hair, etc., have been repeatedly observed. Nothing certain is known as to the anatomical cause of arsenical paralysis, but the theory of its peripheral origin is most probable on account of the initial pains.

The course is usually favorable, sometimes rapid and sometimes lasting for months. The treatment is the same as in lead paralysis.

Copper paralysis, zinc paralysis, etc., are very rare, and therefore they will not be described fully here. The essentials in regard to alcoholic paralysis may be found in the chapter on neuritis (*vide infra*). We may mention here briefly that, after subcutaneous injections of ether on the extensor side of the forearm, paralysis of the extensor communis digitorum has been observed in a few cases.

CHAPTER III.

THE DIFFERENT FORMS OF LOCALIZED SPASM.

1. Spasm in the Motor Distribution of the Trigeminus.

Tonic spasm of the muscles of mastication is called trismus. As an independent disease it is very rare, but it often occurs as one symptom in complicated forms of spasm and other nervous affections, as in tetanus, in the epileptic attack, in hysteria, meningitis, etc. Both jaws are pressed firmly together, and we can feel through the cheeks the hard and tense masseters. In unilateral spasm of the pterygoids the lower jaw is pushed laterally in the opposite direction.

Clonic spasm of the muscles of mastication—masticatory facial spasm—consists of a paroxysmal and constant movement of the lower jaw, almost always in a vertical, but rarely in a horizontal direction. The single movements follow one another usually in a regular, rapid rhythm, and produce an audible chattering of the teeth. The mucous membrane of the mouth and the tongue is often injured.

The cause of these spasms can not always be established. Sometimes they seem to be of reflex origin, as in affections of the lower jaw, the teeth, or even of distant parts. We once saw a case which lasted for a year which was said to have arisen from a severe fright, and also a case of chronic spasm in the masseters and mylo-hyoids of hysterical origin in a ten-year-old boy.

The treatment, apart from the treatment of the primary affection, must aim at removing the cause of the disease, if there be one, such as decayed teeth. In other respects electricity is of value in many cases, applied either by passing the constant current through the muscles, or by faradizing them, or by using the wire brush.

Of internal remedies we may try narcotics, such as morphine, cannabis indica, bromide of potassium, atropine, arsenic, iodide of potassium, valerianate of zinc, etc.

Artificial feeding is of great importance, if the patient can not take food voluntarily from a persistent trismus. It is best to introduce a small œsophageal tube through the nose into the œsophagus. Rectal feeding is insufficient for a permanent method, but still it is sometimes of service. In some cases a successful attempt has been made to overcome the closure of the jaws gradually by pushing wooden wedges between the teeth.

2. Clonic Facial Spasm.

(*Mimetic Facial Spasm. Convulsive Tic.*)

We know little that is definite as to the ætiology of facial spasm, the most frequent and practically the most important form of spasm. We can rarely make out any cause for its origin. In some cases, perhaps, the disease is to be referred to a lesion of the trunk of the facial, from exposure to cold, aural affections, or disturbances at the base of the skull, or else to a reflex irritation of the nerve in trigeminal neuralgia, and also in diseases of the sexual organs, etc. It may be that many cases are not of peripheral but of central origin, from the facial center in the cerebral cortex. The disease may also appear after violent mental excitement. Finally, imitation and the habit of grimacing play a part in many cases, especially in children, that is not to be underestimated. Repeated observations have established the fact that the disposition to the disease is heightened by a general hereditary neuropathic taint.

The symptoms of convulsive tic consist of alternating, short, lightning-like contractions in almost all the muscles supplied by the facial. The disease is usually unilateral, often extending over the whole distribution of the facial, but sometimes confined only to individual parts, the partial facial spasm. Sometimes the contractions are almost constant, though varying in intensity, so that the patient involuntarily makes the strangest faces; but they often appear in separate attacks, which last but a short time, and are interrupted by completely free intervals. The attacks arise without any special cause, or they may be excited by talking, voluntary movements, sensory and mental impressions, etc. In some very severe cases the contractions also invade the neighboring territory—the muscles of mastication, the tongue, and the muscles of the neck. Voluntary motion in the muscles is perfectly normal, except for the disturbing influence of the spasmodic movements. All sensory disturbances are also wanting; there is neither anæsthesia nor pain.

A common partial form of facial spasm, which is entirely or almost entirely isolated, deserves special mention—blepharospasm, or spasm of the eyelids—that is, a tonic or clonic spasm in the orbicularis palpebrarum. The tonic form may be of reflex origin in affections of the eye, but sometimes it is excited from other regions of the trigeminus. It is, as a rule, bilateral, and may last for days or weeks, sometimes with occasional interruptions. The pressure points, which are found in this form, and which were first carefully described by Graefe, are very remarkable. They are usually found at the points of exit of the branches of the trigeminus, but sometimes on the vertebral column or on other parts of the body. By pressure on these points the spasm at once ceases, so that the lids "fly up as if by a spring." Clonic spasm of the lids, nictitating spasm, consists of a spasmodic winking and contraction of the eyes, which is sometimes almost constant. Here, too, we can often make out a reflex origin for the spasm, but frequently we can not find any cause.

Facial spasm, in its severe forms, is always a troublesome disease for the patient, and causes very much disturbance, especially in blepharospasm. The course is often very tedious. Sometimes there are long intervals, as we have seen during pregnancy, and then the spasm begins anew. In not very rare cases the affection becomes habitual and lasts for life.

Treatment is, therefore, usually a difficult and thankless task. The best results may be obtained where we can succeed in removing any reflex cause of the spasm, as in extracting decayed teeth, treating affections of the eye, or, in some cases, resection of the supra-orbital nerve. In applying electricity, our chief attention must be directed to any pressure points, to which we make a stabile application of the anode of the constant current. If there are no pressure points, we put the anode on the trunk of the facial and the different branches of the pes anserinus. In cases of reflex origin Berger obtained good results by applying the anode to the occiput, just under the protuberance, while the cathode was held in the hand—galvanization of the medulla. The single sitting should last five or ten minutes. The faradic current—a slowly "swelling current"*—has also been recommended. Of internal remedies we should first try bromide of potassium, and then arsenic, atropine, curare, or oxide of zinc. Their action is always very uncertain. A favorable result, however, has been obtained by nerve-stretching, in a part of the cases operated on, at least so far that the ensuing paralysis troubles the patient less than the constant twitching. When the paralysis ceases the twitchings usually begin again, but in some cases the benefit has been permanent. Finally, we must mention that the use of the red-hot iron has sometimes been followed by a decided improvement of the spasm in old cases. We employ cauterization along the cervical vertebræ, on the trunk of the nerve, or on any existing pressure points, by means of Paquelin's thermo-cautery.

[*Convulsive tic* (*Maladies des tics convulsifs*).—Under this term Gilles de la Tourette has described an affection allied to hysteria, which usually begins in young children, especially those with a neurotic heredity. It is characterized by spasmodic twitchings of the facial muscles, and sometimes of the other muscles of the body, and by explosive utterances. These utterances may be a repetition of some word which the child hears (echolalia), or the repetition of some profane or filthy word (coprolalia). Actions may also be imitated. Coprolalia is one of the most distinctive symptoms. Other mental symptoms, particularly certain forms of imperative conceptions, are occasionally observed. The prognosis is bad. The treatment is largely the same as that of hysteria (*q. v.*).—K.]

3. Spasm in the Region of the Hypoglossal Nerve. Lingual Spasm.

Although the tongue is often implicated in complicated forms of spasm, such as hysterical or epileptic spasms, isolated spasms in it are very rarely seen. They occur, however, either in a clonic or a tonic form, and then they cause a marked disturbance of speech, or even of respiration, if the spasm draw the tongue backward. In the latter case it may be necessary to use inhalations of chloroform, and to draw the tongue forward by force.

4. Spasms in the Muscles of the Neck.

Tonic and clonic spasms in the region of the muscles of the neck are not very frequent, but they appear in very various forms, and are at times a very severe and persistent affection. We can usually discover nothing definite as to the ætiology of this condition. Only in a few cases can we make out coarse anatomical

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[* A current that begins weak, and is gradually made stronger and then weaker.—Trans.]

disease of the nervous system or of the cervical vertebræ, rheumatic or other evil factors, reflex influences, etc. Although the spasms in the different muscular regions are often combined with one another, we can still distinguish the chief separate forms.

SPASMS IN THE DISTRIBUTION OF THE ACCESSORY (CIRCUMFLEX) NERVE.—In clonic spasm of the accessory there are paroxysmal twitchings of the head, which may attain great severity. Where there is a predominating unilateral spasm of the sterno-mastoid, the head is turned to the opposite side at every contraction of this muscle, and the chin is also somewhat raised. In unilateral spasm of the trapezius the head is drawn backward toward the affected side against the shoulder. In bilateral and combined spasm of these muscles we see severe shaking and nodding movements of the head—the so-called nodding spasms, "salaam convulsions"—which have been observed chiefly in children. They may also be excited in like manner by contractions of other muscles of the neck. In tonic spasm of the accessory the head is constantly fixed in the abnormal position described above, and it can not be brought back passively to its normal position at all, or only incompletely. The oblique position of the head in unilateral tonic spasm of the sterno-mastoid is called spastic torticollis (*caput obstipum spasticum*), or rheumatic torticollis, if exposure to cold be regarded as the cause.

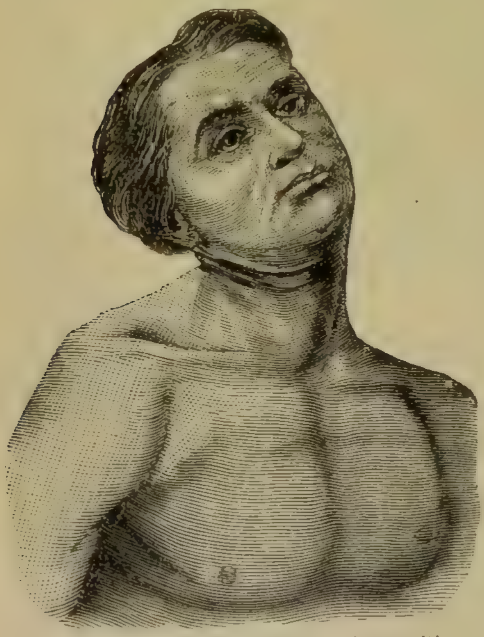
FIG. 79.—Spasm of the right splenius capitis. (From DUCHENNE.)

Tonic and clonic spasm of the splenius (Fig. 79) is also isolated, or combined with spasm of the accessory. In this the head is drawn backward and toward the affected side, and thus we can feel the muscular swelling protruding to the outside of the cervical portion of the trapezius.

Spasm of the obliquus capitis is probably the cause of the so-called rotatory tic, in which there are genuine spasmodic rotatory movements of the head. The recti capitis antici and postici are perhaps implicated in many cases of nodding spasm.

The prognosis of the forms of spasm described is usually doubtful. There are many mild "rheumatic" cases which recover in a short time, but, on the other hand, many of these forms of spasm develop into a chronic affection. In these cases any steady occupation, such as reading or writing, is rendered almost impossible by the constantly recurring spasmodic lateral and rotatory movements of the head. Any excitement, a feeling that he is noticed, etc., increase the patient's spasms, while they become milder when he is completely at ease. Many cases of combined tonic-clonic spasm of the muscles of the neck form a very severe affection, which may last for years or for life, which is extremely distressing and painful to the patient, and which may also reduce the strength and the nutrition to the utmost degree.

Treatment.—In some cases electricity has brought about recovery, or at least improvement. The method of treatment consists in the application of the anode to the affected nerves and muscles, or in the use of a swelling faradic current, or of the faradic brush to the skin over the affected muscles. Very often we have to change the method, and we must try to find out, by testing, the most efficient way to employ electricity. Of other remedies, narcotics, such as subcutaneous injections of morphine, are indispensable in severe cases. We may also try bromide of potassium, antipyrine, valerianate of zinc, arsenic, and other nervines. In severe cases we resort to the use of the red-hot iron to the back of the neck. Other observers, and we ourselves, have seen good results from it, but of course benefit does not always follow. We may also advise nerve-stretching, but it is of doubtful advantage. Finally, it must be mentioned, that we may give great relief to many patients by suitably applied mechanical supports, the more so because in some cases the spasms occur only when the head is kept free, but if they lean their heads on anything, as in lying or sitting, the spasms cease at once.

5. Spasms in the Muscles of the Shoulder and Arm.

Clonic spasms in the upper extremity are probably usually of central origin. They are rarely isolated, as in the pectoralis major, but are more frequently combined with other forms of spasm and other nervous symptoms. They sometimes seem to be of reflex origin, as in the clonic spasms associated with brachial neuralgia, and also the spasms sometimes seen in amputation-stumps.

Isolated tonic spasms, in single muscles, or groups of muscles, in the upper extremity, have been repeatedly observed. Tonic spasm of the rhomboidei causes an oblique position of the scapula, so that its inner border runs obliquely upward and outward from below and inward. This makes it hard to raise the arm above the horizontal line, as in serratus paralysis, but the separation of the scapula from the chest-wall, which is so very characteristic of the latter, is wanting. Tonic spasm in the levator anguli scapulæ is rare, except in connection with spasm of the rhomboidei or trapezius. In it the shoulder is raised and the head is bent a little to the affected side. Isolated tonic spasms in the pectoralis major, latissimus dorsi, deltoid, etc., are, on the whole, easy to recognize, but they are only of very rare occurrence. Tonic flexor spasms of the hand and fingers are more common. We have ourselves seen several such cases, which lasted for months. In one case the spasm could at once be relieved by placing the anode of a moderately strong galvanic current on the median nerve. In another case the flexor spasm of the fingers had followed a mild acute inflammation of the wrist-joint.

The special causes of all these spasms are still wholly unknown to us. Prognosis and treatment follow the same general rules which are given for other forms of spasm. Most is to be expected from electricity: the stabile action of the anode, the faradic brush, or faradization of the antagonistic muscles.

6. Spasms in the Muscles of the Lower Extremity.

Clonic spasms in the muscles of the lower extremity are always, with rare exceptions, a symptom of spinal or cerebral disease. Of the tonic spasms the most frequent and the best known are the painful spasms in the calves, or cramps, which are apt to come on after great muscular exertion, mountain-climbing, or dancing. Many persons have an especially marked predisposition to such cramps, which readily come on, especially after making certain movements or holding the foot in a certain position. Similar painful cramps sometimes come in other muscles besides the calves, as in the abductor hallucis, etc. Other tonic spasms in the muscles of the lower extremity are rare, but individual cases of isolated tonic spasm in the abductors, in the ilio-psoas, in the muscles of the calves, etc., have been observed. More extensive tonic contractures of the muscles of the leg are not very rare in hysteria, especially in the hysteria of children.

Saltatory Reflex Spasm.—In this place we may mention a peculiar form of spasm, to which Bamberger has given the name of "saltatory reflex spasm."

This shows itself in the muscles of the lower extremities, never when the patient is quiet in bed, but only when he tries to stand or to walk. As soon as the soles of the feet touch the floor, such vigorous contractions are set up in the muscles of the legs as to force the patient to keep up a constant hopping, jumping, or tripping. The heels are also usually raised spasmodically, and in many cases the patient would certainly fall if he were not supported. In the pure forms of saltatory spasm, on physical examination we can usually make out nothing but an extraordinary increase of the reflexes, especially of the tendon reflexes, but in some cases there may also be other nervous symptoms. In general, it seems that saltatory reflex spasm is not to be regarded as a special disease, but as a peculiar symptom, which may arise in different neuroses in consequence of a very decided exaggeration of the reflexes. Many cases especially seem to us to be hysterical.

[Certain similar affections exist as endemic neuroses. In the "jumpers" of Maine the spasms may be produced by any excitement. In other forms the victims are compelled to imitate everything they see or hear. The curious affections observed in Siberia, Java, and elsewhere, known as latah or emeryaki (myriachit), are allied to this trouble. Alcoholism, cold, and privation seem to be important ætiological factors, and they probably have some relation to hysteria.—K.]

Arthrogryposis.—As an appendix we would here consider briefly a remarkable disease, the so-called arthrogryposis, which occurs chiefly in children in the first years of life, and consists of persistent tonic spasms and contractures of one or often of all four extremities. The disease usually develops quite acutely, and may run its course with fever and rather severe general symptoms. The legs are found either in a position of rigid extension, or they are drawn up spasmodically to the body, and can not be extended passively even with violence. The arms are flexed, and the hands and fingers are also fixed in some position of contracture. The milder cases may recover after a few weeks, but we have also seen two cases which ended fatally, in which the autopsy gave a perfectly negative result. The nature of this quite rare affection is still wholly unknown to us. In regard to treatment, prolonged warm baths are especially to be recommended.

[**Paramyoclonus multiplex.**—Under this term Friedreich has described a peculiar affection consisting of clonic bilateral spasms of several muscles of a physiological group. Hence the movements seem to have a purposive character. The spasms are rapidly repeated from fifty to one hundred and eighty times a minute, and occur in paroxysms. The muscles chiefly affected are those attached at one or both ends to the trunk. Fascicular tremors of the affected muscles are observed in the intervals. The reflexes are exaggerated, and the attacks may be brought on by irritation of the skin or tendons. The nature of the affection is unknown. The prognosis is favorable. The most effective treatment has been the application of strong galvanic currents to the spine and neck.

Electrical Chorea.—This term was first applied to an affection met with chiefly in northern Italy. It consists of sudden muscular contractions, resembling those caused by electrical stimulation. With these are associated progressive paralysis and muscular wasting. Many of the cases terminate fatally. Similar sudden contractions are seen in ordinary chorea, hysterical spasmodic conditions, etc., and hence the term has been frequently applied to these. Compare the chapter on hysteria.—K.]

The form of tonic spasm known as tetany will be described in a special chapter.

7. Spasms in the Respiratory Muscles.

Tonic spasm of the diaphragm has been seen in a few rare cases. The lower part of the thorax is much expanded, the epigastrium is protruded, and the respiration, which shows intense dyspnœa, can be performed only by the upper part of the thorax. The depression and immobility of the diaphragm can be made out on percussion. Many patients have a severe pain in the region of the diaphragm. The condition is not without danger and demands instant interference: inhalations of chloroform, subcutaneous injections of morphine, a warm bath with a cool shower-bath after it, faradization of the skin in the region of the diaphragm, galvanization of the phrenics, etc.

Clonic Spasm of the Diaphragm.—Singultus.—The well-known "hiccough" or "sob," due to a sudden spasmodic contraction of the diaphragm, is in its mild forms a very frequent condition, which soon passes off; but it sometimes increases to a persistent, obstinate, and very troublesome affection, which may last for weeks or months. It sometimes comes on after mental excitement, and is a not very rare symptom of hysteria. Persistent hiccough may also be excited reflexly in affections of the stomach, intestines, peritoneum, etc. In some cases the hiccough depends upon a direct lesion of the phrenic nerve, as we have seen in one case of tubercular mediastino-pericarditis. We have also seen hiccough lasting for hours in cerebral apoplexy, and also in chronic myelitis extending to the cervical cord.

In the milder cases hiccough soon passes off without special treatment. Holding the breath, pressure on the closed glottis, blows on the back, etc., are procedures generally known by the laity, and often used to suppress hiccough. In more severe cases we must try narcotics, opium, cannabis indica, or inhalations of chloroform. The faradic brush to the region of the diaphragm, or the direct action of electricity on the phrenic nerve, is sometimes of advantage. In hysterical hiccough we may obtain very rapid results in this way, or sometimes by one of the different nervines, valerian, zinc, atropine, or Fowler's solution.

Complicated respiratory spasms, either in the form of spasmodically accelerated and forced breathing, or spasms combined with all sorts of other symptoms, with many gurgling noises, eructations, etc., are almost exclusively confined to hysteria. We have ourselves counted in one such case over two hundred respirations a minute! The best remedy against most of these forms of spasm, and one which often acts instantly, is a cool bath with energetic cold shower-baths. The yawning spasm (*chasmus*, *oscedo*), sneezing spasm (*sternutatio convulsiva*, *ptarmus*), laughing and weeping spasms, coughing spasm, etc., also belong to the respiratory spasms. We once saw a very remarkable example of the latter in a boy ten years old. A peculiar, reflex, hollow, barking cough came on, either spontaneously or on pinching any part of the skin. The affection lasted for some weeks, and then disappeared quite suddenly.

CHAPTER IV.

WRITER'S CRAMP AND ALLIED PROFESSIONAL NEUROSES.

WRITER'S cramp (*graphospasm*, *mogigraphia*) is the commonest form of a whole class of peculiar disturbances of motion, to which Benedikt gave the appropriate name of "professional neuroses of co-ordination." Their characteristic feature is that the disturbance in a certain group of muscles appears only when these muscles come into harmonious action, in quite a definite and usually a delicate and complicated occupation. Although the persons who suffer from writer's cramp can move and use the muscles of their right arm and hand for ordinary purposes in a perfectly normal manner, these same muscles at once refuse their service when the patient begins to write. In this case the disturbance can not lie in the innervation of the individual muscles themselves, but it must be referred to the form of their associated action—that is, it must be a disturbance of co-ordination; but the details of this disturbance are still entirely unknown to us, nor do we know in what part of the nervous system we are to look for the seat of the disease. Over-exertion in writing plays the most important part as an ætiological factor. Writer's cramp is therefore seen chiefly, but of course not exclusively, in those persons whose occupation is connected with continuous writing, especially in secretaries, merchants, bureau officials, etc. A general nervous predisposition seems also to increase the tendency to writer's cramp. Attention has also been called to the fact that bad pens, such as hard steel pens, bad methods of holding the pen in writing, etc., may favor the development of writer's cramp.

Symptoms.—The essential symptom of writer's cramp is the appearance of certain disturbances at every attempt to write, which render writing very difficult or entirely impossible. The affection usually begins gradually, but increases quite rapidly. For the precise characterization of the disturbance Benedikt has distinguished three forms of writer's cramp, but they run into one another in various ways. The spastic form is the most frequent. The patient scarcely begins to write when contractions or tonic spasms come on in the different fingers, so that the pen can no longer be held firmly, or it makes abnormal irregular movements, or it is firmly pressed into the paper, etc. We very often see a tonic spasm of the pronators of the forearm set in on each attempt to write. Writing is wholly impossible, or it is done only with the greatest exertion, and the characters are also utterly distorted, unequal, and mingled with false strokes and blots. In the paralytic form* the disturbance in writing is chiefly a rapidly arising tired feeling, like paralysis, in the right arm, which is often associated with painful sensations. Finally, in the tremulous form of writer's cramp there is such a marked tremor in the right hand at every attempt to write that the letters are completely illegible. We have seen several such cases in children, which we have regarded as distinctly hysterical.

As has already been said, motility in every other respect is perfectly normal, but sometimes analogous symptoms also appear in the same patient in many other fine employments, such as sewing, piano-playing, etc. The sensibility is usually perfectly normal, except for the muscular pains already mentioned, and a frequent feeling of numbness in the forearm and fingers. A few painful pressure-points have sometimes been found on the cervical and dorsal vertebræ. We should also examine the peripheral nerves, since painful thickenings are sometimes to be detected in them, which possibly stand in a causal relation to the disease. If we are dealing with people who are generally neurotic, they often complain at the same time of headaches, mental uneasiness, and general weakness.

The diagnosis of writer's cramp is almost always easy. We must guard against confusing it with other nervous diseases, such as chorea, paralysis agitans, multiple sclerosis, beginning muscular atrophy, or agraphia, which, of course, under some circumstances may lead to disturbances in writing.

The prognosis is always to be given with reserve. Complete recoveries without doubt do occur, but many cases are extremely obstinate and others are incurable. Relapses are also very common even after improvement has set in. Many patients are obliged to choose another calling in consequence of their trouble.

The treatment begins first with the command to give up writing entirely for several weeks or months. If this command can be obeyed, the mere rest may be of service in mild incipient cases. Certain contrivances for writing, which the patient can best test himself, are often of advantage, such as sticking the pen-holder through a cork, using a large pen-holder, a change in the way of holding the pen and in the position of the arm, etc. Nussbaum has lately invented a kind of bracelet to which the pen-holder is fastened, and which is held firmly by the outspread fingers. Learning to write with the left hand, which is often tried by patients, usually leads to no good result, since the cramp very soon appears in the left hand also with remarkable rapidity.

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*It is particularly illogical to class this disturbance with writer's cramp, since we can not speak of the "paralytic" form of a "cramp."

Of the special methods of treating writer's cramp the application of galvanism deserves the first mention. Avoiding all strong currents and variations of the current, we apply the stabile anode to the brachial plexus, and also to the different nerves (especially if they be sensitive to pressure) and the affected muscles, for five or ten minutes. The cathode is placed in the vicinity of the cervical vertebræ. If painful points are found, they receive special treatment. We may also use galvanism, as an experiment, through the head. Of late, massage and methodical gymnastics have shown still more favorable results than electrical treatment, but the use of them demands special technical skill, and therefore they have so far obtained excellent results chiefly in the hands of certain specialists. We can very rarely promise success from internal remedies such as subcutaneous injections of strychnine, atropine, etc., but those methods of treatment often act favorably which aid the general strength of the nervous system, such as cold-water cures, sea-bathing, and residence at a mountain resort.

As an appendix we will mention here some other professional neuroses that are occasionally seen. They are the piano-player's cramp, which is seen especially in young conservatory pupils, violin- and 'cello-player's cramp, telegrapher's cramp, tailor's cramp, milker's cramp, the peculiar disturbances of innervation in the hands which often occur in cigar-rollers, etc. In the lower extremities there seems to be an analogous affection in ballet-dancers, and also in sewing-machine girls, turners, etc. We have seen a professional cramp in the tongue in a clarionet-player. The special points in the symptomatology and treatment of all these forms of cramp are in large part analogous to the conditions described in writer's cramp. In piano-players the neurosis appears chiefly in the paretic form—mild fatigue—and is usually associated with quite severe pains, that come on during playing, in certain parts of the arm. In regard to treatment, the best results are obtained by energetic massage. Finally, it may here be noted that in certain laborious occupations that are persistently practiced, a severe group of nervous symptoms may also arise. For example, B. Hirt has lately described an affection which occurs in sewing-machine girls, and is characterized by disturbances of sensibility, pain, paræsthesia, and in some cases anæsthesia, ataxia, absence of the tendon reflexes, and swaying with the eyes shut. The disease thus recalls very closely the picture of locomotor ataxia, but it is curable with proper treatment. Therefore Hirt suspected an affection of the peripheral nerves. Like symptoms are also seen in other classes of laborers, as in farm-hands after laborious toil in the fields.

CHAPTER V.

SIMPLE AND MULTIPLE DEGENERATIVE NEURITIS.

Ætiology and Pathology.—We term those changes in the nerves acute neuritis (inflammation of the nerves) where their vessels become very hyperæmic, and where the transudation of fluid and cellular elements takes place from the walls of the vessels into the surrounding tissue. The inflamed nerve is therefore swollen and thickened, its color is manifestly reddened from the marked vascular hyperæmia, and we can aften recognize with the naked eye a few or many small hæmorrhages. Microscopic examination shows an abundant infiltration of round cells into the nerve sheaths and the interstitial tissue, and these may be so numerous that the inflammation in some cases may be recognized, even on macroscopic examination, as a purulent neuritis. The nerve-fibers themselves sometimes show no visible changes, but in more severe neuritis we usually find an evident disintegration of the medullary sheaths and the axis-cylinders, and finally a complete destruction of the nerve-fibers. The "fatty granular cells" which are seen in these cases are probably white blood-corpuscles (perhaps, also, endothelial cells ?), which have taken up the fat from the disintegrated medullary substance. The destruction of the nerve-fibers, "parenchymatous inflammation," is partly a mechanical result of their compression by the surrounding exudation, but very largely probably the result of the direct injury which the nerve-fibers undergo from the causes that provoke the inflammation.

In its further course the neuritis advances to the stage of the new formation of connective tissue and the regenerative processes. The nerve appears firmer and denser than normal; a large amount of interstitial connective tissue is formed between the single nerve-fibers that are still preserved, which, if produced in excess, like a sort of callus formation, may lead to quite considerable partial thickenings of the nerve, the so-called *neuritis nodosa.* The capacity for regeneration of the peripheral nerves is relatively very considerable, so that in moderate and even in severe degrees of neuritis we may have a complete *restitutio ad integrum.* There may be a partial regeneration of the nerve-fibers even in the worst cases. Chronic neuritis either proceeds from acute neuritis, or it develops *de novo* in an insidious fashion. In the latter case the first acute stage of hyperæmia and cellular infiltration is entirely absent, and the destruction of the nerve-fibers and the new growth of connective tissue proceed in a chronic manner from the beginning. In such cases the interstitial inflammatory changes in the connective tissues and the vessels are often so subordinate to the degenerative processes in the nerve-fibers themselves that we have almost a genuine parenchymatous degeneration. Such conditions, perhaps, can not be properly termed "neuritis" at all, but rather "primary chronic degenerative atrophy of the nerves." They arise especially as a result of certain infectious and toxic influences (*vide infra*), which have a directly deleterious action upon the nerve-fibers. They often appear in many nerves at the same time, and consist of a slowly progressive disintegration of the medullary sheath, and later of the axis-cylinders also. It is possible that these last-named changes begin in the terminal branches of the peripheral nerves, and gradually advance from them toward the centers, but this has not yet been certainly confirmed; at any rate, the affection often remains confined to the peripheral nerves. The anterior spinal roots and the cord itself in such cases are found perfectly intact, or altered only in a subordinate way. [According to recent observations distinct changes may be found in the cord, chiefly in the white substance, especially in the more chronic forms of neuritis, and the forms of toxic origin.—K.]

In inquiring into the causes of neuritis we encounter first the same injurious influences which play the chief part in the inflammations of other organs. We very often speak of a traumatic neuritis, which is supposed to mean a neuritis produced by various mechanical injuries of the nerve. As far as this implies open wounds—like stabs, cuts, gun-shot wounds, etc.—that involve the nerve, the development of a genuine traumatic neuritis can not be doubted; but in such cases we not only have to do with mechanical lesions, but with an accidental complication of the wound, with the entrance of organized agents of inflammation through the wound into the nerves. Only in this case can there be an inflammation (ascending neuritis, *neuritis migrans*) advancing continuously or by leaps in the nerve-trunk from the point of lesion in the nerve, whereas, if the wound remains aseptic, as the experiments of Rosenbach and Kast have shown, such a propagation of the inflammation above the point of lesion never occurs. In the subcutaneous injuries of the nerve-trunk from a blow, from pressure, from dislocations of the bones, etc., we are, of course, not justified in speaking of a traumatic neuritis; but in such cases we have a purely mechanical destruction of the nervous elements, which is followed by the regular processes of secondary degeneration (*vide supra*), increase of connective tissue, and final restoration.

Another cause for the origin of a genuine neuritis lies in the extension of an inflammation from the neighboring organs to the nerves. In inflammations of the bones, as in caries of the bones of the skull and of the vertebræ, of the joints and of the different internal organs, the inflammatory process may directly involve a nerve-trunk by contiguity. In this way, perhaps, are developed the atrophy and paralysis of the neighboring muscles which are often seen after affections of the joints. Leyden has also tried to explain a number of the so-called "reflex paralyses"—that is, paralyses which sometimes develop as a result of inflammatory affections of certain internal organs, especially the intestines and the urinary and sexual organs—by a neuritis arising from the organ originally affected, and even extending to the spinal cord.

Primary neuritis, however, is of special interest. This may either be caused by some evident agency, or it may develop, apparently spontaneously, without any cause as far as known. We have already learned to recognize one group of these primary neuritides in the preceding chapters: those which, in all probability, underlie most of the "rheumatic" paralyses on the one hand, and certain forms of neuralgia, such as sciatica, intercostal neuralgia with zoster, etc., on the other. As we have said, little that is definite is known as to the precise cause of these neuritides. Some of them seem to arise from external agencies, such as exposure to cold; others, perhaps, from infectious influences. Certain toxic paralyses belong to a second group of primary neuritides, among them, for instance, the lead and arsenic paralyses already described, and the disease of the nerves produced by chronic alcoholism, which will be more fully described below. Finally, the so-called multiple neuritis forms the third recognized group, a special form of disease in which several nerves are usually affected at the same time or soon after one another. As will be stated in the description of the course of the disease, we probably have in these cases a definite form of infectious disease, which is localized exclusively, or at least mainly, in the peripheral nerves. In the acute cases the anatomical changes in the nerves seem to be really of an inflammatory nature, but in the chronic cases a simple degenerative atrophy of the nerves underlies the morbid symptoms. The ætiological position of these chronic cases of multiple neuritis, however, is, in our opinion, to be judged with caution, since at least a part of the cases so far published are certainly to be classed with alcoholic neuritis (*vide infra*).

Clinical History of the Different Forms of Neuritis.

1. SECONDARY NEURITIS.—The chief symptom of secondary neuritis, following wounds, inflammations of neighboring organs, etc., is pain, which comes on with great intensity, not only in the region of distribution of the affected nerve, but also along the whole trunk of the nerve. In these cases there is also a marked

sensitiveness of the nerve to pressure. In many cases we can succeed in feeling the thickened nerve plainly through the skin.

Besides these direct symptoms, the inflammation soon renders the necessary results of disturbed nervous conduction apparent. There is a dullness of sensibility in the distribution of the affected nerve, at first in the form of a subjective feeling of numbness, but later as a manifest objective anæsthesia, which, however, rarely reaches a very high degree. The motor symptoms at first show themselves as motor weakness, which in severe cases becomes a pronounced paralysis. It goes without saying (see page 538) that the paralysis, in all severe cases, is followed by a degenerative atrophy of the paralyzed muscles, and the appearance of electrical reaction of degeneration. In the skin, trophic and vaso-motor disturbances have been repeatedly observed, especially slight œdema of the subcutaneous cellular tissue, eruptions of herpes, etc.

The course of simple secondary neuritis may vary. Its onset is usually quite acute, or more rarely it begins gradually. Many cases seem to recover before the severer consequences are reached, while others take a chronic, tedious course, and lead to permanent disturbances of function.

2. PRIMARY SIMPLE NEURITIS.—We must seek for the cause of many primary paralyses (that is, paralyses coming on without special cause or after cold, etc.) in the distribution of individual peripheral nerves in a pure neuritis. For example, "rheumatic" facial paralysis is certainly due to a mild or a severe neuritis of the facial nerve. Precisely similar primary neuritic paralyses occur, although more rarely, in the distribution of other nerves, especially in the upper and more rarely in the lower extremities; thus we see neuritic axillary or deltoid paralysis, ulnar paralysis, peroneal paralysis, etc. All these cases are characterized by the fact that they begin with more or less severe pain or paræsthesia in the territory of the affected nerve. Motor paralysis sooner or later develops, which in the severer cases leads to degenerative atrophy of the affected muscles with reaction of degeneration. Sensory disturbances in the skin can usually be plainly detected, at least to a slight degree, on careful examination. The prognosis of these paralyses is, as a rule, favorable; but sometimes permanent disturbances of function remain, and they may also recur.

3. PRIMARY MULTIPLE DEGENERATIVE NEURITIS.—Primary multiple neuritis is probably not a very rare disease, but yet it has been carefully investigated only in the last few years. The first definite observations upon it were made in the years 1864 and 1866 in France by Duménil. Since then a whole series of well-established cases have been published by Eichhorst, Eisenlohr, Joffroy, Leyden, Vierordt, the writer, and others, so that the type of the disease is at present quite accurately known. Probably in former days multiple neuritis was often confused with poliomyelitis and certain cases of "acute ascending paralysis" (*vide infra*). An interesting discovery was first made by Scheube, that the peculiar disease, of endemic occurrence, long known in Japan and the East Indies as "*kak-ke*" or "*beri-beri*," is in its clinical and anatomical relations a well-characterized multiple peripheral neuritis. With us, also, multiple neuritis occasionally shows an epidemic increase in frequency (Eisenlohr). [Cases have been observed among sailors, fishermen, etc., in the seaport towns of New England, where the symptoms resembled those of neuritis and were akin to beri-beri. J. J. Putnam thinks that in 1881 and 1889 there were epidemics of this disease in the fishing towns of Massachusetts, characterized by pain, loss of power, numbness, and œdema, the trouble being chiefly in the legs.—K.]

Multiple neuritis usually begins acutely, sometimes almost in an apoplectiform manner, and without any definite occasion, precisely like an acute infectious disease. Febrile symptoms, with temperatures from 102° to 104° (39°–40° C.), come on in persons previously in good health, usually in adults in youth or middle life, with severe general disturbance, loss of appetite, dullness, headache, and sometimes even mild delirium. In these acute cases albuminuria and a slight enlargement of the spleen have sometimes been observed, which symptoms also point toward the infectious nature of the disease. In other cases the initial symptoms are less severe, but they are almost never entirely absent. The pains, which are hardly ever absent, are very characteristic. They are described as pulling and tearing, are felt chiefly in the loins and the extremities, and sometimes follow approximately the course of the large nerve-trunks. Since in some cases a number of the joints are swollen, the disease at first may be mistaken for acute articular rheumatism. The first symptoms of paralysis, usually in the lower extremities, appear very soon after these initial symptoms, or at the same time with them. The patient notices that he can not readily move one leg, and soon after he notices the same of the other. The paralysis often extends rapidly to one or both arms. If we examine the paralyzed parts more carefully we find a perfectly atonic and more or less extensive paralysis. The legs are often not paralyzed, but distinctly ataxic, a condition which is often seen in alcoholic neuritis (*vide infra*). The reflexes are almost always diminished, the tendon reflexes are usually entirely absent, and the cutaneous reflexes are weak, or they also have almost wholly disappeared. In only a few cases are the reflexes increased—a symptom which is to be regarded as analogous to cutaneous hyperæsthesia. We can usually make out, after a few days, a decided diminution of electrical excitability in the affected nerves and muscles, which finally becomes a pronounced reaction of degeneration. If the paralysis is of longer duration, there is a decided atrophy of the muscles. In these cases the severe initial symptoms of sensory irritation, as a rule, rapidly disappear, although slight pains, paræsthesia, and especially a considerable sensitiveness of the paralyzed parts to pressure and to passive motion, often last for a long time. In many acute cases the hyperæsthesia of the skin and of the deeper parts reaches a very high degree, but it is remarkable that the objective disturbances of sensibility are very slight in the great majority of cases. Marked anæsthesia is a rare exception, so that we may rightly suppose that primary multiple neuritis affects chiefly the motor nerve-fibers. We usually find no disturbances in the distribution of the cerebral and bulbar nerves. An affection of the optic nerve has been mentioned in only a few cases. The marked increase in the frequency of the pulse, which is usually present, is important, and probably depends upon a disturbance of the vagus. Trophic disturbances in the skin, hair, and nails are not very rare. Œdematous swelling of the affected extremities has also been repeatedly observed. The functions of the bladder and rectum, however, almost always remain undisturbed.

In regard to the course of the disease, in the severest cases it may soon terminate fatally, almost always because the paralysis extends to the muscles of respiration. The inspirations are labored, and are performed with the upper part of the thorax only, while the epigastrium is motionless, or sinks in on inspiration from the paralysis of the diaphragm. There is also paralysis of the other muscles of respiration, the abdominal muscles, etc., so that, after the disease has lasted a week or ten days, death ensues with all the signs of respiratory insufficiency. A second class of cases also begins quite acutely, but then takes a chronic course. The initial acute febrile symptoms cease after a few days, and the paralysis develops to some extent. Then the affection seems to come to a stand-still, and the first signs of improvement gradually begin to appear. Since there is a more or less pronounced atrophy of the muscles in these cases, it always takes quite a long time—usually several months—for recovery. A third class of cases follow a chronic course from the outset, although even in these cases there may be more acute exacerbations of the disease. In these cases quite extensive atrophic paralysis gradually develops in the lower extremities, and usually in the upper extremities also. The reflexes disappear; the sensibility is as a rule somewhat, but very rarely much diminished. Pains are always present at first, but later on in the disease they often become subordinate. The bladder and rectum usually remain intact in their functions. If the disease advance gradually it may terminate fatally even at a late period, after a course of months, usually from a final paralysis of respiration; but, on the other hand, even after a protracted course, the disease may come to a stand-still, and even to a complete or at least to a partial recovery. It is worthy of note that a combination of multiple neuritis and pulmonary tuberculosis has quite frequently been observed; but nothing definite is yet known as to the form of connection between the two diseases.

The diagnosis of multiple neuritis is, as a rule, easy, for one who is acquainted with the disease and notes the different symptoms carefully. In regard to diagnosis, the chief importance should be placed on the generally acute beginning, with pronounced symptoms of sensory irritation, with frequently a very considerable sensitiveness of the nerves to pressure and general cutaneous hyperæsthesia; and also on the appearance of a rapidly extending paralysis, whose peripheral nature may be attested by the presence of reaction of degeneration, muscular atrophy, and the absence of the cutaneous and tendon reflexes. Such a paralysis can be produced by nothing but an affection of the peripheral nerves or poliomyelitis (*vide infra*). As we have shown above, this latter disease may, in fact, often be confused with multiple neuritis, but a careful attention to the initial symptoms, especially to the disturbances of sensation, will usually make the differential diagnosis possible.

The prognosis of multiple neuritis is doubtful, as is shown by the description of the course of the disease, but it is by no means very unfavorable. If the first acute stage of the disease has been gone through with without accident, we may hope for recovery, or at least actual improvement, even with extensive paralysis. Such striking results in the way of recovery, after paralysis that has lasted for months, are also important in regard to diagnosis, since such extensive processes of regeneration are possible in affections of the peripheral nerves, but scarcely in spinal diseases; and hence they are sometimes an additional confirmation of the diagnosis of a neuritis.

Treatment.—In the first stage of the disease, especially if severe pains, swelling of the joints, or high fever be present, it is advisable to try the exhibition of salicylic acid, from which several observers have seen a favorable effect. We give ten grains (grm. 0·5) of the acid every hour, or a few larger doses, a drachm to a drachm and a half (grm. 4–6) of salicylate of sodium. Instead of salicylic acid we have lately used antipyrine and phenacetine with good results. When the pain is very severe we must use narcotics, such as injections of morphine. Embrocations of chloroform, and sometimes protracted warm baths, have also a palliative effect. In the further course of the disease proper care, a suitable position for the limbs, and diet—nourishing food—are the main things for the patient. The regenerative processes of recovery begin spontaneously, if at all, but we may hasten recovery and make it complete by a subsequent electrical treatment, especially galvanism. For the completion of the recovery, bathing (simple warm baths, salt baths, etc.) is serviceable, and also the baths at Teplitz, Wiesbaden, and Rehme.

4. The Chronic Neuritis of Alcoholic Subjects.

(*Pseudotabes of Alcoholic Subjects, Ataxia of Drinkers.*)

It has long been known that peculiar nervous affections often occur in alcoholic subjects (M. Huss, Lendet, and others); but up to the present time a disease of the spinal cord has been assumed to be the cause of the symptoms, and only of late have we obtained the knowledge that at least a great part of the cases of this class are to be regarded as a special form of chronic multiple neuritis (Lancereaux, Moeli, and others). The practical importance of this alcoholic neuritis is not slight; first, because it may easily be confused with other nervous diseases, especially with tabes dorsalis, and, second, because its proper and timely diagnosis is of great significance in regard to treatment.

Alcoholic neuritis occurs chiefly in two forms: in one form actual atrophic paralysis develops chiefly in the lower extremities, but in the other form the paresis is subordinate to the other disturbances of innervation. The first symptom of the disease is usually tearing and drawing pains in different parts of the lower, or, more rarely, of the upper, extremities. The pains are usually quite severe, but sometimes only of moderate strength. Sooner or later, but sometimes not for years, a pronounced disturbance of the gait is added to the pains. More careful examination shows that in such cases there is partly an actual paresis of the muscles of the legs, and partly a form of ataxia—a defective innervation which is manifest by an uncertainty, a staggering and reeling in the gait. If there be marked paresis, the affected muscles are quite atrophic, and electrical examination usually gives a decided diminution of excitability, or even pronounced reaction of degeneration. The patellar reflex is usually lost quite early. The sensibility, too, is very rarely perfectly normal; we find sometimes quite marked anæsthesia, especially in the lower legs, and also in other parts of the skin. The cutaneous reflexes are also often feeble and slow. There is sometimes considerable tenderness on pressure on the deeper parts and over the nerves. The course of the disease is usually chronic. If the cause of the evil—the abuse of alcohol—be removed in time, perfect recovery is possible; but in far-advanced cases the disease may go on to complete paralysis even of the upper extremities, and to a fatal termination. [In women, especially, mental symptoms are often pronounced. It is a characteristic feature of the delusions of alcoholic neuritis, that the patient will tell of walks taken, and of people seen, although at the time he is helpless in bed.—K.]

As may be seen, the disease is decidedly like tabes dorsalis, and in the early stages, with pain, ataxia, and absence of patellar reflex, the differential diagnosis has no slight difficulty. In regard to this it must be borne in mind that the reflex immobility of the pupils, the girdle sensation and disturbances of the bladder, seem to be absent in alcoholic neuritis, at least as a rule, while, on the other hand, the development of atrophic paralyses may almost certainly exclude tabes.

The treatment demands, in the first place, the entire abandonment of the further use of alcohol, if possible. In mild cases we can obtain a decided improvement by this alone. In more advanced cases electrical treatment and tepid baths do the best service. We would also recommend the internal or subcutaneous use of preparations of strychnine.

CHAPTER VI.

NEW GROWTHS IN THE PERIPHERAL NERVES.

THE new growths in the peripheral nerves are usually divided into false and true neuromata. The former are not newly formed nervous tissue proper, but are fibromata, myxomata, sarcomata, etc., which develop on the nerves. Infectious tumors, also, especially syphilitic gummata, and still more frequently the new growths arising in leprosy, may have their seat on the peripheral nerves. The true neuromata consist of newly formed, usually medullated, nerve-fibers (*neuroma myelinicum* of Virchow), which are imbedded in a frequently very abundant connective-tissue stroma. These neuromata develop most frequently in the cut ends of nerves in amputation-stumps (amputation neuromata), but they may form after other injuries of the nerves, and probably many neuralgias and persistent pains after injuries are due to the formation of such little neuromata. The multiple occurrence of neuromata, which has also been repeatedly observed, is very remarkable. These develop by hundreds in the same individual, chiefly on the spinal nerves, only occasionally and exceptionally on the sympathetic or cranial nerves. In such cases the different tumors are by no means metastases from one original tumor, but are the expression of a general and often hereditary predisposition of the peripheral nervous system to the formation of tumors. Sometimes multiple neuromata are combined with other anomalies of the nervous system, such as cretinism. Besides the medullated neuromata, there are also new growths of non-medullated nerve-fibers (*neuroma amyelinicum*), but their histological diagnosis is always very difficult.

The symptoms of neuromata vary very much in different cases. Many of them cause no symptoms at all, but in other cases they are the cause of extremely severe and persistent neuralgias and neuralgiform pains, which come on with varying intensity, are usually remittent or intermittent, and are often iencrased by external causes, the influence of the weather, etc. Marked symptoms of pressure, especially anæsthesia and motor paralysis, are only exceptionally developed, but they sometimes do occur, especially in neuromata of the cauda equina. Direct or reflex symptoms of motor irritation, such as tremor or tonic spasms, are somewhat more frequent.

The so-called *tubercula dolorosa* deserve special mention. By this term we mean little nodules which may be felt beneath the skin, and are usually readily movable and very sensitive to pressure. They are not very rare, and are usually associated with drawing pains, which are rarely decidedly neuralgic and are not very strictly localized. They are situated in the extremities, especially in the arms, and in the back, the neck, etc. It is worthy of note that the symptoms are only at times very prominent, and then they disappear again, and that with this disappearance is certainly sometimes associated a spontaneous disappearance of the nodule. The anatomical nature of the tubercula dolorosa is not always to be established with certainty. Many of them are true neuromata, but others belong to other kinds of new growths.

The course of neuromata is of course very chronic. In some cases the persistent severe pain may give rise to considerable general disturbance, but a final spontaneous cessation of the symptoms, and even a disappearance of the new growths, have also been observed.

The diagnosis of neuromata is possible only when the tumors can be felt through the skin, and when their seat, as well as their clinical symptoms, correspond to the course and distribution of a nerve. In multiple neuromata the diagnosis has been repeatedly confirmed by the excision and examination of one of the tumors.

The only successful treatment of neuromata is extirpation, which is to be undertaken only when the symptoms are very severe. If extirpation be not practicable, or if we have to do with multiple neuromata, the patient's trouble can be alleviated only by symptomatic means, narcotics, and electricity. If we can compress the nerve above the neuroma, we can often cause by this means a temporary cessation of the pain.

II.—Vaso-motor and Trophic Neuroses.

CHAPTER I.

PRELIMINARY REMARKS UPON VASO-MOTOR, TROPHIC, AND SECRETORY DISTURBANCES.

BESIDES the disturbances of sensibility and motility described in the preceding sections, we also see in nervous patients frequent anomalies of the vaso-motor and trophic functions, but up to the present time we know comparatively little that is certain as to the precise nature of their occurrence.

Physiology, as is well known, distinguishes two varieties of vaso-motor nerves —the vaso-constrictors and the vaso-dilators; but since experiments have detected the latter variety in only a few places—for example, in the chorda tympani, the nervi erigentes, and the sciatic—they have not acquired a very great significance in human pathology. We are at present much more disposed to refer every abnormal constriction of the vessels to an irritation, and every abnormal dilatation of the vessels to a paralysis of the vaso-constrictor nerves, although perhaps pathological conditions of irritation of the vaso-dilators may not be at all rare. In regard to the precise anatomical course of the vaso-motor nerves, we must first state that vaso-motor irritations may certainly proceed from the cerebrum, as is shown by the well-known symptoms of blushing and pallor from mental emotions. In experiments on dogs, Eulenburg and Landois have succeeded in producing a fall of temperature on the opposite side by irritating certain portions of the cortex in the immediate vicinity of the motor centers, and by extirpation of the same parts they have produced a rise in temperature. Furthermore, we know with certainty that there is an important vaso-motor center in the medulla oblongata (in the region of the upper olivary body in rabbits), the irritation of which, directly or reflexly, is followed by an almost universal vascular constriction, and its destruction by an almost universal vascular dilatation. We must probably seek the further course of the vaso-motor nerves very largely (or exclusively ?) in the lateral columns, from which they pass out chiefly by the anterior roots; but there are also experimental data (Stricker) suggesting the presence of vaso-motor nerves in the posterior roots. It is not known with certainty whether there is any decussation of the vaso-motor fibers, or, if there is, where it occurs. The larger part of the vaso-motor nerves collect, at any rate, in the principal trunks of the sympathetic, from which, as is well known, the separate plexuses that surround the vessels arise. It is not improbable, however, that there is also in part a direct passage of vaso-motor fibers from the cord into the peripheral nerves. In conclusion, we must mention that, according to Goltz's experiments, there are reflex vaso-motor centers in the cord for the different parts of the body.

The clinical vaso-motor symptoms are chiefly to be observed in the external skin. We distinguish them as follows:

1. *Symptoms of Vaso-motor Paralysis.*—We conclude that there is a paralysis of the vaso-motors if there is an abnormal redness of the skin. Such a redness is almost always associated with an objective and often a subjective feeling of an increase of temperature. Such conditions are observed either in connection with other nervous symptoms—as in fresh spinal or cerebral paralyses, and also

very often in certain functional neuroses, such as hysteria and neurasthenia—or in the form of independent affections—the pure vaso-motor neuroses, injuries of the cervical sympathetic, etc. There are cases in which the only symptoms are a persistent or paroxysmal redness of the skin, especially of the head, associated with a great feeling of heat, with palpitation of the heart, strong pulsation of the arteries, anxiety, ringing in the ears, and sweating. If the affection be confined to a single extremity, in which there are paroxysmal redness, diffuse swelling, and pain, we have the condition described by Weir Mitchell as *erythromelalgia.* As was stated above, it is at present impossible to decide whether many of the symptoms just mentioned do not perhaps depend upon an irritation of the vaso-dilator nerves.

2. *Symptoms of Vaso-motor Spasm.*—Spasm of the small vessels becomes apparent by a striking pallor and coolness of the skin. There is often, with this, a decided feeling of formication and stiffness in the affected parts, which may even increase to an actual feeling of pain. Such vaso-motor spasms affect the hands especially, and form a chronic trouble that is not very rare. They are usually seen in people who are generally nervous and irritable, and sometimes also in washerwomen. A vascular spasm is sometimes seen in the extremities as one symptom of complicated paroxysms, such as nervous angina pectoris (*q. v.*), especially at the beginning of the paroxysm. A persistent spasm of the small arteries may give rise to considerable subsequent trophic disturbance. At least, the rare cases of so-called "spontaneous symmetrical gangrene" [Raynaud's disease of the extremities], and also certain forms of scleroderma and some similar affections, are referred by many observers to a primary spasm of the vessels. [Other observers are disposed to attribute many if not all the cases of symmetrical gangrene to arterio-sclerosis or syphilitic endarteritis affecting the smaller vessels. —K.] There is a condition, seen especially in the hands, in which, without known cause, the skin becomes dark blue and icy cold, and the epidermis is raised in different parts into bullæ. This condition may even attain to a circumscribed gangrene—spastic gangrene.

We have much less information concerning the trophic nerves than we have concerning the vaso-motor. As is well known, the controversy is still going on as to whether we have any right to assume the existence of special trophic nerves. Clinical facts speak decidedly in favor of this assumption, although we have already stated that many trophic disturbances are probably due to vaso-motor changes, and also that the anæsthesia of many parts is a very favorable circumstance for the occurrence of disturbances of nutrition (compare what was said in regard to anæsthesia of the trigeminus on page 514).

Those changes in the skin which depend essentially upon an abnormally great exudation from the vessels form a transition between vaso-motor and trophic disturbances. Among them are, first, peculiar cases of disease which have been termed "**acute angioneurotic œdema**" (Quincke, Strübing, and others). In these cases œdematous swellings appear suddenly in various parts of the body, and sometimes disappear after a few hours, but they may be very often repeated. Dangerous symptoms may arise if the œdema affect the pharynx or the entrance to the larynx. The patient's health otherwise is sometimes perfectly good, but in other cases it is more or less affected. Gastric disturbances especially (attacks of vomiting and gastralgia) have been observed at the same time in such patients. Acute angioneurotic œdema is manifestly closely allied to urticaria and erythema exudativum. In regard to the occurrence of herpes zoster in nervous diseases, compare what was said on page 525. Vesicle formations analogous to herpes zoster intercostalis may also occur along the track of other nerve-trunks in peripheral, or even in purely spinal (?) nervous affections.

Among those symptoms which chiefly support the theory of the existence of specific trophic nervous influences, we have already learned to recognize the degenerative atrophy of the muscles and nerves (see page 538). Various other sorts of trophic disturbances in the skin and the deeper parts are seen in nervous diseases. Especially after wounds of the peripheral nerves we often notice a peculiar shining, smooth, atrophic condition of the skin—the "glossy skin" or "glossy fingers" of the English authors. In other cases anomalies in the pigmentation of the skin seem to be connected with the nervous disturbances; thus spots deprived of pigment (*vitiligo*) often develop as a result of severe neuralgias. We must also bring to mind here the appearance of changes in pigmentation from nervous causes, especially the ætiology of Addison's disease (*vide infra*) and the occurrence of the so-called nervous nævi. Among the severe neurotrophic disturbances of the skin many observers, especially Charcot, class the appearance of acute bedsores in many spinal and cerebral paralyses, but we have never been able to convince ourselves of the occurrence of a "neurotrophic decubitus," and we believe that every bedsore is due in the first instance to external influences, uncleanliness, and pressure on the skin.

We may here mention **myxœdema** (*cachexie pachydermique* of Charcot), which was first described in England by William Gull and Ord. The disease has taken its name from a peculiar thickening and swelling of the skin, which is most marked in the face, but which is sometimes seen in the extremities, the trunk, the tongue, and even in the internal organs. This swelling is not œdema, but it is due to the development of a sort of myxomatous new growth, rich in mucine, in the connective tissue. Other trophic disturbances usually co-exist: atrophy of the teeth and nails [spade-like hands, coarseness and] loss of hair, failure in the sweat secretion and consequent dryness of the skin, etc. Besides these there gradually develops a general physical and mental weakness, which may increase to great hebetude or even to complete dementia. Disturbances of the sensory functions may also occur. It is a fact of especial interest that we find quite commonly a diminution and even a complete atrophy of the thyroid gland. It is not improbable that all the symptoms of the disease may arise from the failure of function of the thyroid gland. This hypothesis is confirmed by the repeated experiments of Kocher and others, that after complete extirpation of the thyroid in man almost the same symptoms ensue as in myxœdema (*cachexia strumipriva*). From this we must suppose that certain injurious substances accumulate in the body which can no longer be made innocuous by the thyroid gland.

Besides the trophic disturbances in the skin, we often see analogous changes in the nails and hair in nervous patients. The nails become brittle and cracked, assume a darker color, and often show a considerable thickening (*onychogryphosis*). We also see at times a loss of the nails. A loss of hair is seen in frontal neuralgia, in certain forms of headache, and not infrequently as an apparently independent nervous disease (*alopecia*). A very rapid whitening of the hair after mental excitement is well known to have occurred in some cases.

Among the trophic disturbances of the deeper parts the symptoms sometimes seen in the bones and joints deserve a brief mention. The implication of the bones in atrophic processes is seen chiefly in progressive unilateral facial atrophy (*vide infra*). A retarded growth of bone in the affected extremities is also a symptom frequently seen in the spinal and even in the cerebral paralyses that develop in childhood, which proves most plainly that the processes of growth depend upon the nervous system.

A "trophic disturbance" chiefly of the bones, but also involving the soft parts, forms the basis of that peculiar and rare disease recently termed **acromegaly** (P. Marie, Erb, etc.). The affection develops slowly in women and men, usually in youth or middle age. Besides the general symptoms of dullness, fatigue, and quite severe neuralgic or rheumatic pains in the head and the extremities, there gradually develops a striking increase in the size of the hands and feet, and thickness and plumpness of the face, due chiefly to enlargement of the nose, chin, and lips, which become puffed out to a considerable size. The hands and feet become actual paws. Later in the disease the legs and forearms also increase in circumference. The hypertrophy seems to affect the bones chiefly, but in some cases the skin also seems thicker, without showing those marked changes characteristic of myxœdema. In a case we have lately seen the tongue also showed distinct enlargement; sugar was also occasionally present in the urine, and with this there was a profuse and almost uninterrupted secretion of sweat. The voice was weak, deep, and hoarse, as a result of an evident paresis of the vocal cords. The few autopsies in acromegaly have thus far not been able to contribute much in the way of explanation of this remarkable affection. In all cases hyperplasia of the hypophysis cerebri was a striking lesion, and in the case of Klebs the persistence and hyperplasia of the thymus gland. Erb found in his cases a dullness in the upper sternal region, which he is disposed to attribute to a persistent thymus. In our case this dullness could not be detected. The significance of all these discoveries is still an utter enigma.

Trophic affections of the joints have been repeatedly confirmed in cerebral and spinal diseases, especially in tabes dorsalis (*vide infra*). As a special vaso-motor-trophic articular neurosis we may mention here the so-called **hydrops articulorum intermittens**. We mean by this a very rare but perfectly typical disease, in which large swellings, usually of the knee-joint, but sometimes of the other large joints, develop at perfectly regular intervals of one to four weeks. They continue without fever, and usually without any great pain, and disappear again in a few days. Such attacks may be repeated at intervals of different lengths, during years and years. Their nervous character is attested especially by the rapid onset and disappearance of the affection, and also by the combination of it with other nervous disturbances, such as angina pectoris, exophthalmic goître, vaso-motor symptoms, etc., which combination has often been observed. In regard to treatment we may try salicylic acid, quinine, Fowler's solution, and subcutaneous injections of ergotine.

In addition to the trophic disturbances we must consider the disturbances of secretion. These are not infrequent. We have already learned to recognize anomalies in the secretion of saliva in facial paralysis, and of the lachrymal secretion in trigeminal neuralgia. Analogous symptoms are occasionally noticed in other nervous diseases. Disturbances of the sweat secretion are the easiest to confirm. Our understanding of them comes substantially from the discovery of the "sweat nerves," arising mainly from the sympathetic, which was first made by Luchsinger. In nervous patients we have seen quite frequently, on the one hand, an abnormal increase of the sweat secretion (*hyperidrosis*, *ephidrosis*), and, on the other, a diminution or a complete disappearance of it. The former is seen on the paralyzed side in many hemiplegias and in spinal paralyses, the latter in tabes dorsalis. Anomalies of the sweat secretion are quite frequent, and are usually combined with vaso-motor disturbances, in certain general neuroses, such as hysteria and neurasthenia. In a few rare cases a genuine hæmatidrosis (bloody sweat) has been confirmed. The condition known as unilateral hyperidrosis (unilateral sweating) is also especially interesting. In this there is an abnormal secretion of sweat, chiefly in one half of the face, more rarely in one arm or over the whole of one side. The affection has usually been observed in connection with hemicrania, exophthalmic goître, hysteria, etc., and, in at least a number of cases, it seems to be due to direct lesions of the sympathetic. On the other hand, we have repeatedly seen persons, who were otherwise perfectly healthy, in whom the secretion of sweat, coming on under normal conditions from heat or physical exertion, remained limited to one half of the body, especially the face.

In conclusion, we would briefly mention here the symptoms which have been observed in direct **injuries of the cervical sympathetic,** wounds, pressure of neighboring tumors, etc. If we have to do with a paralysis of the sympathetic, we see almost constantly on the affected side a contraction of the pupil from paralysis of the dilator pupillæ supplied from the sympathetic, in many cases associated with a slow reaction to light. We also frequently see a narrowing of the opening of the lids from paralysis of Müller's muscle, and in old cases a retraction of the bulbus oculi, and occasionally increased redness and warmth in the ear and cheeks from vaso-motor disturbance. In a few cases we see an increased sweat secretion. We may add that, according to Möbius, the normal reflex dilatation of the pupil, from a painful irritation of the skin of the face, is absent in paralysis of the sympathetic. The opposite symptoms are found in conditions of irritation of the sympathetic. In both cases there are sometimes slight trophic disturbances in the cheeks. It is also worthy of note that in tumors of the neck, and after injuries of the brachial plexus, there are sometimes disturbances in the sympathetic, especially changes in the pupils, which are due, as is supposed, to a lesion of the communicating branches between the principal trunk of the sympathetic and the brachial plexus. The occurrence of sympathetic symptoms in certain injuries of the brachial plexus has already been mentioned above (page 567).

CHAPTER II.

HEMICRANIA.

(*Migraine.*)

Ætiology.—By hemicrania we mean a peculiar form of unilateral headache, due probably to vaso-motor disturbances, or at least almost always associated with vaso-motor symptoms. The affection occurs especially in women, more rarely in men, and almost always begins in youth, generally at the period of puberty, but typical cases of migraine have been repeatedly observed in school-children. Quite frequently, but not always by any means, the disease affects women who must be regarded as "generally nervous," who are anæmic, or who suffer from disturbances of menstruation. Heredity often plays a part, since hemicrania is both hereditary as such, and often appears in families which have suffered from other nervous diseases, such as epilepsy, hysteria, or the psychoses. We may mention as exciting causes, which may be made answerable both for the onset of the disease and quite often for the individual attacks, physical and mental overexertion, great mental excitement, and disturbances of digestion. [Eye-strain (*vide supra*) may be a cause of migraine as well as of other forms of headache, and a careful search for any errors of refraction should be made in every case.—K.]

We do not know the special cause of hemicrania. Considering the accompanying vaso-motor symptoms, which are present in migraine as a rule (*vide infra*), it is almost universally assumed that we must regard the disease, from its chief cause, as an affection of the sympathetic; but we must agree with Möbius that this assumption is by no means confirmed, and that it is possible that the accompanying sympathetic symptoms may be only secondary and of reflex origin, in consequence of the pain. We also have no definite information as to the special seat of the pain in migraine, but it is most probably to be placed in the meninges—the pia and dura mater.

Symptomatology.—Migraine always comes on in separate attacks, which are repeated at intervals of varying lengths, although some cases often show a remarkably great regularity. The onset of the attack in women often has some relation to the menses. The left half of the head is much more frequently affected than the right. In some cases it happens that the pain affects the right and the left sides alternately; in others it is not generally very strictly limited to one side.

The attack of migraine usually begins with certain prodromal symptoms, which the patient soon recognizes as sure signs of his approaching suffering. These prodromal symptoms consist of general uneasiness, discomfort, pressure in the head, vertigo, at times tinnitus, spots before the eyes, chills, malaise, abnormal yawning, etc. In a short time the pain begins. It is felt most either in the anterior frontal region or in the temporal or parietal region; it generally shows a continuous character, not intermittent as in neuralgia, and may increase to a very great intensity. Special painful points are usually absent, but the whole skin over the head on the affected side is usually hyperæsthetic. The general malaise continues with it; the patient has absolutely no appetite; there is often great nausea, and almost always a great sensitiveness to external impressions, to any bright light, or to any noise. In many cases of ophthalmic hemicrania ocular disturbances are especially prominent; bright scintillations before the eyes, scintillating scotoma, and quite often a pronounced hemianopsia, may be made out during the attack.

The vaso-motor symptoms are of special interest because they are of value in the theory of the disease. From them migraine is usually divided into two subdivisions—*hemicrania sympathico-tonica* or *spastica*, and *hemicrania sympathico-paralytica* or *angio-paralytica*.

In hemicrania spastica, first described by Du Bois-Reymond from observations on himself, the forehead and ear on the affected side are pale, the skin is cool, the temporal arteries contracted, the pupil is often decidedly dilated, the secretion of saliva increased—in short, there are a whole row of symptoms present which all agree in pointing to a condition of irritation in the sympathetic (*vide supra*).

In hemicrania paralytica, however, which was first described by Möllendorff, also from observations on himself, the face is reddened on the affected side, it feels hot, the temporal arteries are dilated and pulsate strongly, there is sometimes unilateral sweating of the face, the pupil is contracted—all symptoms which can depend only on a paralysis of the sympathetic.

As has already been hinted, the significance of all these symptoms is not absolutely certain, and we must also add that the cases which occur in practice can not by any means always be inserted into one or the other typical scheme without further ceremony. The vascular symptoms are sometimes but slight, conditions of paralysis and of irritation of the sympathetic sometimes seem to alternate with each other in the same attack, and we may even frequently meet with apparently contradictory symptoms at the same time, such as pallor joined with contraction of the pupil. The peculiar method of the origin of the pain is at present also almost wholly unexplained. If we assume primary vascular changes in hemicrania, we must look for the cause of the pain in the disturbance of the circulation, or perhaps, in spastic hemicrania, in the spasmodic contraction of the vessels itself.

The duration of the attacks of migraine differs very much in different cases. It usually lasts several hours, or a whole day; then the pain gradually disappears, and there is often considerable vomiting and sometimes a profuse discharge of urine toward the end of the attack. In the intervals between the different attacks most patients are perfectly well and free from pain.

The whole course of migraine is very chronic, and may last for years and years. It is usually a trouble to which the patient finally becomes accustomed. We must generally be quite guarded in our prognosis, for many cases resist very obstinately all attempts at cure. We can give the patient only the consolation that the trouble generally disappears of itself in advanced life. It is not usually attended with any special danger. In only a few cases has it been noticed that attacks of hemicrania of years' duration have preceded a severe cerebral disease that developed later, or tabes dorsalis.

Treatment.—Very many patients who suffer from migraine finally renounce any special treatment, after they have exhausted all possible remedies. They withdraw to their rooms when the attack comes on, darken the windows, take nothing but some tea, Seltzer-water, or cracked ice, put a cold compress about the head, perhaps try a foot-bath, and for the rest wait quietly until the attack is over. In fact, our remedies for cutting the attack short are quite uncertain. They sometimes aid, but they often leave us in the lurch, especially if used repeatedly. We must note especially that narcotics, such as morphine, are almost always ill borne in migraine, and do no good; but antipyrine, sodic salicylate (20–30 grains [grm. 1·5–2·0], in strong black coffee), antifebrine, and phenacetine in many cases undoubtedly have a favorable action. Which remedy acts the best must usually be tested in the individual case. We have ourselves seen excellent results formerly from sodic salicylate, and lately from antipyrine; the attacks of migraine, when the remedy was taken immediately upon the onset of the first symptoms, have become distinctly milder and have run their course more rapidly. Of course the action often ceases with time, and we must then try another of the remedies mentioned. Of the other remedies used we may mention guarana (*Paullinia sorbilis*). half a drachm to a drachm (grm 2–4) of the powder, and caffeine, or sodio-salicylate of caffeine, in three- to five-grain doses (grm. 0·2–0·3), which is sometimes very serviceable. On theoretical grounds, inhalations of nitrite of amyl, three to five drops on a napkin, have been used in spastic hemicrania, and subcutaneous injections of ergotine in the paralytic form (aqueous extract of ergot, 2·5; dilute alcohol and glycerine, each 5; or one part of dialyzed ergotine in four parts of distilled water; injections of either, three to fifteen minims). Many other nervines, such as bromide of potassium and Fowler's solution, have been recommended for continued use, and also extract of cannabis indica; and, lately, nitrite of sodium, two parts in 120 of water, a teaspoonful one to three times a day. Its action is analogous to that of nitrite of amyl. Nitro-glycerine, in troches containing $\frac{1}{120}$ to $\frac{1}{60}$ grain (grm. 0·0005–0·001), also has the same dilating action on the vessels. In ophthalmic migraine the treatment by large doses of bromide of potassium is praised, especially in France, as being very successful.

In many cases the general treatment is very important. Preparations of iron, sea-bathing, a mountain residence, and cold-water cures are often of distinct service. In patients with co-existing gastric symptoms a course at Carlsbad is sometimes of permanent benefit. The persistent application of electricity has also shown some good results, but we must not build very great hopes upon it. In the spastic form the action of the anode on the sympathetic is to be especially tried, and in the paralytic form the action of the cathode, while the other electrode is to be placed on the cervical cord, or as high as possible on the occiput in the region of the medulla. Cautious galvanization of the head, and weak primary faradic currents, may also be used. Specialists in massage praise their mode of treatment in migraine; they massage either certain painful spots in the head, or the gastric region. Finally, we must mention that migraine often seems to be connected with diseases of the nose, especially with hyperplasia of the erectile tissue in the nose, and that in such cases treatment of the primary disease with the galvano-cautery may result in the cessation of the migraine.

CHAPTER III.

PROGRESSIVE FACIAL HEMIATROPHY.

(*Unilateral Progressive Facial Atrophy.*)

Unilateral facial atrophy is an extremely rare disease, of which only about ninety cases have been recorded in literature up to the present time. The disease consists in a very slow and gradual but usually constantly progressive atrophy of one half of the face, and affects the skin, and also the fatty tissue, the muscles, and the bones, either in a uniform or a very diverse manner. The affection usually begins in youth. The female sex seems to be more disposed to the disease than the male.

The atrophy, which has its seat much more frequently on the left side than on the right, begins usually in a circumscribed spot either on the cheeks or on the chin. The skin, as a rule, gradually assumes a whitish or brownish color. The affected part, and finally the whole half of the face, gradually sink in more and more, so that the disease can be recognized at the first glance. The atrophy shows a sharp limitation at the median line. In many cases the muscles apparently remain almost wholly intact, but in some cases they show a marked atrophy, especially the muscles of mastication. The corresponding half of the tongue and the soft palate has sometimes been found implicated. Exceptionally, the atrophy involves the neighboring region of the shoulder and the upper extremity. The bones also atrophy, especially in the cases which arise in early youth. The hair on the affected half of the head often falls out in great amount, and it becomes thin and atrophic. The sensibility remains perfectly intact. Marked vaso-motor and secretory disturbances have only rarely been observed. The accompanying illustration (Fig. 80) shows a patient who was described by Romberg about thirty years ago, and who at present still frequents the German cliniques in order to show himself.

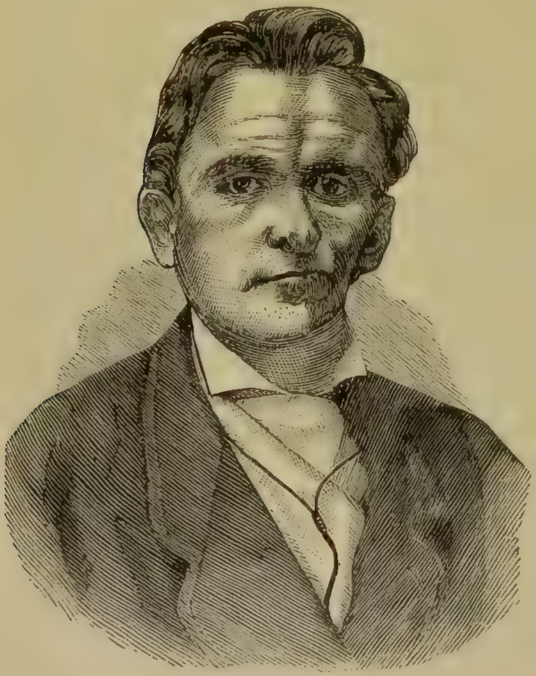

Fig. 80.—Left facial hemiatrophy.

Nothing definite is known in regard to the nature of the affection. Most observers at present agree that it is a trophic neurosis, an affection of trophic nerves or nerve-centers, but where we are to look for the special seat of the disease, whether in the trigeminus or in the sympathetic, we do not know. Mendel lately found in a case which came to autopsy a distinct neuritis in the trigeminus.

The disease is not dangerous in itself, and usually causes no special subjective disturbance, but it seems to be incurable. In cases at their beginning we can at most make an attempt to bring the disease to a standstill by a long-continued application of electricity.

As an appendix it may be briefly mentioned here that there is a unilateral hypertrophy, which is also possibly connected with neurotrophic disturbances. We have at present under observation a ten-year-old boy, otherwise perfectly healthy, in whom a striking hypertrophy of the left side of his face and of the left arm has gradually developed.

CHAPTER IV.

EXOPHTHALMIC GOITRE.

(*Graves's Disease. Morbus Gravesii. Basedow's Disease. Morbus Basedowii. Glotzaugenkrankheit.*)

Ætiology.—The special group of symptoms to which the name of exophthalmic goître has been given, and whose three cardinal symptoms are acceleration of the pulse, goître, and exophthalmus, was first carefully described in Germany, in the year 1840, by the Merseburg physician Basedow, although similar but less precise observations had been published in England by Graves five years earlier. The anatomical cause of this disease is still entirely unknown to us, but the whole type, and almost all the single symptoms of the affection, point definitely to an affection of the nervous system, which, with regard to the most prominent symptoms, is usually considered a "vaso-motor neurosis" or an "affection of the sympathetic"; although, as may be seen from what follows, this hypothesis still lacks any basis in fact.

With reference to the special ætiology of the disease all those factors are prominent which generally play the first part in the ætiology of neuroses. In many cases a hereditary predisposition can certainly be discovered. The disease has been repeatedly seen in several members of the same family. Furthermore, exophthalmic goître is also quite frequent in those families in which there is a hereditary predisposition to neuroses in general—epilepsy, the psychoses, or hysteria. Among the exciting causes we must first mention great mental excitement—grief, terror, anger. Sometimes real injuries, as well as these "psychical injuries," seem to have an influence on the development of the disease—that is, great general bodily concussion, such as a fall. Many authors have laid considerable weight on diseases of the female sexual organs, but the importance of this factor seems to us to be exaggerated. It is certain, however, that the first symptoms of exophthalmic goître often develop at the period of pregnancy.

The influence of sex upon the origin of the disease is plain, since women, especially somewhat anæmic, "nervous" women, are much more frequently affected than men. Exophthalmic goître usually appears in middle life, while it is seen only exceptionally in children and old people.

Symptomatology.—Of the three cardinal symptoms of exophthalmic goître named above, of which, of course, one or another is often absent or only slightly developed, the acceleration of the pulse is the most constant and usually the earliest. The frequency of the pulse averages 100 to 120, sometimes only 80 or 90, but in other cases even 140 or 160. It is not alike at all times, but has many variations—the symptoms lasting for long periods and coming on in single paroxysms. A very vigorous action of the heart, and as a rule the subjective feeling of palpitation, are usually associated with the acceleration of the pulse. There is a vigorous pulsation of the carotids, and sometimes of the smaller arteries. We do not discover any qualitative changes of the pulse. The pulse is usually quite regular, but arhythmia has been repeatedly observed. In some cases the patients suffer from pronounced angina pectoris.

Physical examination of the heart in many cases shows nothing particular, except an accelerated and violent action of the heart, but we sometimes find, as we can affirm from several cases in our own experience, a manifest hypertrophy of the left ventricle, and also dilatation of the heart, and even actual valvular disease. In the diagnosis of the latter some caution is necessary, because functional heart murmurs are often present in exophthalmic goître.

The goître usually develops somewhat later than the first symptoms in the heart. In many cases it is entirely absent, or present only in a slight degree. The swelling of the thyroid gland is generally only exceptionally very marked. There are sometimes marked variations in it in the course of the same case. The comparative softness of the tumor, the frequent and strong pulsations in it, and the loud vascular murmurs, which are often but not always heard, and which arise in the dilated vessels of the thyroid gland, are characteristic of the goître in Graves's disease. By laying the hand on it we can often feel the thrill and pulsation.

The exophthalmus, the protrusion of the eyeballs from their orbits, is always bilateral, although it is sometimes more marked on one side than on the other. In many cases it is entirely absent; in others it may attain so high a degree that an actual "dislocation of the eyeball" has been described. In the marked degrees of exophthalmus there is often a peculiar staring expression to the countenance. A peculiar symptom, first described by Graefe, is also worthy of mention. On raising and lowering the eyes, the corresponding associated movements of the upper eyelid, which are always present under normal conditions, are absent. This "Graefe symptom" may sometimes be one of the earliest signs of the disease, and may therefore be of diagnostic value; but we must lay stress upon the fact that, in our own experience, this symptom is at any rate only rarely pronounced. Sometimes severe inflammatory processes have been seen in the eye, which are probably to be referred to the impaired protection of the eye by the upper lid as a result of the exophthalmus. Disturbances of the pupils and of accommodation are unknown in exophthalmic goître, but we have ourselves repeatedly observed anomalies in the movements of the eyeball, especially temporary strabismus. We might mention one symptom, which Möbius has first noticed, and which we also have repeatedly but not constantly seen, especially in patients with more marked exophthalmus. It consists in the fact that one eye very soon deviates outward if we have the patient converge the eyes strongly, as in fixation of a near object—"insufficiency of convergence."

Besides the chief symptoms of exophthalmic goître thus far described, we must also mention a list of other symptoms which come to our observation, both in the typical cases and still more often in anomalous cases—the so-called "*formes frustes*" of the French. Among them are some other nervous symptoms, especially a peculiar tremor to which Marie in particular has lately called attention. This tremor affects the whole body, or the extremities alone; it shows at times temporary remissions and exacerbations, and it may be so severe as to form the patient's chief complaint. In a case under our observation marked tremor was one of the first symptoms of the disease. It may become so violent at times that there are even spasmodic twitchings in the extremities and in the muscles of the face. We have repeatedly seen lesser degrees of tremor, especially in the handsi and we regard it, indeed, as quite characteristic. We may also mention among the nervous symptoms which are sometimes present, headache, vertigo, weakness of memory, and sleeplessness. The peculiar nervous anxiety and the irritable disposition of the patient are, however, very frequent, and in fact very characteristic, in many cases of the disease. The anxiety and the haste in all movements, in speaking, etc., often show themselves, even during the physician's examination, in so striking a way that they must be regarded as not unimportant factors in diagnosis. Exophthalmic goître is sometimes complicated with other neuroses—with actual hysteria, epilepsy, chorea, the psychoses, etc. The marked subjective feeling of heat, from which many patients suffer, is probably due to vaso-motor disturbances. Objective elevations of temperature up to 100° or 101·5° (38°–38·8° C.) have also been repeatedly confirmed by others (Eulenburg) and by ourselves. A marked increase of the sweat production, which in rare cases is only unilateral, is often associated with the feeling of heat. On the other hand, one of our patients complained of a constant dryness in the mouth.

Of symptoms which are referred to other organs we must first consider some disturbances on the part of the respiration. The respiration is usually moderately accelerated, and many patients complain of dyspnœa and of a feeling of oppression in the chest. In one case we saw at times deep spasmodic inspirations; in other cases a peculiar dry, "nervous cough" appears. There are also symptoms on the part of the digestive organs. Vomiting is common, and in severe cases it may become so persistent, distressing, and uncontrollable as to constitute one of the chief dangers of the disease. It is sometimes associated with peculiar paroxysmal sero-mucous or even bloody diarrhœa. Icterus may also develop. Finally we must mention certain disturbances in the skin: vitiligo has often been observed, and also diffuse brownish pigmentation of the skin or chloasma-like pigment-spots and urticaria. A very rare but dangerous occurrence, of which we ourselves have seen a striking example, is an apparently spontaneous gangrene of the extremities. In our case, which ended fatally, the gangrene affected the right leg. Not the slightest anomaly could be made out in the vessels at the autopsy. This gangrene in exophthalmic goître recalls decidedly the so-called "spontaneous symmetrical gangrene" (*vide supra*), for which we also must assume a neurotic origin.

The general nutrition of the patient suffers in most cases; a certain degree of anæmia and emaciation is often present. In severe cases, especially in those of rapid development, there is often in a very short time a high degree of emaciation associated with much general weakness. Muscular atrophy frequently develops, chiefly in certain regions—the arm or leg muscles. [The electrical resistance of the body is said to be diminished.—K.]

Pathological Anatomy and Pathogenesis.—Although all the symptoms of exophthalmic goître point to an affection of the nervous system as a cause of the disease, as we see from the symptomatology, the results of pathological investigations are still very meager. There is a class of cases in which changes in the sympathetic, and especially in the lowest cervical ganglion, are said to have been present; but the pathological significance of the discovery is not placed beyond all doubt; and in other cases nothing abnormal at all could be found in the sympathetic. The theory that all the symptoms of exophthalmic goître are derived from a disturbance of the sympathetic also meets many difficulties and contradictions. If we regard only the three cardinal symptoms, we can bring the acceleration of the pulse, and perhaps the exophthalmus, into harmony with the theory of irritation of the sympathetic; but not the goître, which is due to a dilatation of the vessels. The theory of a paralysis of the sympathetic explains the goître, and also the exophthalmus—if we assume as the cause of the latter a dilatation of the vessels in the back of the orbit—but again the acceleration of the pulse remains unexplained. The experiments by Filehne, interesting in themselves, in which symptoms similar to those of exophthalmic goître were produced by dividing the restiform bodies in young rabbits, have had up to the present time no bearing upon human pathology. We must therefore admit that the cause of exophthalmic goître is still completely unknown. Possibly the recent experiments on the significance of the thyroid gland (*vide supra*) may throw some light on the origin of this remarkable affection.

Course and Diagnosis.—The course of the disease is in most cases very chronic, and may extend over years and years, but there are also more acute cases with a rapid development of all the symptoms and a comparatively unfavorable course.

We may often see considerable variation in the intensity of the symptoms. All the symptoms of the disease may often almost wholly disappear, to recur after the lapse of years. In general, the cases beginning in youth give a more unfavorable prognosis than those arising in later years. Complete recoveries have certainly been observed, but they are at all events rare. The disease sometimes terminates fatally with the signs of general marasmus, but more frequently from complications in the heart or lungs. We would note especially, however, that mild and to some extent rudimentary cases of the disease are not rare, and that these in no way endanger life; and even in severe cases we sometimes see marked improvement, or at least an arrest of the disease. The diagnosis of undeveloped cases is difficult at times, since the three cardinal symptoms are not always by any means fully developed. We must then pay careful and especial attention to the other symptoms of the disease, chiefly to the general nervous irritability, the tremor, the subjective feeling of heat, and the tendency to sweating. In well-developed cases, however, the diagnosis is almost always to be made with certainty and without difficulty. The peculiar expression of the face, altered by the emaciation and the exophthalmus, often permits us to recognize the disease at the first glance.

Treatment.—In the first place we must consider the general treatment of the patient. Physical and mental rest, good food, and the avoidance of all stimulants —such as alcohol or strong coffee—and the cautious use of cold-water cures, especially sponging, may cause an actual improvement of the condition. For anæmic patients we prescribe iron, alone or in combination with small doses of arsenic. A course at the springs springs at Franzensbad, Schwalbach, Pyrmont, Elster, and Cudowa, is also sometimes attended with good results. Residence in mountainous regions or by the sea often seems to act still more favorably.

Of other remedies, electricity is first to be mentioned, especially the application of galvanism to the neck—the so-called galvanization of the sympathetic at the inner border of the sterno-mastoid. The slowing of the pulse, which sometimes comes on at once (vagus irritation?), is striking. Vigouroux praises faradization of the sympathetic and of the goître as the best method of treatment. Among internal remedies we may recommend atropine or tincture of belladonna, and ergot or ergotine. We believe we have repeatedly seen good results from the latter. Digitalis has often been prescribed for the palpitation, but usually without any good result. The use of iodine preparations against the goître is also almost always unavailing. With great exophthalmus the eyes must be protected from external injuries. The occasional severe symptoms on the part of the digestive organs (vomiting, diarrhœa) must be treated symptomatically, by ice, opium, and champagne.

In some cases the extirpation of the goître has been practiced, and favorable results have been claimed for it. Should the above-mentioned relation of the morbid symptoms to the functions of the thyroid gland be confirmed, further advance in this direction would perhaps be worthy of trial.

III.—The Diseases of the Spinal Cord.

CHAPTER I.

DISEASES OF THE SPINAL MENINGES.

1. ACUTE INFLAMMATIONS OF THE SPINAL MENINGES.

Ætiology and Pathology.—Isolated acute inflammation of the spinal meninges is very rarely primary, so far as we know, but inflammatory processes in the neighborhood quite frequently involve the meninges, or a spinal meningitis. occurs as one symptom of a general cerebro-spinal meningitis. This latter condition is seen chiefly in the idiopathic, generally epidemic, cerebro-spinal meningitis, which is a specific infectious disease, and has already been described in detail in a previous chapter. A tubercular spinal meningitis is also very often combined with tubercular inflammation of the cerebral meninges, but, since the symptoms of the latter are usually in the foreground of the picture, we will treat of tubercular cerebro-spinal meningitis in the section on diseases of the cerebral meninges. Secondary cerebro-spinal meningitis is sometimes seen in the course of certain other infectious diseases, and is then probably to be regarded as a special localization of the specific poison of the disease. This is the explanation of the occurrence of acute spinal and cerebral meningitis as a sequel of croupous pneumonia, and also of its occurrence in pyæmic and septic diseases, and, very rarely, in typhoid and the acute exanthemata. We must mention, finally, the occurrence of a purulent cerebro-spinal meningitis as a sequel of empyema, pulmonary gangrene, etc., which, although very rare, we have repeatedly noticed. In these cases the infection of the meninges also results from the primary foci of disease, but the channel of infection is not yet exactly known. Perhaps the intercostal nerves are the media of communication.

In all the cases so far mentioned we have chiefly an inflammation of the pia mater, a so-called *lepto-meningitis;* the dura mater is not implicated in the disease at all, or only to a slight degree. The condition is different in those inflammatory processes which gradually invade the meninges from the neighboring parts outside the cord. Thus we very often see circumscribed inflammations on the outer surface of the dura (pachymeningitis) in caries of the vertebræ, and these inflammations often invade the inner surface of the dura, or more rarely reach the pia mater. Acute purulent peripachymeningitis is a very rare disease; it is a purulent inflammation of the connective tissue between the dura mater and the vertebral column, which in almost all cases is of secondary origin. We have seen a very characteristic case of this sort in the course of a puerperal pyæmia. The inflammation had spread from a purulent inflammation of the pelvic cellular tissue, through the foramina of the vertebral canal, and had finally set up a purulent inflammation on the outer surface of the dura, extending up to the cervical cord. We meet with an affection of the pia mater from an extension of the inflammation, chiefly in diseases of the spinal cord, since the pia takes part in the process to a greater or less extent in many cases of myelitis.

We do not know with certainty whether other influences, especially injuries and exposure to cold, can lead directly, as has often been claimed, to inflammation of the spinal meninges.

We need to say but little in regard to the pathological anatomy of acute spinal meningitis. The changes in purulent inflammation of the pia mater have been described in the chapter on epidemic meningitis. Precisely the same conditions are found in the other forms of acute leptomeningitis. The changes in pachymeningitis are completely analogous. The dura mater is traversed by dilated vessels, and therefore is reddened; it is also thickened, and on its internal or external surface (*pachymeningitis interna* or *externa*, or *peripachymeningitis*) there is usually found a purulent or a sero-purulent exudation.

Symptoms.—An accurate distinction between acute inflammations of the pia mater and those of the dura mater can not be made clinically. The symptoms of the disease include the symptoms of any primary disease present, the general symptoms, such as fever, etc., and in addition the necessary consequences which the presence of a disturbance of the meningeal circulation and of the meningeal exudation exerts on the cord and nerve-roots. These consequences are due both to a mechanical compression of the parts named, and often probably to an invasion of the substance of the cord itself by the inflammation. To these is added the frequent combination of spinal symptoms with the symptoms of a co-existing cerebral meningitis.

Those symptoms which occur in acute spinal meningitis, and are especially referred to it, are all of them already known to us from the description of epidemic meningitis (see page 103). Recapitulating them briefly, we may mention chiefly the very severe pain in the back, the great sensitiveness of the vertebral column, and its stiffness. To these may be added usually symptoms of irritation on the part of the nerve-roots: eccentric pains in the trunk and the extremities, hyperæsthesia of the skin and of the deeper parts, symptoms of direct or reflex motor irritation, muscular tension, contractions, etc. The cutaneous and tendon reflexes are often, but not always, much diminished or abolished in consequence of the lesion of the nerve-roots. There are at times disturbances in the passage of urine and fæces. If, in the later course of the disease, there are actual paralysis and anæsthesia, they are usually a sign of a more marked implication of the cord itself.

Diagnosis.—From the symptoms named, we can in many cases make a diagnosis of spinal meningitis. Of course a meningitis is found often enough on the autopsy-table whose symptoms during life were completely obscured by other severe general symptoms, while, on the other hand, with severe constitutional symptoms the symptoms of meningitis may be illusory, as in typhoid or pyæmia. Fuller information as to the seat and the extent of the inflammation is afforded by considering the most painful parts of the vertebral column, the predominance of pain and cutaneous hyperæsthesia in the arms (cervical region) or legs (lumbar region), etc. When the meningitis involves the upper portion of the cord and the medulla there may also be disturbances of respiration, symptoms in the pupils, and anomalies in the innervation of the heart. We can decide as to the form of the meningitis, whether purulent or tubercular, only by a consideration of the history, the other morbid symptoms, and the course of the disease.

Prognosis.—We have seen an undoubted recovery, in severe cases, only in epidemic cerebro-spinal meningitis, and in the sporadic cases of idiopathic meningitis, which are probably identical in ætiology. In all other cases reported with a favorable termination the diagnosis may be doubted, for in general the rule is certain that, in extensive, acute purulent leptomeningitis and pachymeningitis, the prognosis is almost absolutely unfavorable, whether it be secondary to another infectious disease or arise from propagation from some neighboring focus of inflammation. We may, perhaps, make an exception of certain mild, circumscribed cases, which do not come to suppuration, but these are always uncertain in regard to diagnosis.

Treatment.—In regard to treatment we must refer entirely to what has been said under epidemic and tubercular meningitis.

2. Chronic Spinal Leptomeningitis.

Although chronic leptomeningitis (usually wrongly termed chronic spinal meningitis) once played quite a large part in the diagnosis and pathology of diseases of the spinal cord, we must at present assert that its occurrence as an independent disease may justly be doubted. Almost all the cases reported come from a time when the diagnosis of many diseases of the cord itself was still perfectly impossible, and when the thickenings and opacities of the meninges were much more striking at the autopsy than the far more essential changes in the substance of the cord itself, which could not be made out with the naked eye, but only by a careful microscopic examination. At any rate, we may say that a case of primary chronic leptomeningitis, which can be surely and convincingly proven clinically and anatomically, does not exist, and that our present clinical experience also by no means favors the assumption of the existence of mild and curable forms of it. Among many cases of spinal disease we shall scarcely be induced even to assume the probability of the existence of primary chronic meningitis. It goes without saying that we can not dispute the possibility of its occurrence, although even for this we can scarcely find an analogy.

The case is different with secondary chronic leptomeningitis. This, in the first place, in rare cases, is the termination of an acute meningitis. Secondary leptomeningitis may be certainly made out, especially in epidemic meningitis. We also find chronic meningitis frequently as a secondary affection in primary diseases of the cord and the vertebræ. Thus, for example, in old cases of chronic spinal disease, associated with atrophy, such as tabes dorsalis, progressive muscular atrophy, etc., the pia is almost always quite opaque, thickened, and often united to the cord and the dura by very many firm adhesions, while a cloudy sero-gelatinous exudation is found in the meshes of the arachnoid. All these anomalies, however, are of a secondary nature, and have no clinical significance, for the same changes, though rarely so marked, are quite often found in old people, where they are analogous to the equally frequent opacities of the cerebral meninges, the pleuritic adhesions, etc., and where, during life, they have not caused the slightest symptom of spinal disease.

The symptoms which have been set down as characteristic of leptomeningitis correspond precisely to those of acute meningitis, except, of course, that they are relatively less intense, and that the course of the disease is more protracted. Pain and stiffness in the back and neck, abnormal painful sensations and paræsthesia in the extremities, a girdle sensation, and finally paresis, anæsthesia, and vesical disturbances, are the leading features of the type of disease as constructed, in whose fabrication there have been, at any rate, many confusions with myelitis, spondylitis, beginning tabes, multiple neuritis, etc.

It is clear that under such circumstances no special rules for the treatment of chronic spinal meningitis can be given. Given a case, we would try local applications to the vertebral column; painting with iodine; dry, or, exceptionally, in strong patients, wet cups; also protracted tepid baths, 90° to 95° (26°–28° R.), or cautious cold-water treatment; and finally the use of the galvanic current. Of internal remedies, iodide of potassium would be most indicated. We may refer to the description of the treatment of myelitis in regard to all further details.

3. PACHYMENINGITIS CERVICALIS HYPERTROPHICA.

Pachymeningitis cervicalis hypertrophica was first fully described, as a special form of disease, by Charcot in 1871, and later by his pupil Joffroy. Little is known as to its origin; it has been attributed to exposure to cold and the abuse of alcohol.

Anatomically, the disease is characterized by a chronic and often very considerable thickening of the dura, almost always, as it seems, in the cervical portion of the cord, while the pia takes but a comparatively small part in the affection. The dura may attain a thickness of six or seven millimetres, and usually appears composed of a number of concentric layers. Histologically, the hypertrophy consists of a new growth of dense connective tissue. The clinical symptoms of the disease arise from the fact that, first, the penetrating nerve-roots, and later on the cord itself, undergo a considerable mechanical compression. If this is of high degree and persistent, there are, as a necessary result, secondary degenerations of the motor nerves and muscles, and a secondary descending degeneration of the pyramidal tract in the cord.

The clinical symptoms are easily understood from this. The disease almost always begins with severe pain, which shoots from the neck into the occiput and the arms. Besides this, there are paræsthesia and a numb feeling in the arms and hands. Rarely there is an eruption of herpes. All these symptoms depend upon the irritation of the posterior roots.

After this first period of the disease (*période douloureuse* of Charcot) has lasted some two or three months, the second period begins—the period of paralysis. An atrophic paralysis in the upper extremities gradually develops, mainly as a result of the compression of the anterior motor roots. This affects, in a remarkable manner, chiefly the distribution of the ulnar and median nerves, while the distribution of the radial on both sides usually remains free. The hand, therefore, assumes a characteristic position (Fig. 81), as a result of the contracture of the antagonistic extensors. The paralyzed muscles rapidly become atrophic and show a marked reaction of degeneration. In this stage there may also be partial anæsthesia of the skin.

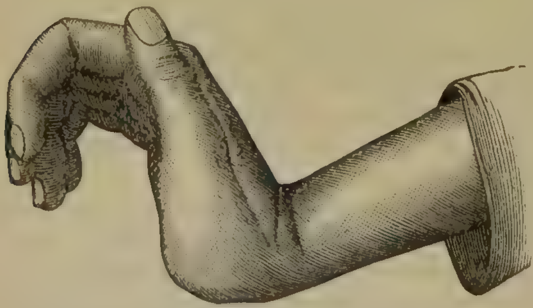

FIG. 81.—Position of the hand in pachymeningitis cervicalis hypertrophica. (From CHARCOT.)

If the compression of the cord advances, the motor fibers for the lower extremities, which pass through the cervical cord, must necessarily at last be involved sympathetically—the third period of the disease. The result of this is a spastic paralysis of the lower extremities—that is, a paresis or paralysis with increased tendon reflexes, but of course without muscular atrophy, because the trophic centers for the muscles of the legs, in the anterior cornua of the lumbar cord, remain perfectly intact. The compression of the cervical cord, however, may finally lead also to anæsthesia of the lower extremities, to paralysis of the bladder, and bedsores, under which symptoms death ensues; but, on the other hand, it must be mentioned that probably cases of recovery, or at least of actual improvement in pachymeningitis cervicalis hypertrophica, may occur even after it has lasted for years.

The diagnosis of the disease is based first upon the fact that the affection begins with pains in the arms, and upon the later appearance of the characteristic paralyses. It may easily be confused with tumors in the cervical cord and with cervical spondylitis. Amyotrophic lateral sclerosis is distinguished, however, by the absence of disturbances of sensibility, by the final appearance of atrophy in the lower extremities, by the bulbar symptoms, and by the fact that the functions of the bladder remain intact.

Treatment can do little directly, and must be chiefly symptomatic. Baths, iodide of potassium, and electricity are most used. Joffroy recommends the use of the hot iron to the neck.

4. HÆMORRHAGE INTO THE SPINAL MENINGES.

(*Hæmatorrhachis. Meningeal Apoplexy. Pachymeningitis spinalis hæmorrhagica interna.*)

Large hæmorrhages into and between the spinal meninges are of rare occurrence. They arise chiefly from traumatic influences, from concussion or fracture of the vertebral column, or from direct injuries of the meninges, such as stabs or gun-shot wounds. In a few cases great physical exertion may also lead to a meningeal apoplexy. Diseases of the vertebræ, caries, and carcinoma, may also lead to a hæmorrhage from the erosion of a vessel. The frequent little meningeal hæmorrhages which appear as a complication of meningitis, in hæmorrhagic diseases, in the course of severe general infectious diseases, septic infections, typhoid, and small-pox, and as a result of severe general convulsions, very rarely have any clinical significance. Finally, it may be mentioned that aneurism of the aorta or its branches may rupture into the vertebral canal.

The clinical symptoms of meningeal hæmorrhage are almost always sudden and "apoplectiform," but are unattended by any disturbance of consciousness. Their intensity depends entirely upon the degree of compression which the nerve-roots and the cord undergo from the effused blood. The symptoms of irritation usually predominate—severe pain in the back, paræsthesia, and neuralgic pains in the extremities; and also symptoms in the motor distribution, tension, tremors, and contractures of the muscles. With large hæmorrhages, symptoms of paralysis, partial anæsthesia, disturbances of the bladder, symptoms of unilateral lesion, etc., may ensue. Therefore the different types of the disease, depending upon the seat of the hæmorrhage, follow the same general rules which are to be considered in judging of the seat of any other affection of the cord (*vide infra*). On the whole, the diagnosis of meningeal hæmorrhage can but rarely be made with any certainty, unless suggestive ætiological factors precede and the symptoms and manner of beginning are especially characteristic.

The course is quite favorable in many cases if the blood be rapidly reabsorbed, but sometimes a permanent disturbance of function is left.

In regard to treatment, complete rest and the energetic local use of ice are chiefly to be recommended, and also local bloodletting—dry cups, or leeches, where there are severe initial symptoms of irritation. If permanent disturbances be left, they should be treated by the ordinary methods—iodide of potassium, baths, and electricity. [When the diagnosis of hæmorrhage into the meninges is reasonably certain, an attempt should be made to relieve the condition by opening the spinal canal and removing the clot.—K.]

We must here speak of **pachymeningitis interna hæmorrhagica** as a special form of disease, which usually occurs at the same time with hæmatoma of the cerebral dura mater (*vide infra*), and is precisely analogous to it in its ætiology and pathological anatomy. Encapsulated collections of blood are found on the inner surface of the dura; these may have quite a considerable circumference, and contain blood already disintegrated, detritus, hæmatoidin crystals, etc., since they are usually of old standing. Besides this, there are also the signs of a fibrinous inflammation—as in the cerebral dura—which, according to the opinion of most observers, is the primary process, so that the hæmorrhages into the newly formed false membrane are secondary. The symptoms of the affection—which has been observed chiefly in the chronic insane (general paralytics) and drunkards—are rarely pronounced, and consist chiefly of pain in the back, stiffness of the vertebræ, and some signs of compression on the part of the nerve-roots and the cord; but we can very rarely make a definite diagnosis.

CHAPTER II.

DISTURBANCES OF CIRCULATION, HÆMORRHAGES, FUNCTIONAL DISTURBANCES, AND TRAUMATIC LESIONS OF THE SPINAL CORD.

1. **Disturbances of Circulation.**—Our knowledge as to the occurrence and as to the clinical significance of pure disturbances of circulation in the spinal cord is very slight. All that is stated in regard to it in the descriptions of the pathology of the spinal cord corresponds much more to theoretical hypotheses than to the actual objective facts.

It goes without saying that a complete anæmia of the spinal cord must destroy its function; this fact is best illustrated by the well-known experiment of Stenson: if we compress the abdominal aorta of an animal, and thus cut off the blood-supply to the lumbar cord almost completely, a paralysis of the posterior portion of the body very rapidly ensues. Some precisely analogous observations have been made in man in the rare cases of obstruction of the aorta by emboli or thrombi. Pronounced spinal symptoms in general anæmia, which may be referred to a co-existing anæmia of the cord, are rare, and at any rate are of much less clinical prominence than the important results of a co-existing cerebral anæmia (*vide infra*). In only a few cases has paraplegia been seen after a great general loss of blood, as after metrorrhagia or intestinal hæmorrhage.

Any statement which may be made as to the occurrence of hyperæmia of the spinal cord is still more uncertain. We do not know whether active hyperæmia of the cord has in itself any clinical significance. The hyperæmia from stasis, in general disturbances of the circulation, in which certainly the spinal cord often takes part, causes no especially marked symptoms.

2. **Hæmorrhage into the Substance of the Spinal Cord—Spinal Apoplexy—Hæmatomyelia.**—Primary hæmorrhage into the spinal cord is as rare as hæmorrhage into the brain is frequent. In some cases it may arise from traumatic influences, but in others we are disposed to assume a primary disease of the spinal vessels. Perhaps there are occasionally aneurismal dilatations in the cord, similar to those found in the smaller vessels in the brain, and these may give rise to hæmorrhage. Finally, the sudden onset of spinal paralysis has been seen after great physical exertion, and this perhaps is due to a spinal apoplexy. The small spinal hæmorrhages which are seen as a complication in tumors of the cord, and in inflammatory affections such as myelitis, epidemic meningitis, etc., and in the general hæmorrhagic diathesis, as in scurvy or severe general infectious diseases, but rarely have any special significance.

Anatomical experience in regard to primary spinal apoplexy is still extremely slight, but the conditions met with do not differ materially, at any rate, from similar processes in other organs. If the hæmorrhage be abundant, we find the substance of the cord destroyed to a great extent. The apoplectic center usually extends principally in the long axis of the cord. The blood is still fluid in fresh cases. Later on it undergoes all those changes which are fully described in the chapter on cerebral apoplexy.

The symptoms of spinal apoplexy must in the first place depend entirely upon the seat and extent of the hæmorrhage. The sudden, apoplectiform beginning of the symptoms is always characteristic. Usually in a very short time a more or less complete paralysis, with a severe pain in the back, comes on; the paralysis being usually in the lower extremities, or rarely in the muscles of the trunk and the upper extremities also. There are usually at the same time anæsthesia and paralysis of the bladder; but in this respect, as also in regard to the condition of the reflexes, there are of course many variations according to the seat of the hæmorrhage. Hæmorrhage into one half of the cord sometimes produces the symptoms of unilateral lesion. We need not go into a precise description of the details, since they will follow of themselves from the general rules in regard to the localization of affections of the cord.

The course of hæmorrhage of the cord may in many cases be comparatively favorable. If the blood be absorbed, and essential paths of conduction be not permanently destroyed, the symptoms of paralysis gradually pass away, and recovery or at least improvement and an arrest of the symptoms follow. In many cases, of course, the severe type of spinal paralysis develops, with bedsores, cystitis, etc., and this, after a longer or shorter time, leads to death.

We must always be very guarded in making a diagnosis of spinal hæmorrhage. We can do so with some probability only when the symptoms begin in a pronounced apoplectiform manner, and when definite ætiological factors are to be made out; but we should never forget that many forms of multiple neuritis (*vide supra*), acute myelitis, and even chronic spinal affections, may also show a remarkably sudden onset, or at least may suddenly become worse. The distinction between genuine spinal apoplexy and meningeal hæmorrhage can hardly ever be made with certainty.

Treatment.—If we have the rare opportunity to be able to interfere at the beginning of the symptoms, we should prescribe a perfectly quiet position in bed, local use of ice, and eventually ergotine. Later on the treatment should be directed according to the methods generally in use in spinal paralysis.

3. **Functional Disturbances.**—In practice we very often see cases where the patient complains of a set of symptoms which are in all probability of spinal origin; but since all the objective signs of a severe spinal affection are entirely absent, and since the whole development and further course of these cases oppose the theory of a coarse anatomical disturbance in the cord, we have a right to regard them as mere "functional disturbances," and thus to express their relation to certain injurious ætiological influences, and their comparative freedom from danger. We know nothing at all definite as to whether the symptoms are based upon unknown disturbances in the nervous mechanism itself, or whether circulatory disturbances, on a basis of abnormal vaso-motor influences, play a part here; but the types of disease met with clinically are very characteristic, are usually easy to be recognized, and, on account of their frequency, have the greatest practical significance. As a rule, the spinal symptoms are joined to certain cerebral symptoms, since the morbid phenomena present are an expression of a disturbance of the whole central nervous system. The type of disease which is briefly described in what follows, for which the names of spinal irritation or spinal neurasthenia are most in use, is often, then, only a complication of a general neurasthenia, to the description of which we must therefore refer for the details.

The ætiology of the disease is often easy to discover. We have to do with patients upon whom one or more of those injurious influences have acted, which have an undoubted influence in the development of almost all the neuroses: severe and persistent emotional excitement, mental and physical overexertion, improper methods of living, toxic influences, such as alcohol and nicotine, sexual excesses, such as onanism, etc. Beside these there is very often a hereditary predisposition, a congenital weak resistance of the nervous system, which is often increased by a poor state of the general nutrition. Finally, a hypochondriacal disposition is of great ætiological significance, as it causes not only an abnormally increased attention to the symptoms, but also an abnormal hyperæsthesia to all subjective sensations. The constant anxiety about the dreaded results of any excesses committed is often much more injurious than the excesses themselves. Hypochondriacal dread, too, usually plays the largest part in the neurasthenic conditions frequent among physicians.

The symptoms of the morbid conditions in question usually begin gradually. The patient begins to complain of weakness and fatigue in walking, and also very often of pains in the back and loins, and not infrequently in the extremities also. In spite of the vivid description which the patients give of their pain, they usually have to admit, if questioned about it closely, that the intensity of the pain is really not very great. Besides the pain there are usually many forms of paræsthesia—numbness, formication, cold feelings, etc. The more the patient knows, or at least believes he knows, of the symptomatology of spinal diseases from reading and from associating with other patients, the more detailed are his complaints. Disturbances of the bladder are usually present only in a slight degree, but still they do occur. They often depend merely upon the disturbance of the involuntary reflex mechanism, due to the added factor of increased voluntary attention. There are very often sexual disturbances, which are usually to be referred to former excesses, especially to onanism or to the hypochondriacal condition of the patient.

If we make a physical examination of the patient we can not discover definite signs of a spinal affection. In some of the cases we find an increased sensitiveness of the vertebræ on pressure, which may be limited to a few definite spots, a symptom to which the name of "spinal irritation" is often given; but we often fail to find this tenderness of the vertebræ. Nothing abnormal is to be discovered in the pupils or the reflexes. The tendon reflexes are sometimes quite lively and sometimes weak. The sensibility is objectively perfectly normal; neither can we discover actual paresis or atrophy of the muscles. Vaso-motor disturbances, however, are often seen: abnormal coldness, pallor or redness of the hands, tendency to sweating, etc. The manifold cerebral symptoms, that are usually present at the same time, will be mentioned in the description of neurasthenia. The condition of the general nutrition in many patients remains unchanged, but, of course, some become pale, thin, and weak.

The diagnosis of functional disturbances of the spinal cord is usually not difficult, as we have said, and it may often be made from the history, from the patient's whole outward behavior, and from the manner of his complaints; but we can not lay too much stress upon the injunction that a careful physical examination should always be made, in order to avoid confusion with an incipient serious disease. We shall repeatedly call attention in what follows to the symptoms which must chiefly be observed for this purpose.

In regard to prognosis and treatment we will refer to what is said in the chapter on neurasthenia.

4. **Traumatic Lesions of the Spinal Cord.***—In spite of the protected position of

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* "Spinal concussion" (commotio spinalis) will be considered later, in the chapter on the "traumatic neuroses."

the spinal cord, it is often the seat of severe acute traumatic lesions. Fractures and dislocations of the vertebræ are the most frequent, and these may give rise to considerable injury of the spinal cord by the dislocation of the vertebræ, or by the projection of a fragment of bone. In many cases the cord is injured, not by the affection of the bones directly, but by the occurrence of traumatic hæmorrhage. Gun-shot wounds of the cord are quite frequent, in which the bullet either penetrates the cord itself or produces injury of the vertebræ and hæmorrhage, which involve the cord indirectly. Stabs and incised wounds of the cord have been repeatedly seen. The point of a knife or sword may penetrate the spinal canal through the intervertebral disks and cause a partial section or at least a contusion of the cord. As in all other traumatic lesions of the cord, a secondary "traumatic inflammation" (*vide infra*), with its results, may be added to the direct injury in such cases.

We need not go into all the details of the pathology and symptomatology of the traumatic lesions of the spinal cord, since the number of special conditions is, of course, almost inexhaustible; but it is usually not particularly difficult to judge of a given case, if we follow the rules which generally obtain in the pathology of the cord. We can easily tell when the cord is involved in injuries in its vicinity by the appearance of pronounced motor and sensory disturbances, which, however, differ very much according to the seat and the extent of the affection of the cord. There is usually at first a pronounced and often complete motor paralysis of the lower extremities, and sometimes, when the cervical vertebræ are injured, of the upper extremities also. There is also anæsthesia, differing very much, of course, in extent and intensity in the different cases; and there is very often vesical and rectal paralysis. In many severe cases the secretion of urine seems at first much diminished or wholly wanting. If the spinal roots be affected there are severe shooting pains and paræsthesia. The reflexes are usually diminished at first, but, later, if the injury be above the reflex arc, they are increased; but if the arc itself be broken, they are permanently absent. We often see in men a more or less complete and persistent erection of the penis, which is probably due to a direct or reflex irritation of the nerves of erection. In injuries of the cervical cord we often see a great and general increase of temperature, up to 110° or 112° (43°–44° C.), especially in severe and rapidly fatal cases; this is of physiological interest, and agrees with the results of experiments. On the other hand, there are also, especially in injuries of the dorsal cord, as it seems, great falls of temperature, down to 90° or 86° (32°–30° C.).

The further course of the affection differs very much. In the worst cases death ensues in a few hours or days. In other cases the patients recover from the first "shock," but permanent paralysis remains, which may sooner or later lead to death from the ensuing sequelæ, cystitis and bedsores; but we often see partial improvement and a cessation of all the symptoms. Although certain functional disturbances remain permanently, life is not further endangered. Finally, in one class of comparatively mild cases, there may be a complete recovery.

The treatment of the primary affections belongs to the domain of surgery; especially any attempt to trephine the vertebral column, in order, if possible, to relieve the existing pressure on the cord by reducing the dislocation of the vertebræ or the splinters of bone. In most cases we have to confine ourselves to putting the patient in a proper position on a water-bed, and guarding as carefully as possible against bedsores and cystitis. Locally, the constant application of ice is most to be recommended. We can expect but little from local blood-letting, inunctions with mercurial ointment, etc. If the first acute stage pass off favorably, the treatment of any paralytic symptoms remaining follows the ordinary rules (baths and electricity).

5. **Diseases of the Spinal Cord after a sudden Reduction of the Atmospheric Pressure [Caisson Disease].**—In bridge-builders and others, who have worked for hours under water in the so-called "caissons," under an external pressure of two or three atmospheres, we sometimes see the appearance of peculiar symptoms after leaving the caisson—that is, on the sudden reduction of the atmospheric pressure. Besides the frequent mild symptoms of pain in the ears and hæmorrhage from the ears, articular and muscular pains in the back and the extremities, slowing of the pulse and vomiting, there are also severe disturbances of motility and sensibility, which point unequivocally to an affection of the spinal cord. Usually the lower half of the body is alone affected. The legs are more or less completely paralyzed, the skin is usually anæsthetic up to the trunk, and there is generally retention of urine. The patient sometimes recovers in a few weeks, but in other cases the condition terminates fatally in a comparatively short time—in a few weeks or months. The first special anatomical investigations in question (Leyden, F. Schultze) gave in such cases a disseminated but extensive affection in the dorsal cord, chiefly in the posterior columns and the posterior portions of the lateral columns. The nervous tissue in the diseased parts was completely destroyed, and instead of it was found detritus and a collection of large, round, finely granular cells (fatty granular cells?). Hæmorrhages into the cord, which might perhaps be expected, have up to the present time not been found.

Nothing definite is known as to the precise process in this form of spinal disease. Leyden suspected that there was a development of gas from the blood which caused a rupture of the surrounding tissue, under the influence of the rapidly diminished barometric pressure, as Hoppe-Seyler and P. Bert have discovered by experiment; but the circumscribed limitation of the affection to the dorsal cord, and the lack of any signs of vascular hæmorrhage, speak against this theory.

The treatment is the same as in acute myelitis.

CHAPTER III.

THE PRESSURE PARALYSES OF THE SPINAL CORD.

(*Slow Compression of the Spinal Cord, especially in Caries and Carcinoma of the Vertebræ.*)

Ætiology.—Many pathological processes which develop in the vicinity of the spinal cord may exert a gradually increasing pressure upon it, and thus, on the one hand, inhibit the conduction of nervous irritation, and, on the other, cause coarse mechanical injuries in the substance of the cord. The seat of such affections is in the first place in the membranes of the spinal cord. In the chapter on meningitis we have already mentioned the compressing action of the masses of inflammatory exudation on the nerve-roots and the cord, and we have learned to recognize, especially in pachymeningitis cervicalis hypertrophica, a characteristic example of a gradually increasing compression of the cervical cord. Precisely similar conditions are found in the rare meningeal tumors, whose special pathology will be described in connection with the tumors of the cord itself.

By far the most frequent pressure paralyses of the spinal cord, and hence the most important practically, are caused by certain diseases of the vertebræ, and first of all by chronic caries of the vertebræ (spondylitis, Pott's disease, spondylarthrocace).* There is no longer any doubt at present that certainly the greatest part, if not all, of the cases of vertebral caries are of tubercular origin, that vertebral caries is a local tuberculosis of the vertebræ. Although these facts were formerly rendered very probable by the histological conditions of the process, and by its frequent relation to other unquestionable tubercular diseases, such as phthisis, miliary tuberculosis, and tubercular meningitis, they have of late been confirmed beyond a doubt by the discovery, which can usually be made, of tubercle bacilli in the cheesy nodules of the vertebral caries. Tubercular spondylitis occurs at almost any age; it is rare only in old people. It often develops in children, but it is almost as frequent in adults. The ætiological significance of injuries, such as a fall or a blow, which are often mentioned by the patients themselves or their parents, is in most cases doubtful; but we can very often succeed in finding ætiological factors for the onset of tubercular disease in general—the tubercular habit, hereditary tendency, or tubercular disease elsewhere, such as phthisis, pleurisy, other affections of the bones, etc.

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* In the kyphoscolioses of the vertebral column, not due to spondylitis, there are practically never any symptoms of compression in the cord, even in very pronounced cases. In these cases the cord manifestly shows quite a great adaptability.

Cancer of the vertebræ, as well as caries, leads to pressure paralyses of the spinal cord; but it is relatively much rarer than caries, it develops chiefly in older persons, and it occurs both as a primary new growth and also as secondary to cancer of other organs—the breast, the stomach, or the œsophagus.

We must mention here briefly, as very rare causes of compression of the spinal cord, aneurism of the aorta, which gradually erodes the vertebræ, echinococci in the vertebral canal, exostoses of the vertebræ, and syphilitic new growths.

Pathological Anatomy.—Vertebral caries is most common in the dorsal portion of the vertebral column (dorsal spondylitis), somewhat rarer in the cervical portion (cervical spondylitis), and rarest in the lumbar portion (lumbar spondylitis) and in the sacrum (sacral spondylitis). It usually extends over several adjacent vertebræ, or more rarely two separate foci of disease are seen. The process itself, the details of which can not be discussed here, probably always begins in the spongy substance of the bodies of the vertebræ. We see here, on section, in incipient cases, roundish, pale reddish, or yellowish nodules, which consist of newly formed fungous tissue—that is, tubercular granulation-tissue. The bony substances become more and more destroyed by the invasion of the new growth, which itself shows the characteristic tendency of all tubercular new growths to cheesy degeneration. Thus there is often an extensive destruction of the bodies of the vertebræ, which later on also involves the vertebral processes, the intervertebral disks, and the other articular connections between the different vertebræ.

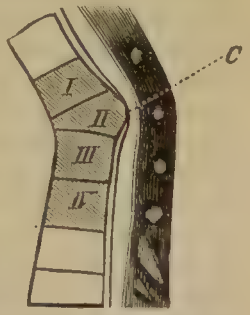

Fig. 82.—Schematic representation of vertebral displacement in spondylitis. The point of compression of the cord, at the level of the second dorsal vertebra, is at *C*.

There are essentially two factors to be considered in regard to the question which chiefly interests us here—that is, in regard to the occurrence of compression of the spinal cord. In the first place, it is clear that the complete or partial destruction of the bodies of one or even more vertebræ, and of their articular connections, can not remain without influence upon the position of the other adjacent vertebræ. In fact, we very often see dislocations of the vertebræ as a result of it, usually by the pushing backward of the partly destroyed vertebra by the movements of the vertebræ above and below the diseased portion on one another (see Fig. 82). There arises, on the one hand, a contraction of the vertebral canal, and with it often a very considerable limitation of the space for the cord; and, on the other hand, that characteristic projection of the spinous processes in the region of the diseased portion of the vertebral column which forms the so-called Pott's boss—the angular kyphosis. In very slight degrees of the disease there is only a slight projection of one or more spinous processes, but in other cases it gradually becomes an extensive deformity of the vertebral column, which strikes us at the first glance. It goes without saying that, under some circumstances, the Pott's boss may be entirely absent in vertebral caries.

The second factor, which is often to be considered in the mechanism of compression of the cord, is the formation of foci of cheesy pus on the posterior surface of the bodies of the vertebræ. Since the inflammatory tubercular new growth involves the periosteum, there are often formed here large collections of cheesy pus, which are situated beneath the periosteum, raise it, and push it out far into the vertebral canal. In other cases, the tubercular new growth still further directly involves the outer surface of the dura, and forms here extensive cheesy masses which of course may also cause a compression of the cord. The inner surface of the dura at the corresponding parts is usually markedly injected, but a direct invasion of the pia by the tubercular process through the dura is rare.

If now a considerable contraction of the vertebral canal have arisen from dislocation of the vertebræ, or from the projection inward of the cheesy purulent masses into the canal, the necessary mechanical consequences in the spinal cord itself are usually to be recognized with ease. The cord appears smaller at the point of compression. If the narrow part correspond to a bend in the vertebral column, we can very often see a marked angle of bending on the anterior surface of the cord. The consistency of the cord at the part affected, the extent of which seldom exceeds a few centimetres, is usually diminished; the cord is soft and may be easily bent. In old cases only do we find the cord itself harder than normal and sclerosed (*vide infra*). It is very remarkable, however, that often, as we have repeatedly seen, marked symptoms of compression may be present during life without finding any coarse mechanical lesion of the cord in the cadaver, so that the cord may show an almost perfectly normal appearance. As in the peripheral nerves, so in the spinal cord, a moderate pressure is manifestly enough to excite a partial break in the conduction, without being at the same time necessarily associated with an actual mechanical destruction of nervous elements. On careful microscopic examination of the cord, we find in such cases, in spite of the existence of a complete paraplegia during life, that most of the nerve-fibers are still completely preserved, and that there are only here and there a few lacunæ, corresponding to single fibers, which have been destroyed. This discovery is especially interesting because it makes us understand the possibility of recovery, even in apparently severe cases (*vide infra*).

Even where we can find considerable histological changes in the spinal cord, however, where the softness of the cord points to a coarse lesion of it, and where the microscope shows the destruction of a great part of the normal tissue at the point of compression, all these changes are merely the necessary results of the purely mechanical pressure on the spinal cord. As we must maintain, against the theory generally received at present, on the ground of many of our own investigations, we have not the slightest reason to refer the occurrence of paralysis in spondylitis to a secondary myelitis. Such a "compression myelitis"—that is, an inflammation of the spinal cord arising from the pressure as such—is to be rejected from general pathological reasons; and the microscopic examination of the cord also shows nothing which points to an inflammation, or which may not be entirely the result of mechanical compression. If we take a portion from the soft place of compression for fresh examination, we find sometimes many, sometimes only a few, granular cells, according to the amount of disintegrated nerve-medulla, the remains of which are taken up by the white blood-corpuscles—the wandering cells. If we make stained cross-sections of the hardened cord, we see under the microscope no signs of vascular changes, of hyperæmia, of accumulation of cells about the vessels, and only exceptionally a little traumatic hæmorrhage; but we do find, besides many still preserved nerve-fibers, other fibers, which are involved in the disintegration or are already destroyed. Very commonly the changes are distributed in the form of nodules. We find groups of greatly swollen axis-cylinders, which have wholly, or almost wholly, lost their medullary sheaths, and in other places we notice already the signs of their disintegration, or the already empty lacunæ in the meshes of the neuroglia. If the destruction of the nervous tissue have advanced to a certain degree, there is in the later stages, as in all analogous processes, a secondary implication of the neuroglia. Now follows an increase of the interstitial connective tissue. Its proliferations, which take the place of the destroyed nervous tissue, seem diffuse—at first loose, but later firm and fibrillary. Thus it happens that in old cases we find nothing at the point of compression but a loss of nerve-fibers in the cord, and instead of it there is a firm fibrous tissue. All the changes mentioned are always much more developed in the white matter of the cord than in the gray.

Finally, after a more protracted compression of the cord we find an ascending and descending secondary degeneration of certain systems of fibers in the cord (*vide infra*).

We need not go more fully into the details of compression of the cord from other causes, since the results, as far as they are of a purely mechanical nature, are precisely the same. In cancer of the vertebræ there may also be dislocations of the vertebral column after the destruction of some of the vertebræ, but usually the compression depends upon the direct growth of the newly formed tissue into the dura. In these cases the compression of the nerve-roots in the intervertebral foramina is also of importance.

Clinical History.—Many cases of spondylitis run their course without involving the cord, or at least involving it only in quite a subordinate fashion. In other cases the symptoms of disease of the vertebræ exist for a long time alone, until at last, slowly or suddenly, the signs of compression of the cord are added to them. Finally, in a third group of cases, the vertebral disease is so latent that only the cord symptoms are prominent in the type of the disease, and the disease of the vertebræ may easily be entirely overlooked.

Usually the symptoms of the developing primary disease, the affection of the vertebræ, precede the appearance of the first cord symptoms by some time. The patient feels a dull pain at a definite part of the spine, which is increased by movements of the trunk, by bending or straightening up. Many patients notice the stiffness of the vertebral column of themselves, and sometimes even a beginning deformity. The first cord symptoms usually consist of painful sensations, which are not confined to the place of the disease, but shoot out approximately along the course of the nerve-paths. These pains, which are due chiefly to an irritation of the nerve-roots caused by the compression, extend, according to the seat of the affection, into the shoulders and arms, into the lateral portions of the trunk, or into the lower extremities. They are sometimes very severe, and then they usually have a pronounced neuralgiform character, or they may be more dull and dragging. Besides the special pains, there are also many forms of paræsthesia, such as formication and cold feelings.

The disturbances of motility begin to appear at the same time with these symptoms or soon after them. A stiffness and weakness arise which impede the gait, usually not in both legs at once, but first in one and then in the other. This paresis increases rapidly or slowly, and it may finally go on to a complete motor paralysis. If the seat of the affection be in the dorsal vertebræ, as it usually is, or if it be in the lumbar vertebræ, the paralysis affects the lower extremities only, and the arms, of course, remain intact; but in cervical spondylitis the arms are usually first and chiefly affected. Only on great compression of the cervical cord is the conduction of the fibers passing through it for the lower extremities impaired, and then there are also disturbances of function in these.

Disturbances of sensibility are often found, independently of the pains and paræsthesiæ above mentioned, but in many cases of pressure paralysis they are present in only a comparatively slight degree. It seems that the sensory nerves resist pressure more than the motor nerves, just as in the pressure paralyses of peripheral nerves; but possibly their position in the gray matter of the posterior cornua protects them better from mechanical attack than is the case, for example, with the motor fibers in the pyramidal tract (see Figs. 66 and 67). The fact is that often, even in complete motor paraplegia, there is little if any diminution of sensibility, and that marked anæsthesia is rare, and is seen only in the last stages of the disease. We find most frequently a slight blunting of sensibility equally to all forms of sensation, especially to the sense of pain. The different parts of the skin not infrequently act differently, so that we find portions with quite normal sensibility, as well as very anæsthetic portions.

The condition of the reflexes is interesting. If the seat of the compression be above the reflex arc, which we must assume to be in the lumbar cord for the reflexes of the lower extremities, we should expect that the reflexes would persist, and in many cases even be increased, corresponding to the decline in the inhibitory influences coming from above. The latter takes place invariably with the tendon reflexes, which are always increased in the lower extremities in the pressure paralyses arising from the cervical or dorsal cord. The increase of the tendon reflexes may reach so great a degree as to show in the lower extremities the pronounced type of spastic paralysis (*vide infra*). The limbs are then found in a rigid tonic extension, they can only with difficulty be flexed passively on account of the muscular resistance, and they show a very vigorous ankle clonus, sometimes degenerating into a general tremor of the leg, and also marked patellar reflex, adductor reflex, etc.; but even in flaccid paraplegia the tendon reflexes may be quite vigorous. In cervical spondylitis the tendon and periosteal reflexes in the arms are also increased sometimes, but in other cases, if the reflex arc be injured, they are absent. Where the seat of the compression is above the lumbar cord, the cutaneous reflexes sometimes show considerable vigor, but their increase is much less frequently as marked as is the increase of the tendon reflexes. In severe pressure paralyses in the dorsal cord the cutaneous reflexes are even frequently diminished. They are probably never entirely absent, but one must understand testing them, and must employ long-continued cutaneous irritation, such as pinching and pricking, in different parts of the skin in order to provoke them.

Trophic disturbances are often found in the paralyzed parts. If there be severe symptoms of sensory irritation there may sometimes be eruptions of herpes, corresponding to the course of the nerves. More frequently there are chronic disturbances in the nutrition of the skin in severe and long-continued cases. It becomes dry, the epidermis scales off, and the nails become brittle. Bedsores form very easily in severe cases on the sacrum, on the buttocks, on the inner side of the knees, and on the heels, especially when the patient has insufficient care. The muscles retain their normal volume and their normal electrical excitability in many cases as long as their trophic center remains uninjured; but sometimes, even when the point of compression is above the lumbar cord, there is a great atrophy of the muscles of the legs, although the electrical reaction of the nerves is normal, or at most a little reduced in quantity. If the lesion involve the lumbar cord itself, or the fibers of the cauda equina, in caries of the sacrum, there must, of course, be an atrophic paralysis in the legs, with reaction of degeneration. There may also be an atrophic paralysis in the arms in cervical spondylitis.

Disturbances of the rectum and bladder occur in almost all severe cases of pressure paralysis. The difficulty of micturition is often an early symptom of the disease; later on there is complete retention of urine, and, in the more advanced stages of the disease, there is usually incontinence. With this the danger of the development of cystitis becomes very great. The bowels are usually constipated, but sometimes there is also incontinence of fæces.

Thus we see, under some circumstances, in compression of the cord the whole group of symptoms arising which are the necessary consequence of the break in conduction in the spinal cord, and which we shall likewise meet again in various other spinal affections, especially in myelitis and in tumors. The intensity and selection of the symptoms must, of course, vary very much in the different cases. If the compression be quite slight, there are only mild symptoms of sensory irritation and slight paresis. One of the earliest and most constant signs of a compression of the cord, in the dorsal or cervical region, is a decided increase of the patellar reflex. We sometimes find it at a time when there is scarcely a single other cord symptom present. If the compression increases, the paresis becomes more marked, the disturbance of sensibility is greater, and vesical disturbances arise, until finally the complete type of an entire transverse interruption of conduction in the cord is developed; but the latter is but rarely the case, since, as we have said, the conduction of sensory impressions is usually not wholly abolished. The time required for the development of the symptoms of spinal compression differs very much. They sometimes attain a considerable height in a short time, and sometimes they develop only after a course of months. Variations in the intensity of the symptoms are frequent, and they point perhaps to a corresponding variation in the severity of the compression.

In regard to the result of pressure paralyses, it, of course, depends first upon the nature of the primary disease. In tumors, especially in cancer of the vertebræ, recovery is not to be thought of, but spondylitic processes without doubt can recover, which is by no means in contradiction to their character as a local tubercular process. In this connection the fact is of great practical importance that even the pressure paralyses may be completely restored, as far as the cause of the compression can be removed by the absorption of inflammatory and tubercular new growths, so that a complete and permanent recovery may take place even after the paralysis has lasted for some months, or a year, or even a year and a half. Such recoveries have been seen in great numbers by others and also by ourselves.

Although, according to this, the prognosis in a part of the cases of pressure paralysis from spondylitis is comparatively good, still many other cases terminate unfavorably. The cause of this lies either in the occurrence of dangerous sequelæ of the paralysis, such as bedsores, cystitis, or pyelo-nephritis, with fever and increasing general weakness; or in the development of other tubercular diseases, especially phthisis, or more rarely miliary tuberculosis or tubercular meningitis, which prove fatal.

Diagnosis.—The frequency of the pressure paralyses of the spinal cord admonishes us to examine the vertebral column carefully in every case of spinal disease, especially if the case can not be put under one of the special types of systemic diseases (*vide infra*). We should look especially for the stiffness of special parts of the vertebral column on movements of the head or trunk, and also for the pronounced tenderness of single vertebræ to pressure, and, finally, as a most important and most certain sign, for the deformity of the vertebral column, the marked projection of a single spinous process, or the formation of an evident angular kyphosis. If we find such a Pott's boss the diagnosis is easy, and we can then at any rate refer the existing cord symptoms to a compression of the cord caused by a disease of the vertebræ.

The diagnosis is more difficult if the signs of an affection of the vertebræ be not evident. It must once for all be stated that vertebral caries does not invariably and necessarily result in a manifest Pott's boss, and that even the tenderness of the vertebræ to pressure is sometimes very slight in spondylitis. In such cases the examination of the vertebral column must be repeated more frequently, since even slight anomalies obtain a diagnostic value if constantly present; and the whole course of the disease is also to be considered. The most characteristic features of a compression of the cord are its beginning with symptoms of sensory irritation, the preponderance of symptoms of motor paralysis with comparatively little disturbance of sensibility, and finally the frequent asymmetry of the symptoms on the two sides, which may even recall the type of the so-called "unilateral lesion" of the spinal cord (*vide infra*). Sometimes the cause of the spinal symptoms is at first obscure, and later on in the disease a marked anomaly of the vertebral column develops.

If the diagnosis of an affection of the vertebræ be certain, the next question is as to the nature of it, especially whether we have to do with a spondylitis, or with a cancer of the vertebræ. Since spondylitis is by far the more frequent disease, we must always think of that first, especially in young people, and where we have the formation of a pronounced angular kyphosis. In cancer of the vertebræ the coarser changes in the form of the vertebral column are generally less marked. This develops usually in older people, after the age of forty, and is manifested by the great intensity of the initial symptoms of sensory irritation. The "*paraplegia dolorosa*," the paralysis of the lower extremities associated with severe pains, is the most characteristic symptom of cancer of the vertebræ. The discovery of a primary nodule of cancer, as in the breast, or, as we ourselves have seen, the appearance of a swelling of the inguinal glands, may serve to support the diagnosis. Finally, a certain stress is to be laid on the well-known general habit of patients with cancer, and on the peculiar cancerous cachexia.

The place of compression is, in the majority of cases, to be recognized by the evident localization of the disease of the vertebræ. In other respects the same rules hold for localization which we shall discuss more fully in the description of myelitis in the following chapter.

Treatment.—In regard to the special treatment of spondylitis, especially the orthopædic treatment, we must refer to the text-books of surgery. In general, we have not thus far gained the impression that a particularly favorable influence can be exerted on the symptoms of spinal compression by contrivances for extension of the vertebral column. These are often injudicious where there is paraplegia, since they increase the pain and make it harder to guard against bedsores. We would not deny, however, that in many cases certain supports for the vertebral column, and contrivances for extension, may be used to advantage. Permanent rest in bed is, at any rate, always of the greatest importance. Local applications to the vertebral column are much used—dry cups, painting with iodine, and especially the hot iron. The use of the latter in spondylitis has even to-day warm advocates, and, in fact, deserves to be tried, the procedure with Paquelin's thermocautery being especially easy; we make some three or four eschars on each side of the diseased vertebræ.

Among other remedies we may mention stabile galvanization at the point of pressure, and the electrical treatment of the paralyzed extremities; also the use of baths, especially salt baths, and, finally, the internal use of compounds of iodine—iodide of potassium and iodide of iron. In regard to the symptomatic treatment we will refer to the following chapter.

[In a few cases great benefit has been derived from trephining the vertebræ and removing the inflammatory products which have compressed the cord. In some cases a patient who was completely paraplegic has regained considerable use of the legs. It is obvious that the operation should not be too long delayed after symptoms of pressure have once developed.—K.]

CHAPTER IV.

ACUTE AND CHRONIC MYELITIS.

(*Diffuse Myelitis. Transverse Myelitis.*)

Preliminary Remarks.—The pathological processes in the spinal cord known to us at the present time may be divided into two groups. In the first group we find this peculiarity, that the pathological changes are confined, with a remarkable constancy, to certain definite parts of the spinal cord, so that consequently the clinical symptoms of the disease may be quite exactly defined. To this group belongs the disease known as anterior poliomyelitis (πολιός, gray), which is localized almost exclusively in the anterior cornua of the gray matter of the spinal cord, and also a class of affections, such as tabes dorsalis, amyotrophic lateral sclerosis, etc., in which perfectly definite fasciculi in the cord are diseased. From the comparison of the anatomical lesions in these cases with our other knowledge as to the structure and functions of the spinal cord, it has been shown that the diseased portions occupy a distinct position, even in their anatomical and physiological relations. Hence we are justified in terming these affections of the cord systemic diseases. We can not at present give a correct explanation of the remarkable fact that such isolated diseases may occur in parts of the cord which have perfectly definite functions, in "systems of fibers." We must imagine that the factors which cause the disease, in such cases, do not exert their influence upon the whole cord, but only upon the fibers and cells of a definite system; an idea which finds a fitting analogy in the action of many poisons, such as curare, strychnine, lead, etc.

Besides the systemic diseases there is a second group of affections of the cord in which there is not, by any means, such a limitation of the process to definite portions of the cord. In these cases the disease extends more or less widely over the cross-section or the length of the cord, and forms either one large focus, or several single smaller foci, separate from one another. To this group, to the unsystemic, diffuse diseases of the spinal cord, belong the hæmorrhages and traumatic lesions already described, and the new growths, the acute and chronic "inflammations" of the cord (diffuse myelitis), multiple sclerosis, etc.

Since in the diffuse diseases of the spinal cord all those portions may be affected whose isolated affections form the systemic diseases, of course all the clinical symptoms of the latter may also be found in the diffuse affections; for the individual symptoms of spinal disease, as such, never depend upon the form of the pathological process, but only upon its situation, and upon the irritation or interruption of conduction in certain nerve-tracts caused by it. The diagnosis of spinal diseases, therefore, is, in the first place, always a topical diagnosis. We seek to recognize, from the functional disturbances prominent in the different cases, the place in the cord in which the affection must be situated, which has as a consequence these disturbances. By comparing all the existing morbid symptoms, and by attending to the functions which are still normal, we can decide whether the affection is limited in a systemic fashion to a special physiological region, or whether it extends in a diffuse, irregular fashion over a greater portion of the cord. In the former case we usually have no difficulty in finding a connection with the different well-known typical forms of disease; in the latter case we can at least decide the main point, as to the extent and seat of the disease, and then from the whole course and the combination of the morbid symptoms, we can also draw our conclusions, as far as it is possible, as to the form of the affection.

After these general remarks we will pass on to the description of myelitis.

Ætiology.—Hardly anything certain is known as to the causes of diffuse myelitis, in the same way as little is known in regard to the ætiology of diseases of the spinal cord in general. We often see the disease develop in men previously healthy, without being able to discover any influence which may act as a cause of the disease. In those cases, too, where we may at least ascribe a possible ætiological significance to certain conditions, we are still completely in the dark as to the manner in which they act.

The factors which seem to be most frequently related to the development of myelitis are as follows: Exposure to cold, especially repeated wettings, and working in the open air under unfavorable conditions; bodily fatigue and overexertion, especially when combined with the factors first named, as in the hardships of war, etc. The ætiological significance of profound emotions and sexual excesses, which formerly were often mentioned in the ætiology of myelitis, is extremely doubtful as far as the development of anatomical changes in the cord is concerned.

The occasional appearance of spinal disease after certain acute infectious diseases, such as typhoid, small-pox, or puerperal affections, favors the possibility of infectious causes; but these cases are very rare in comparison with the great number of cases of primary myelitis, and their anatomy is also but little known. Syphilis probably has a greater importance, although our knowledge on this point is not so broad that we can give a definite description of "syphilis of the spinal cord"; but, at any rate, it is quite striking that in the history of patients with diffuse myelitis, especially in myelitis in the upper dorsal region, we quite often get, as it seems to us, an account of a former syphilitic infection. Of course, in any individual case, the actual connection between the two diseases can hardly ever be proven with certainty.

It is proven with regard to purulent spinal meningitis that inflammations of neighboring organs may invade the spinal cord. In most of the other cases which are usually cited in regard to this point, we have to do with confusions between lesions of the cord from mechanical pressure and actual myelitis, as we have explained in detail in the previous chapter on compression of the cord. Hence we consider it unjustifiable to speak of a "traumatic myelitis" except in very rare cases. If spinal symptoms develop after an injury, we usually have to do either with the type of symptoms described above under spinal concussion, or with genuine traumatic lesions of the vertebræ, or sometimes perhaps with traumatic hæmorrhages, etc., which always excite disturbances in the functions of the spinal cord by mechanical conditions alone. Finally, in regard to the theory of an "ascending neuritis"—that is, the supposed extension of an inflammation from the nerves to the cord—it is a hypothesis that is still very much in need of further confirmation.

Pathological Anatomy.—Macroscopic examination of the cord in its fresh condition shows no marked pathological changes except in a small number of cases. At the first glance, the spinal cord often seems almost completely normal, even if there have been severe spinal symptoms during life, and sometimes the opacities and adhesions of the pia, which often strike us at first, have no practical importance. If we test the consistency of the cord carefully by touching it, of course a change in it often strikes the practiced examiner, since the cord over a definite extent is either softer and more flexible, or, on the other hand, harder and firmer. If we now make a number of cross-sections through the cord, we notice that the substance of the cord rises up more on section, that the outline of the gray matter is less distinct, and especially that the white matter is of a reddish-gray color, and that sometimes there is also a reddish, hyperæmic coloring of the gray matter. In some cases we can recognize small capillary hæmorrhages with the naked eye, but the macroscopic examination of the fresh cord is never sufficient for the precise determination of the extent and intensity of the disease.

The changes are much more plainly visible if we harden the cord in chromic acid, or Müller's fluid,* for at least eight or ten weeks. All the normal parts of the white matter of the cord assume a dark-green color from the acid, which is really due to the staining of the medullary sheaths. The diseased portions, in which the medullary sheaths are mainly if not entirely absent, are thus often very sharply distinguished from the healthy, dark-green portions. Since similar differences in color between healthy and diseased tissue are also noticed in the gray matter, although less sharply defined, the cross-section of the cord, well hardened in chromic acid, usually gives quite a correct idea of the extent of the disease.

We obtain more precise disclosures, however, as to the form of the anatomical changes by microscopical examination. When made on the fresh, unhardened cord, it affords little information. The presence of numerous granular cells (*vide infra*) in fresh teased-out preparations is the only thing that is important, since they show with certainty the existence of a pathological change. If, however, we make fine cross-sections of the hardened cord, and stain them with carmine or some similar staining fluid, even the naked eye notices at first a marked difference between the diseased and the healthy tissue, since the former, which is almost always richer in connective tissue, has a much darker staining, and thus is distinguished from the brighter normal tissue. The microscopic examination now shows that in the diseased parts the normal nerve-tissue has been almost wholly or at least partly destroyed. Only occasionally do we see nerve-fibers of normal appearance remaining here and there. In other places the fibers that are still visible are smaller and atrophied, and the axis-cylinders have in part lost their medullary sheaths, or are swollen. The changes in the ganglion-cells are harder to follow, but in more advanced cases they also show marked signs of destruction; they are contracted, rounder, and have lost their processes. The increase of the connective tissue corresponds to the destruction of the nerve-substance. The meshes of the neuroglia extend and swell, so that the space formed by the destruction of the nerve-tissue is in great measure taken up by connective tissue. The older the process, the firmer and more fibrous is the connective tissue. The nuclei of the neuroglia increase in number, and we often find a very great increase in those peculiar, flat, connective-tissue cells with many processes, first described by Deiters, and named after him—the so-called "Deiters' spider-cells." The fatty granular cells are also easily recognized in hardened preparations, so long as they are not treated with alcohol. They lie in the interstices between the meshes of the neuroglia, and are especially numerous about the vessels. They are to be regarded either as white blood-corpuscles, or as endothelial cells from the sheaths of the vessels, which have taken up the fat from the disintegrated nerve-substance. If, therefore, the process be still fresh, or if it be still advancing, the fatty granular globules are to be met with in great numbers, while in old, sclerosed nodules, only a few of them, or scarcely any, are to be found. The changes in the vessels are usually very striking. They are often dilated and congested. Here and there

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[* The formula for Müller's fluid is as follows: Two and a half parts of potassic bichromate, one part of sodic sulphate, and one hundred parts of water.—TRANS.]

there may be hæmorrhages. The vascular walls are thickened, especially in old cases, and sometimes have become peculiarly homogeneous—"hyaline degeneration"; and a large accumulation of nuclei may be found about the vessels. The so-called corpora amylacea are sometimes present in great numbers, and sometimes they are only scanty. Their significance and their genesis are still unknown.

The extent of the whole process varies very much in different cases. We usually find one main focus of myelitis, which extends in a diffuse manner over the greatest part of the transverse section of the spinal cord, and may reach upward and downward for a space of five to ten centimetres or more. The dorsal portion of the cord is most frequently affected (dorsal myelitis), the upper half being usually most involved, but in some cases the lower half is chiefly affected. Nearly the whole of the dorsal cord is often the seat of a diffuse inflammatory affection, which, of course, differs in extent at different levels. In other cases the chief focus of disease is in the cervical cord (cervical myelitis), while it is most rare in the lumbar cord (lumbar myelitis). We often find small, distinct foci in the vicinity of the main focus. In all severe cases there develops later on a systemic ascending and descending secondary degeneration (*vide infra*).

We have intentionally avoided any division of the process into different stages, because, according to our present knowledge, this can be only artificial. As a general rule, the cases where the cord is soft and has more of a reddish-gray color, where the fatty granular cells are still abundant, and the meshes of the neuroglia are not yet fibrous, may be considered as belonging to the comparatively more acute and fresher stages; while in the older cases the cord has become firmer, "sclerosed," in the affected part, through the formation of a denser fibrillary connective tissue, and it has more of a gray appearance; but we can not draw a sharp distinction between acute and chronic myelitis in regard to their pathological anatomy. Genuine transverse myelitis always shows a chronic course, and many cases deserve the name of "acute myelitis" in their clinical aspect only in so far as the beginning of the morbid symptoms is acute and rapid. We may entirely disregard actual abscess of the cord, because it is so rare that it very seldom comes into question as an independent disease. It is still undecided whether there is a softening of the cord analogous to the foci of softening in the brain—that is, as a result of an obstruction of the vessels by a thrombus or embolus. At any rate, an actual softening of the cord is quite rare—that is, a change of the substance of the cord into a soft pulp, which contains nothing but the remains of the nerve-tissue and some fatty granular cells. We have seen ourselves only one such case, in the lower dorsal region, which lasted two years as a chronic transverse myelitis, and ended fatally.

The Individual Symptoms of Myelitis.—The course of transverse myelitis differs so much in the different cases that it is impossible to give a picture of the disease which will be generally applicable. According as one or another part of the cord is involved, the clinical symptoms will affect chiefly the sensibility or the motility, the trophic functions or the reflexes, and will be present in the upper or the lower extremities, or in both at once. The following description will, therefore, be devoted first to the single symptoms, and will give the inferences which, according to the present state of our knowledge, may be drawn from their presence as to the seat and the extent of the anatomical process.

1. SYMPTOMS OF MOTOR PARALYSIS are not only the chief symptoms, as a rule, in well-developed myelitis, but are often the first sign of the beginning of the disease. The patient feels at first only a slight weakness in one or both legs; he gets tired more easily in walking, and begins to "drag" his legs after him. The motor weakness gradually becomes greater and increases to complete paralysis. The

patient is then bed-ridden, and, finally, can not make the least active movement with his legs. The symptoms of paralysis in the arms are analogous.

Since the chief paths for the conduction of voluntary motion are situated, as we have seen, in the lateral columns of the spinal cord, and especially in the lateral pyramidal tract, we conclude, in every spinal disease where symptoms of paralysis are present, that there is an interruption of this tract—that is, an implication of the posterior portions of the lateral columns. Since in transverse myelitis the whole cross-section of the cord is more or less involved, the paralysis also extends to the two halves of the body: motor paraplegia is the characteristic form of paralysis for transverse myelitis. Paraplegia of the lower extremities may of course arise wherever the myelitis is situated, whether in the lumbar, dorsal, or cervical region; but the upper extremities necessarily remain entirely free in every dorsal or lumbar myelitis. The occurrence of paretic symptoms here, and the final development of a brachial paraplegia, point with certainty to an implication of the cervical region, to a cervical myelitis. If the symptoms of paralysis are not alike in the two corresponding extremities, but are more marked on one side than on the other, the anatomical affection must also be more intense on that side of the cord than on the opposite side.

2. Symptoms of Motor Irritation of various sorts are often seen, both at the beginning and during the whole course of myelitis. Single twitchings come on spontaneously in the limbs, which are at the same time paralyzed, or at least paretic, and these twitchings are short and rapid or slow and persistent. The thighs are drawn up on the abdomen, or there are severe spasms of the extensors. The interpretation of these symptoms is not always easy. It is often particularly hard to decide whether they are the result of a direct irritation of motor fibers in the cord, or whether they represent reflexes (*vide infra*). The value of the symptoms of motor irritation for the localization of the disease is accordingly slight, but, of course, in these cases we must chiefly consider the motor tracts in the lateral columns.

Ataxia and intention tremor are comparatively rare, but they are most frequent in the upper extremities. They are also seen in the stage of convalescence in acute cases.

3. Disturbances of Sensibility.—The disturbances of sensibility usually appear to a marked degree only in the later stages of the disease. At the outset we usually notice merely mild symptoms of sensory irritation, such as formication, prickling, numbness, a woolly feeling, etc., while severe pain is hardly ever present in transverse myelitis, and hence it always points to some affection of the vertebræ or the meninges. Slight diminution of sensibility is often to be made out early on careful examination, but, in many cases, the sensibility remains for a long time wholly, or almost wholly, intact, either because the localization of the disease spares the sensory portions of the cord, or because the sensory paths of conduction are more resistant, or can act vicariously for one another to a higher degree. In the further course of the disease, however, there are almost always more marked disturbances of sensibility: at first a simple diminution in the sensitiveness of the skin, sometimes partial paralyses of sensation, analgesia, paralysis of the sense of pressure, and finally frequently a complete anæsthesia. On the other hand, we see in many cases a striking hyperæsthesia to painful stimuli, such as a pin-prick.

From the presence of marked disturbances of sensibility we can conclude with certainty that there is an affection of the posterior columns, and especially of the posterior cornua of the gray matter. With marked anæsthesia the latter are always involved. It is still very doubtful whether the statement made by Schiff, that the conduction of painful sensations is chiefly in the gray matter, and the conduction of tactile sensations is chiefly in the white matter, holds in man. The pathological facts, as we have said before, also give no support at all to the theory that there are sensory fibers in the lateral columns in man.

The disturbance of sensibility gives important service in estimating the height at which the affection in the cord is situated. If we search on the trunk for the line where the cutaneous sensibility becomes normal, we may place the upper boundary of the myelitis, as far as it disturbs the sensibility, at approximately the same level. In myelitis in the lumbar region the disturbance of sensibility reaches to the umbilicus, or even a little higher; in myelitis in the lower dorsal region it reaches about to the lower end of the sternum; in myelitis in the upper dorsal region to the level of the axillæ; and in cervical myelitis the sensibility of the upper extremities is also impaired, but complete anæsthesia is very rare.

4. Cutaneous Reflexes.—As is well known, the reflex arcs in the cord are found at about the same level as the centripetal sensory and the centrifugal motor fibers. They are also connected with fibers which come from above, and to which must be ascribed the property of reflex inhibition. If these fibers above the reflex arc are put into a state of irritation, the reflex is thereby impaired; but if the conduction be broken in these fibers, the reflex activity appears increased, the reflex comes on at a weaker irritation, and the contraction is more vigorous. If the reflex arc itself be broken at any point, the reflex must disappear.

The data from the examination of the patient may generally be harmonized with this scheme, although, of course, the reality probably shows more complicated conditions. In extensive lumbar myelitis, by which the reflex path in the lumbar cord is broken, the cutaneous reflexes in the lower extremities must be diminished or absent. In these cases the loss of sensibility runs about parallel to the diminution of the reflexes. In dorsal and cervical myelitis, however, the reflex arc in the lumbar cord remains unimpaired, but the conduction of sensory impressions to the brain may very well be interrupted. In these cases the cutaneous reflexes are retained, even when there is anæsthesia; or, if the reflex inhibitory influences be removed, they are decidedly increased. The cutaneous reflexes in the legs, however, may be diminished, even in disease above the lumbar cord, in which case we must imagine a loss of irritability in the fibers which take part in the reflex, or an irritation of the reflex inhibitory fibers. The cremaster reflex has its reflex arc about at the point of exit of the first lumbar nerves; diseases of the cord at this point must therefore, under some circumstances, cause a disappearance of the reflex. Of the abdominal reflexes the upper, epigastric, corresponds about to the level of the fourth to the seventh dorsal nerves, and the lower abdominal reflex proper to the lower portion of the dorsal cord.

5. Tendon Reflexes.—The same rules generally hold in judging of the tendon reflexes as are to be considered in judging of the condition of the cutaneous reflexes. We know comparatively little of the course of the reflex arc of the patellar reflex in the lumbar cord. It lies about at the levels of exit of the second to the fourth lumbar nerves. We know, also, that the reflex fails as soon as the middle part of the posterior columns (see the chapter on tabes dorsalis) or the anterior cornua of the gray matter of the lumbar cord are much diseased. The Achilles' tendon reflex, or the ankle clonus, has its reflex arc at the level of the first sacral nerves. It is always absent in extensive disease of the posterior columns and of the gray matter in the corresponding portion of the lumbar cord, so that, besides the other symptoms, the absence of the tendon reflexes in the lower extremities is one of the most important points for the diagnosis of a myelitis of the lumbar cord. In almost all inflammations above the lumbar cord—that is, in dorsal and cervical myelitis—there is, however, a very decided increase of the

tendon reflexes, the result, as we must suppose, of the loss of the reflex inhibitory influences. We have a certain right to assume that the fibers which influence the condition of the tendon reflexes run chiefly in the lateral columns of the spinal cord, but that they are not identical with the fibers of the lateral pyramidal tracts which serve for voluntary motion (see the chapter on spastic spinal paralysis). We may therefore assert that, with a considerable increase of the tendon reflexes in the lower extremities, the seat of the myelitis must be above the lumbar cord—that is, in the cervical or dorsal cord—and that in these cases we have to suppose that the lateral columns are chiefly implicated. In cervical myelitis the tendon reflexes in the upper extremities are often considerably increased.

We have already said, on page 544 *et seq.*, what is necessary in regard to the different signs of the increased tendon reflexes, the exaggerated patellar reflex, ankle clonus, the periosteal reflexes, etc. The peculiar character which the paralysis of the legs assumes from a considerable increase of the tendon reflexes at the same time will be described more fully in the chapter on "spastic spinal paralysis" (*vide infra*).

6. DISTURBANCES IN THE BLADDER AND RECTUM.—Disturbances in micturition are one of the commonest symptoms of myelitis. The first manifestation is usually a difficulty in micturition; the patient has to strain and to wait longer before urinating. There may finally be a complete retention of urine from paralysis of the detrusor urinæ. In the later stages of the disease, however, there is usually a paralysis of the sphincter vesicæ, and consequently incontinence of urine. The disturbances of the bladder give no points for the localization of myelitis, since they may occur with disease at any level of the spinal cord; but we believe we are right in assuming that they always permit us to decide that the posterior columns of the cord are involved.

The clinical significance of disturbances of the bladder in myelitis, and in many other diseases of the cord, apart from the great distress and discomfort for the patient, lies in the fact that they very often—almost always in severe cases—give rise to the development of cystitis. In retention of urine the use of the catheter, by which inflammatory irritants are often brought into the bladder, in spite of all attempts at disinfection, leads to decomposition of the urine and to cystitis; but where there is also incontinence, the imperfect closure of the sphincter and the constant presence of stagnating and decomposing urine in the urethra are the causes of the entrance of these irritants into the bladder. If cystitis have developed, it may be followed under some circumstances by pyelitis and purulent pyelo-nephritis (*vide infra*), which conditions are often the immediate cause of death from the sequelæ connected with them, such as fever, which is sometimes associated with chills, general weakness, and emaciation.

Defecation is also disturbed in many cases of myelitis. There is usually constipation at first, which may depend either upon weakness of the intestinal peristalsis, or upon paresis of the abdominal muscles. Sometimes the constipation reaches such a degree that the bowels move only at intervals of one or two weeks. In many severe cases there is finally incontinence of fæces, as a result of paralysis of the sphincter ani. We can give no details as to the localization of the nerve-tracts in the cord which take part in defecation.

We have yet to note that micturition and defecation are often aroused reflexly in an abnormal fashion where there is increased reflex irritability. On irritation of the skin over the thighs, the perineum, the gluteal region, etc., there is often an involuntary contraction of the bladder, associated with loss of urine.

In conclusion, we may mention, as an addendum, that the sexual functions are often considerably disturbed in many cases of myelitis, and finally may be wholly lost. The tracts involved here lie probably chiefly in the upper lumbar cord, but their precise localization (posterior columns ?) is still unknown.

7. Trophic Disturbances.—The trophic condition of the paralyzed muscles affords extremely important points for diagnosis. In cervical and dorsal myelitis the trophic centers in the lumbar cord for the muscles of the legs remain intact; the paralyzed muscles, therefore, retain essentially their normal volume, and especially their normal electrical excitability. Even in such cases the muscles are sometimes flabbier and of lesser girth than under normal conditions, but this depends partly on the decline in the general nutrition, and partly perhaps on the lack of movement, the "atrophy of inactivity." Only occasionally do we find more marked muscular atrophy, but it is of a simple character and not degenerative, and hence without reaction of degeneration; but if we find in myelitis a genuine degenerative atrophy, with reaction of degeneration in the muscles of the lower extremities, we can from this draw a definite conclusion that the anterior gray cornua or the fibers of the anterior roots in the lumbar cord are affected (see page 538). In an analogous fashion degenerative atrophy with reaction of degeneration in the muscles of the upper extremities points to an affection of the anterior gray matter in the cervical cord.

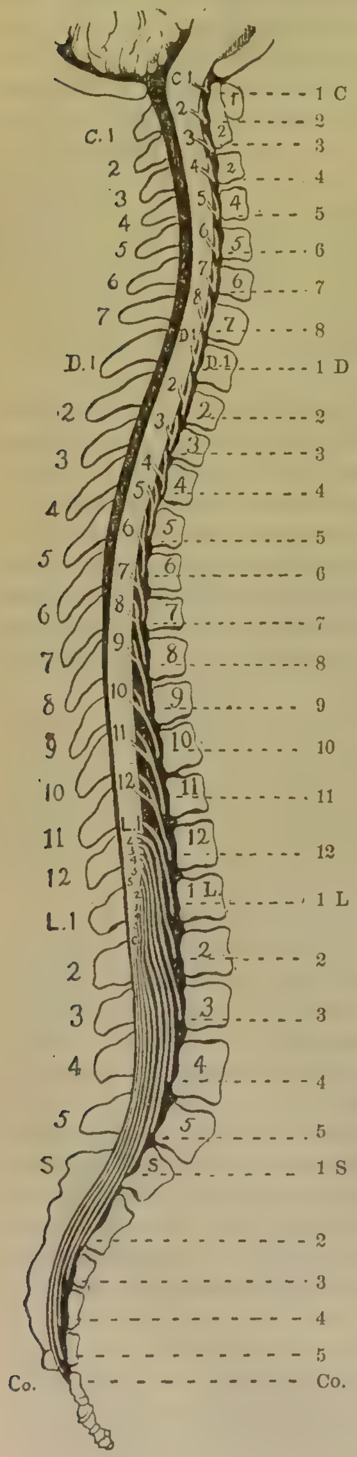

Fig. 83.—A diagram designed to show the relations of the vertebræ to the spinal segments.

Trophic disturbances in the skin are also frequent, but they have no definite diagnostic significance. We often find the skin dry, hard, with a scaly epidermis, and the nails thickened and brittle. Exceptionally there are eruptions of herpes, urticaria, etc. Vaso-motor disturbances also occur. Sometimes the paralyzed extremities show a mottled, cyanotic reddening, and feel cold. Slight œdema is quite frequently present in the paralyzed parts. Disturbances of the sweat secretion are not infrequent. We find either that it is absent or that there is a great increase in it, so that the paralyzed parts are constantly moist. All these symptoms have no value at present for the special topical diagnosis.

The frequent occurrence of bedsores in the sacral region, over the glutæi, or more rarely on the feet or the inner side of the knees, is of great practical importance. Although trophic and vaso-motor influences may play a part in their origin, still their ultimate cause is always to be found in external conditions, pressure, uncleanliness, etc. The more faulty the care of the patient is, the easier bedsores arise. With completely paralyzed and anæsthetic patients, with incontinence of urine and fæces, of course they sometimes can not be wholly and permanently avoided, even with the most careful management. The extent to which a bedsore may reach is sometimes absolutely frightful. A large part of the sacrum may be laid bare, after the overlying soft parts and the periosteum have become gangrenous and been thrown off.

8. Disturbances in the Region of the Cerebral Nerves are entirely absent in most cases of transverse myelitis. In rare cases of cervical myelitis the process may gradually extend upward and give rise to bulbar symptoms. We sometimes see changes in the pupils also in cervical myelitis, such as inequality and spinal myosis; and finally myelitis has been repeatedly found combined with an optic neuritis.

Different Forms of Myelitis, Course of the Disease, and Diagnosis.—The whole picture of transverse myelitis in its different forms may be constructed from the symptoms described in detail in the preceding paragraphs. We can usually determine without difficulty, at least approximately, the seat and extent of the disease. If we group the chief symptoms of the different forms of myelitis together, they are as follows:

Cervical Myelitis.—Paraplegia of the legs, combined with more or less extensive disturbances in the upper extremities, and eventually disturbances of sensibility over a like extent. At times atrophy of single muscular regions in the arms. Muscles of the legs not materially atrophied. Increased tendon reflexes and spastic symptoms in the legs and often in the arms. Cutaneous reflexes in the legs retained, and sometimes even increased. Disturbances of the bladder and rectum. Sometimes changes in the pupils.

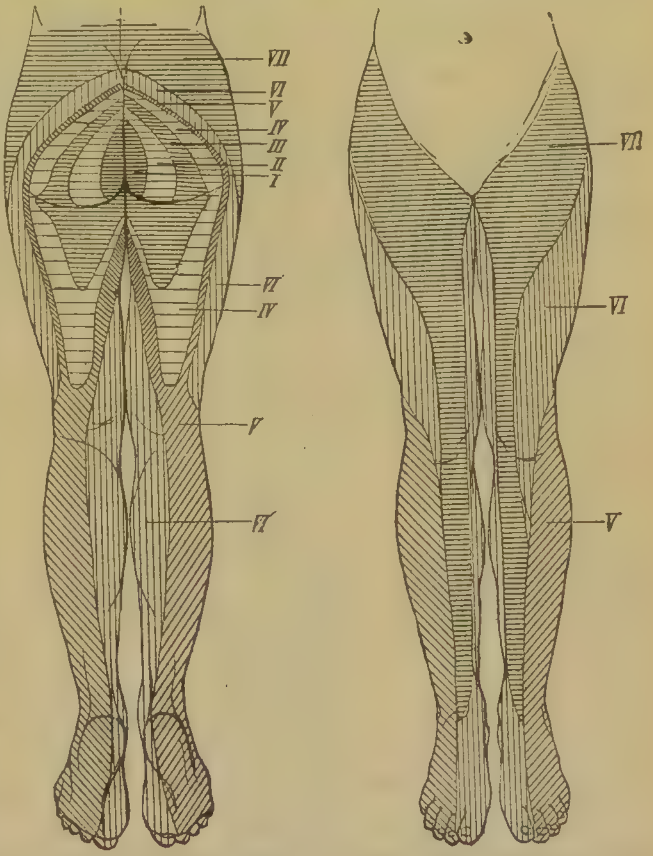

Fig. 84.—Areas of anæsthesia in lesions at various levels of the spinal cord from sacral v. to lumbar ii. *I*, Sacral v. *II*, Sacral iv. *III*, Sacral iii. *IV*, Sacral i. *V*, Lumbar v. *VI*, Lumbar iii. *VII*, Lumbar ii.

Dorsal Myelitis.—Upper extremities free. Motor, and eventually sensory, paraplegia of the legs, without degenerative atrophy. Increased tendon reflexes, especially strong in myelitis in the upper dorsal cord; cutaneous reflexes retained, rarely increased. Disturbances of the bladder and rectum.

Lumbar Myelitis.—Upper extremities free. Motor, and eventually sensory paraplegia of the legs. Cutaneous and tendon reflexes in the legs diminished or absent. Under some circumstances degenerative muscular atrophy, with reaction of degeneration. Disturbances of the bladder and rectum.

[In view of the increasing importance of exact localization of lesions of the spinal cord with regard to surgical interference we have thought it best to add Starr's tables, as modified by Herter, showing the functions of the segments of the spinal cord at the level of the various nerve-roots, and Starr's recent diagrams which show the sensory distribution of the different lumbar and sacral roots (see

Fig. 84). These, with the accompanying illustration (Fig. 83), showing the relation of the vertebræ to the nerve-roots, will serve as a guide to the exact localization of the lesion in cases which may require surgical interference, such as cases of meningeal hæmorrhage, fracture, caries, or new growths.—K.]

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

SEGMENT OF CORD.	NERVES.	MUSCLES.	SENSORY AREAS.	REFLEXES.
Second and third cervical.	Occipitalis major and minor. Auricularis magnus. Superficialis colli. Supra-clavicular.	Sterno-mastoid. Trapezius. Scaleni and neck muscles. *Diaphragm.*	Neck and back of head.	Hypochondrium (?).
Fourth cervical.	Supra-clavicular. Circumflex. Musculo-cutaneous. Musculo-spiral (radial).	*Diaphragm.* Supra- and infra-spinatus. Deltoid. Supinator longus. Rhomboidei.	Neck. Superior surface of shoulder. Outer surface of arm.	Cilio-spinal (fourth cervical to second dorsal).
Fifth cervical.	Supra-clavicular. Circumflex. External cutaneous. Internal cutaneous. Post. spinal branches.	Deltoid. Biceps and coraco-brachialis. Supinator longus and brevis. Rhomboidei. Deep muscles of shoulder-blade. Pectoralis (clavicular part). Teres minor. Serratus magnus. Brachialis anticus.	Back of shoulder and arm. Outer side of arm and forearm.	Scapular (fifth cervical to first dorsal). Biceps jerk. Supinator jerk.
Sixth cervical.	External and internal cutaneous. Radial.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Subscapular. Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Outer side and front of forearm. Back of hand (radial distribution).	Elbow jerk (triceps). Wrist jerk.
Seventh cervical.	External and internal cutaneous. Radial. Median. Post. spinal branches.	Triceps (long head). Extensors of wrist and fingers. Pronators. Flexors of wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	Radial and median distribution.	Palmar (seventh cervical to first dorsal).
Eighth cervical.	Internal cutaneous. Ulnar.	Triceps (long head). Flexors of wrist and fingers. Small muscles of hand.	Inner side of arm and forearm. Ulnar area of hand.	
First dorsal.	Internal cutaneous nerve of Wrisberg.	Extensors of thumb. Small muscles of hand. Thenar and hypothenar muscles.	Inner side of arm and forearm.	
Second dorsal.	Intercosto-humeral.		Inner side of arm near axilla.	
Second to twelfth dorsal.	Intercostals and dorsal posterior nerves.	Muscles of back and abdomen. Erectores spinæ.	Skin of back and upper glutæal region, and of breast and abdomen, in bands running downward and forward.	Epigastric (fourth to seventh dorsal). Abdominal (seventh to eleventh dorsal).
First lumbar.	Ilio-hypogastric. Ilio-inguinal.	Ilio-psoas. Sartorius. Rectus.	Skin over groin and front of scrotum.	Cremasteric (first to third lumbar).
Second lumbar.	Genito-crural. External cutaneous.	Ilio-psoas. Sartorius. Quadriceps femoris.	Outer surface of thigh.	
Third lumbar.	Anterior. Crural. Internal cutaneous. Long saphenous. Obturator.	Quadriceps femoris. Anterior part of biceps. Inner rotators of thigh. Adductors of thigh.	Anterior surface of thigh.	Knee jerk.

Segment of Cord.	Nerves.	Muscles.	Sensory Areas.	Reflexes.
Fourth lumbar.	Internal cutaneous. Long saphenous. Obturator.	Adductors of thigh. Abductors of thigh. Flexors of knee. Tibialis anticus. Peroneus longus.	Inner side of thigh, leg, and foot.	Glutæal (fourth to fifth lumbar).
Fifth lumbar.	External popliteal. External saphenous. Musculo-cutaneous. Plantar.	Outward rotators of thigh. Flexors of knee. Flexors of ankle. Extensors of toes. Peronei.	Outer and back side of leg and foot. Sole of foot.	Ankle clonus.
First and second sacral.	Same as fifth lumbar.	Flexors and extensors of ankle. Long flexor of toes. Small foot muscles.	Same as fifth lumbar.	Plantar (fifth lumbar to second sacral).
Third, fourth, and fifth sacral.	Small sciatic. Pudic. Inferior hæmorrhoidal. Inferior pudendal.	Perineal. Muscles of bladder, rectum, and external genitals.	Back of thigh, anus, perineum, and genital organs.	Vesical and anal centers.
Fifth sacral and coccygeal.	Coccygeal.	Coccygeus.	Skin about anus and coccyx.	

The whole course of myelitis is almost always chronic. We consider it impossible, as we have said, to make a sharp distinction between acute and chronic myelitis. Many cases, of course, show quite a rapid beginning, so that severe spinal symptoms develop in a few weeks. Such cases may be termed acute myelitis, but their further course is almost always chronic. In only a few cases do the morbid symptoms come on suddenly, progress rapidly, and speedily prove fatal. In such cases the autopsy shows a true acute inflammation of the spinal cord which is often disseminated. Sometimes the severe initial symptoms may after a certain time subside. There then remains a more or less severe, quite pronounced stationary type of spinal disease. In such cases the acute myelitis has healed, but the resultant cicatrization (contraction) is the cause of the permanent symptoms.

True chronic myelitis begins very gradually from the start, and in some cases only after years results in complete paraplegia.

As a rule, the disease begins with motor symptoms, either in one leg or in both at about the same time. The paresis gradually increases more and more, spastic symptoms set in, and symptoms of sensory irritation, such as formication, and also disturbances of the bladder, etc. The sensibility is sometimes a little blunted quite early, but it is almost always retained for a longer time than the motility. Only in the last stages is complete anæsthesia common. The whole duration of the disease is seldom under a year, and it often lasts two or three years, or even longer. Remissions, apparent halts, and improvements are not infrequent, and the condition often becomes rapidly worse. Recoveries are not impossible, but they are rare, at any rate. We know of no case that recovered where the diagnosis could be made with certainty. The cases reported where a recovery is claimed are usually cases of pressure paralysis, multiple neuritis, poliomyelitis, etc. The fatal termination is the result of the general weakness which finally sets in; or it comes from cystitis or pyelo-nephritis, both of which are often combined with pyæmic conditions; or from extensive bedsores; or finally from some complications, such as tuberculosis or acute diseases.

The diagnosis of diffuse transverse myelitis is always made by considering the whole group of symptoms prominent in the individual case. The possibility of a compression of the cord must be excluded by a careful examination of the vertebral column, and by consideration of the course of the disease. We must also be sure that the existing symptoms do not correspond to a definite typical disease, or a systemic disease, but that they can agree only with the assumption of an extensive diffuse disease at a certain point in the cord, to be made out accurately according to the symptoms. The further distinction, as to whether this diffuse disease is a myelitis, can of course hardly ever be made with absolute certainty, since diffuse new growths and the formation of cavities in the spinal cord must cause precisely the same symptoms. In these cases the decision can be made only by considering the whole course of the disease, and by the physician's individual acuteness in diagnosis. It is also still impossible at present to formulate with certainty the differential diagnosis between diffuse myelitis and certain combined fascicular or systemic diseases of the spinal cord (*vide infra*).

Treatment.—Although our therapeutic endeavors may rarely hope for a permanent and complete success, still in many cases the treatment can relieve the suffering and delay the end.

We can try to meet the causal indication in cases where the history or the examination shows syphilis. Even if the connection between this and myelitis can not be assumed with certainty, which is usually the case, still we must always try inunction thoroughly, using half a drachm to a drachm (grm. 2–5) of mercurial ointment a day. We give twenty to thirty grains (grm. 1·5–2) of iodide of potassium daily at the same time. We sometimes see decided improvement from this; but in some cases, of course, the result is uncertain, or the treatment seems to exert even an unfavorable influence upon the disease. In the latter case we must stop it at once.

Of the other methods of treatment the chief are electricity, baths, and cold-water cures. We alternate with these. New attempts at cure raise the patient's courage and hope afresh.

Electricity may give improvement in many cases, but of course it causes recovery only exceptionally, at most. In severe and hopeless cases, however, it is at least the best means of consoling the patient. The constant current has the greatest therapeutic value. We use large electrodes placed on the vertebral column, and pass not too strong a stabile or slowly labile current through the cord for about four or five minutes, chiefly through the region where we suppose the seat of the disease to be. We usually take the ascending current, and alternate with the two poles on the diseased part. We should avoid changes and great variations in the current. We associate with this peripheral galvanization, and often faradization of the muscles and nerves of the paralyzed extremities. Single symptoms sometimes deserve special attention—faradization of the skin in anæsthesia, galvanization of the bladder in vesical weakness, etc. The sittings should take place daily or every other day. If we would be successful, the treatment must be kept up persistently for months.

The treatment of myelitis by baths, if prudently used, may also be of evident service. Even simple tub-baths, such as can be had in almost every household, do good service under some circumstances. The chief rules are never to make the baths too warm—about 85° or 90° at most (24°–26° R.)—to limit them at first to ten or fifteen minutes, and to give them at first not oftener than three or four times a week. If the baths are well borne, they can be employed daily. We should be most cautious in incipient and still advancing cases. The best action of the simple warm bath is seen in chronic myelitis with predominant spastic symptoms. In these cases the duration of the baths may be increased to an hour or more. Sometimes the baths work still better than baths of simple water when certain substances are added, especially salt baths, which are made by the addition of five or ten pounds of common salt (Stassfurt salt), or four to six pounds of brine salt, or one to three quarts of brine to the water. By bringing carbonic acid into the water by a perforated tube in the floor of the tub we can easily make "artificial

Rehme baths," which were formerly often used with good success in the clinique in Leipsic.

If we can send patients in easy circumstances to a health resort, the carbonic-acid thermal salt springs at Rehme and Nauheim are most suitable for this purpose, and also sometimes mud-baths such as Marienbad and Elster, and the thermal baths of Ragatz, Teplitz, Wildbad, Gastein, or Wiesbaden.

A methodical cold-water treatment sometimes gives quite good results; but in these cases we should wholly avoid all the more heroic treatment such as douches, violent rubbing, and very cold baths, and employ only short, cool, full or half baths, or mild cold sponging. Hydrotherapeutics are usually combined with electricity.

We can expect but little success from internal treatment, but it can not be dispensed with in practice. Ergotine, strychnine (also given subcutaneously), iodide of potassium, and nitrate of silver are most to be recommended.

The general hygienic and symptomatic treatment is very important. If the first symptoms of a beginning spinal disease show themselves, we should urgently advise the patient to take the best possible physical care of himself, and recommend mental rest. The diet should be strengthening but easily digestible. Large amounts of spirits, much smoking, much tea and coffee, etc., are to be avoided. If the patient becomes bedridden, we must first employ the utmost care to get a good bed in order to guard against bedsores. In severe cases, especially where there are disturbances of sensibility, a water-cushion is most desirable. The patient's position must also be frequently changed, and the sacral region must often be washed and rubbed. Every incipient bedsore must be very carefully treated by Peruvian-balsam ointment (1 to 30) or iodoform, in order to prevent its spreading. When the bedsore is very extensive the continuous bath is the best remedy.

If there is retention of urine and the patient has to be catheterized, the most extreme care must be employed in cleaning and disinfecting the catheter, or else cystitis will develop in a few days. If it does, it is best in severe cases to wash out the bladder regularly with acetate of lead (1 to 1,000) and like remedies. In milder cases we may try chlorate of potassium internally, fifty to seventy-five grains a day (grm. 3–5), astringents, or balsams. If there is complete incontinence it is advisable to introduce a permanent catheter (*sonde à demeure*) into the bladder—that is, a Nélaton's catheter, which lies in the bladder and is fastened to the thighs by strips of plaster. The urine runs away through a rubber tube, and we avoid the constant wetting of the skin and linen.

Constipation must be met according to the general rules. We should be as sparing as possible with cathartics at first, and try to make an appropriate diet and enemata suffice. If there is severe pain, subcutaneous injections of morphine are unavoidable, but we always delay this as long as possible, although finally we let the dose of morphine be unlimited in hopeless cases.

CHAPTER V.

MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD.

(*Disseminated Nodular Sclerosis. Sclérose en plaques.*)

Ætiology and Pathology.—Multiple sclerosis of the central nervous system is a special chronic form of disease, whose anatomical basis consists in the development of numerous disseminated "sclerotic nodules" (*vide infra*) in the brain and cord. We know practically nothing as to its ætiology, for the significance of exposure to cold, overexertion, and mental emotions, sometimes given as causes of the disease, is wholly doubtful. It is also still undecided whether syphilis plays any part in the ætiology of multiple sclerosis. A hereditary predisposition seems to be prominent in some cases. [Marie lays stress upon acute infectious disease as a possible cause.—K.] The affection occurs chiefly in youth, somewhere between the ages of eighteen and thirty-five, but we have ourselves seen one case, which came to an autopsy, in a man of sixty. The disease also occurs in children. No material distinction has been made out as to sex.

In regard to the development of the different sclerotic nodules, nothing definite has been established at present as to their genesis. Various reasons lead us to favor the theory that the disease depends upon anomalies in the vessels, but the proof of this can not yet be given. The nodules are in part easy to recognize with the naked eye, from their gray color, and we can also feel an increased resistance. They are scattered over the whole central nervous system. Their favorite seats in the brain are the centrum ovale, the walls of the lateral ventricles, and the corpus callosum; the nodules are also quite abundant in the pons, less frequent in the medulla, and very abundant and variously distributed in the cord (see Figs. 85 and 86), and chiefly in its white substance. Examined microscopically, the nodules consist of an abundant, reticulated, fibrillary connective tissue, which is traversed only by comparatively few nerve-fibers that are preserved. In the vessels we notice first an increase in nuclei, and later usually a thickening of the walls. Fatty granular cells are always present in fresh cases. Charcot first made the statement, since confirmed by other observers, that the axis-cylinders are preserved in the nodules for a remarkably long time, even after the destruction of the medullary sheaths. Perhaps this is connected with the fact that secondary degeneration in the cord is strikingly absent.

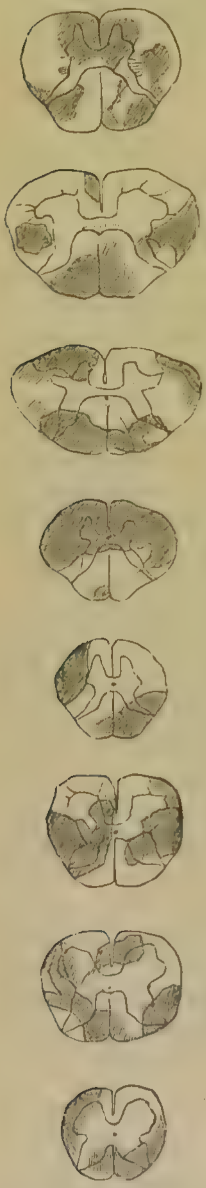

Fig. 85.—Example of disease of the cord in multiple sclerosis. The dark portions are the parts diseased.

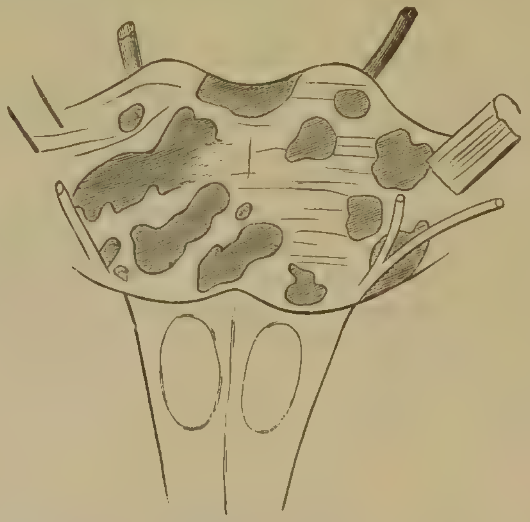

Fig. 86.—Distribution of the sclerosed nodules on the surface of the pons. (From Leube.)

Clinical History.—From the variations which the number and the localization of the nodules show, it may be understood from the outset that a type of the disease which represents all cases can not exist; but a comparison of a number of cases taken together always shows so characteristic a group of symptoms that the diagnosis can often be made with quite great certainty. We will first describe this typical form of disease, for the knowledge of which we must thank Charcot chiefly, and to that we will add some remarks upon the cases which differ from this type ("*formes frustes*"), which are by no means very rare.

The symptom of the typical cases of nodular sclerosis which we must first mention is tremor. This has been the reason why multiple sclerosis has formerly been repeatedly confounded with paralysis agitans, although the tremor in the two diseases shows entirely different peculiarities. In distinction from the constant rhythmical oscillations of the limbs in paralysis agitans (*vide infra*), the tremor in multiple sclerosis comes on only with intended movements—"intention tremor"—and usually does not show a perfectly regular rhythmical character, but is unequal and jerking, although the intended direction of the motion is, on the whole, always retained. The tremor is most marked in the upper extremities, as shown when the patient tries to take hold of a certain object, to bring a glass of water to his mouth, to bring the tips of the forefingers together, etc.; but the tremor also occurs in the head, in the trunk, and in the lower extremities. When the patient is perfectly quiet the tremor ceases entirely. Only a few exceptions to this rule have been known. If the patient be mentally excited the tremor usually becomes more marked. We know nothing as to its peculiar cause. We are ourselves most disposed to assume that it is due to an abnormal cross-conduction of irritation from one fiber to neighboring fibers. The possibility of such an abnormal cross-conduction seems to us to be due to the loss of medullary sheaths in the sclerotic nodules, with persistence of the axis-cylinders (*vide supra*). At any rate, the anatomical fact of the persistence of the axis-cylinders in the sclerotic nodules seems to have some relation to the clinical symptom that, as a rule, in multiple sclerosis we do not see real paralyses but other motor disturbances, especially the pronounced intention tremor.

Two other symptoms which often occur in nodular sclerosis are to a certain degree analogous to the tremor—a peculiar disturbance of speech, and nystagmus. The disturbance of speech depends upon disturbances in the motor innervation of the organs of speech, the tongue and larynx, and may probably be referred to the presence of sclerotic nodules in the pons and medulla. The speech is slow, "scanning," obscure, and finally sometimes almost incomprehensible. The equality in the pitch is often very striking. We often notice tremulous movements in the tongue and the lips on speaking. Nystagmus shows itself in the form of slight and usually lateral twitchings of the eyeballs on fixation, or on intended ocular movements.

Other motor disturbances are usually present besides the symptoms thus far described.

In many cases the crude strength of the muscles is for a long time completely normal, but in other cases there is a marked paresis, which sometimes increases to complete paralysis. The "spastic symptoms," however, are far more characteristic and more frequent (see the chapter on "spastic spinal paralysis"). These depend, in great part at least, upon the very considerable increase in the tendon reflexes, which is almost always present. In the upper extremities the spastic symptoms are less prominent, but even here we almost always find very vigorous tendon and periosteal reflexes on striking the lower ends of the bones of the forearm or the tendon of the biceps or triceps. In the lower extremities we see not only marked patellar reflex and a very intense and persistent ankle clonus (formerly given the unsuitable name of "spinal epilepsy"), but very often a pronounced tonic rigidity of the two legs. Passive motion is difficult, and the gait is completely spastic. If there is at the same time a marked paresis in the legs, the gait, although stiff, is also dragging—a paretic-spastic gait. The disturbances of sensibility are usually remarkably subordinate in multiple sclerosis. Only rarely do we find a blunting of sensation, and quite exceptionally marked anæsthesia. The cutaneous reflexes usually remain completely normal. Of the disturbances of the organs of the special senses we have still to mention that optic atrophy has often been seen, associated with considerable disturbance of vision, such as amblyopia or achromatopsia, or even with complete blindness. Optic neuritis with a subsequent atrophy also occurs, especially in the temporal halves of the papillæ, according to Gnauck. Finally, we sometimes see anomalies in the innervation of the ocular muscles, and diplopia caused by these anomalies.

In one class of cases there are certain cerebral symptoms which may be important with regard to diagnosis. In the course of the disease there often appears a certain mental weakness, an imbecility, which sometimes increases to marked dementia. Conditions of melancholy or exaltation are much rarer. We must also mention the occurrence of apoplectiform attacks. After slight prodromal symptoms, such as headache and vertigo, loss of consciousness and hemiplegia come on quite suddenly. With this the face is usually red, the pulse is frequent, and the temperature may rise to 104° or 106° (40°–41° C.). After a day or two the consciousness gradually returns, and the hemiplegia soon disappears. Epileptiform attacks are much rarer. We saw these repeatedly in a typical case; they were mainly unilateral, and were followed by a hemiplegia which soon passed away. The precise cause of these attacks is still wholly unknown. Vertigo or giddiness is a frequent cerebral symptom, which may develop even in the earlier stages of the disease, and often comes on paroxysmally.

Symptoms on the part of the bladder, the rectum, or the genital organs, are usually entirely absent in the typical cases, or they appear only toward the close of the disease. Trophic disturbances, such as muscular atrophy, are also rare.

In regard to the general course of typical cases, the disease develops very slowly and gradually. Motor symptoms, tremor, paresis, and disturbances in gait, usually appear in the lower extremities first. The patient often complains at the same time of occasional headache and vertigo. The speech gradually becomes more indistinct, the intelligence weaker, and the other symptoms of the disease described above develop. The affection almost always lasts for years and years. Variations, cessations, and remissions are common. We often see the condition rapidly grow worse, especially after the above-mentioned apoplectiform attacks. The last stage is characterized by the gradually increasing disturbance of the general nutrition, and, finally, by paralysis and bedsores. Death ensues from intercurrent diseases, from the increasing weakness, or sometimes in an apoplectiform attack.

Anomalous Cases.—Besides the typical form of multiple sclerosis described, there are often, as we have said, cases that vary from the type. We will mention briefly the following possibilities:

1. The disease may be very latent. We saw one case in which, for a long time, the only symptom was a complaint of slight headache and vertigo. Finally, there was a transitory apoplectiform attack, several months later an epileptiform attack, and a few days after that death took place. The autopsy showed a completely developed multiple sclerosis.

2. Sometimes the disease appears under the exact type of a chronic myelitis. The cerebral nodules cause no symptoms, they are present, perhaps, only in small numbers, and the spinal nodules cause a gradually increasing paraplegia of the legs, with vesical disturbance, loss of sensibility, etc. We have notes of two cases of multiple sclerosis, with autopsies, in which, during life, the diagnosis of a simple transverse myelitis had been made.

3. Cases have been repeatedly known where multiple sclerosis has appeared under almost the exact type of a spastic spinal paralysis (*vide infra*). In these cases many nodules were situated in the lateral columns of the cord. If the spastic symptoms be combined with muscular atrophy (nodules in the anterior gray cornua), the disease may even simulate the type of an amyotrophic lateral sclerosis, with at times co-existing bulbar symptoms (*vide infra*). If multiple sclerosis be localized to an unusual extent in the pons and medulla, the symptoms of a chronic bulbar paralysis may be prominent.

4. Symptoms like those of tabes dorsalis, pain and ataxia, are less frequently of chief prominence. Combinations of multiple sclerosis and gray degeneration of the posterior columns have, however, also been observed.

5. It sometimes happens that multiple sclerosis is the reason for a slowly developing hemiplegia, which may then be falsely regarded as cerebral, while the autopsy shows several nodules in the corresponding side of the cord and pons.

6. In many cases the mental disturbance, dementia, is so prominent that there is the pronounced picture of paralytic dementia (general paralysis), with disturbances of speech, etc.

7. Finally, we must mention here that Westphal has of late described some very chronic cases which have closely resembled multiple sclerosis in their type, although the autopsy usually showed no discoverable anatomical lesion of the nervous system at all. In these cases the symptoms consisted chiefly of muscular paresis, tremor on voluntary motion, paretic-spastic gait, disturbance of speech, difficulty in moving the eyes, rigid expression of the face, and of the presence of the so-called paradoxical contraction in the muscles of the legs (see page 546). A hereditary predisposition to nervous disease was probably of significance in their ætiology. Westphal proposes to call such cases provisionally "pseudo-sclerosis."

The **diagnosis** of multiple sclerosis in atypical cases is sometimes quite impossible, or at best it can be made with a fair amount of probability only when some, at least, of the characteristic symptoms of the disease are present besides the anomalous symptoms. The circumstance, indeed, that the anomalous cases will not properly fit the molds of any other form of disease, should make us think of the possibility of a multiple sclerosis; for in these anomalous cases, of course, there may be all possible combinations of symptoms.

The diagnosis is usually not difficult in the typical cases. The intention tremor, the spastic symptoms, the disturbance of speech, the nystagmus, the gradual manifestation of mental weakness, and eventually the apoplectiform attacks, are the most valuable signs in diagnosis. The distinction from paralysis agitans (*vide infra*) is almost always easy, if we remember that in this latter disease, in distinction from all others, the tremor is chiefly during rest, and the oscillations are much more equal. The diagnosis of "pseudo-sclerosis" can scarcely be made with certainty at present.

The **prognosis** of multiple sclerosis is utterly unfavorable. A case of recovery has never yet been seen with certainty. The disease may, of course, last for a very long time, as was said above.

The **treatment** adopts the same remedies that have been mentioned in the description of chronic myelitis. The galvanic current, tepid baths and sponging, and perhaps the internal use of nitrate of silver, may give the best promise of a temporary benefit.

CHAPTER VI.

TABES DORSALIS.

(*Locomotor Ataxia. Gray Degeneration of the Posterior Columns. Posterior Spinal Sclerosis.*)

We give at present the old name of tabes dorsalis, "consumption of the spinal cord," to a perfectly definite chronic disease of the central nervous system, whose chief anatomical basis is regarded as a typical degeneration of the posterior columns of the spinal cord. The disease has not been accurately known for a very long time. The first description, which, of course, is defective in many respects, is found in a work of W. Horn in 1827. We must thank especially the investigations of Romberg in Germany, in 1851, and of Duchenne in France, in 1858, for a more comprehensive knowledge of the disease, and for a precise distinction between it and the other chronic diseases of the spinal cord.

Ætiology.—But little that is definite was known until lately as to the cause of tabes dorsalis. Hereditary conditions play a very slight part in genuine cases, and even a general "neuropathic taint" can only rarely be made out in ataxic patients. Much weight in regard to ætiology was formerly laid upon previous exposure to cold. It can not be denied that sometimes the first symptoms of the disease follow some pronounced exposure to wet or cold, but much more frequently nothing of the sort can be made out. The case is similar with regard to physical and mental overexertion, which were formerly made answerable for the origin of tabes. It is an utterly ungrounded assertion that sexual excesses may be the cause of tabes. Some observers report that tabes may develop as a result of acute diseases or of injuries, such as a broken leg, etc. In these rare cases, too, it is hard to confirm the connection. The earlier teaching that tabes develops after "suppression of the foot-sweat" is manifestly due to a confusion of cause and effect. The absence of foot-sweat is not the cause, but a symptom, of incipient tabes.

The only ætiological fact which in our opinion is beyond any doubt is the relation of tabes to a previous syphilitic infection. This relation of the two diseases to each other was first confirmed in France by Fournier and in Germany by Erb, and in spite of the vigorous contradiction which the views of these observers at first met with on many sides, this theory of tabes is constantly acquiring more and more disciples.

In the first place, the connection between tabes and syphilis can be confirmed by statistics. Erb was able to find a history of syphilis, with secondary symptoms, in about 62 per cent. of his patients; and Fournier, in 103 cases, found syphilitic antecedents as many as 94 times. Our own observations agree exactly with Erb's data, since 61 per cent. of our patients stated definitely that they had formerly suffered from syphilis. If we also reckon the cases where the patients admit a former sore but no secondary symptoms, the percentage becomes much greater—90 per cent. In general it is worthy of note that, as a rule, in most cases of tabes the previous syphilis has not had a great intensity. Only quite infrequently do we find tertiary syphilitic symptoms as well as tabes; we have seen, for example, severe ulcers of the skin, gummous periostitis, etc. The time between the infection and the beginning of the first symptoms of tabes varies very much; it may be from two to twenty years.

The connection between tabes and syphilis becomes especially striking and convincing when we consider certain peculiar cases. Thus we sometimes see tabes in perfectly irreproachable women; here we can almost always detect previous syphilis in the husband which has been transmitted to the wife. We have ourselves seen tabes arise in this way in both persons. We may also find tabes in very young or very old persons, and then we can discover that syphilis was acquired at a very early or a very advanced age. Some cases of tabes observed in children were in all probability to be referred to hereditary syphilis.

Although we thus recognize the great probability of the connection between tabes and syphilis, we can not, on the other hand, conceal the fact that the perception of the precise nature of this connection causes no slight difficulty at present. The anatomical changes in tabes (*vide infra*) do not correspond at all to the other well-known anatomical products of constitutional syphilis, gummatous new growths; and thus they require an entirely separate classification. We are most disposed to assume that a chemical poison is formed by the action of the syphilitic infection, which has a special deleterious action on the affected system of fibers, these fibers being usually centripetal. Tabes accordingly stands in the same relation to syphilis as a "post-syphilitic disease," as diphtheritic paralysis and ataxia do to previous pharyngeal diphtheria. Whether the same disturbance of nutrition as in ordinary tabes can not also be provoked by other agents such as ergotine, can at present neither be affirmed nor denied. At least provisionally it can not be questioned that there are also cases of tabes where we can not discover a former syphilitic infection. We might state here, in opposition to statements that several authors have made, that the theory that syphilis excites merely an increased predisposition to the disease seems to us to mean absolutely nothing.

Finally, we must mention here the interesting fact discovered by Tuczek, that in chronic ergot poisoning—"ergotism"—symptoms may develop which are precisely analogous to tabes, and which depend upon a corresponding affection of the posterior columns of the cord that can be made out anatomically.

Tabes dorsalis is chiefly a disease of middle life. Most of the cases begin at the age of thirty-five or forty-five. The disease is decidedly more frequent in the male sex than in the female; but it is not especially rare in women, and here we can usually make out, with remarkable frequency, a previous syphilitic infection.

Pathological Anatomy.—If we examine the spinal cord of a patient who has died in the advanced stage of tabes, the smallness and thinness of the cord usually strike us first. The pia mater is thickened and opaque, especially on the posterior surface. We often see the posterior columns appearing through the pia as a gray band extending the whole length of the spinal cord. On cross-section we notice that the smallness of the cord is due chiefly to the atrophy of the posterior columns, which is often very considerable. These have wholly lost their normal backward prominence, and seem flat and sunken. From their pronounced gray color they are very plainly distinguished on cross-section from the rest of the white matter of the cord. The posterior cornua of the gray matter, and the posterior nerve-roots, show exceptionally a considerable atrophy, and appear very small and thin and also of a gray color.

Microscopic examination gives more exact information as to the extent and form of the degeneration. This shows that all portions of the posterior columns are not affected in like manner. The degeneration is always most intense in the lumbar cord; here it affects chiefly the middle and posterior portions of the posterior columns. while the most anterior portion remains intact in all cases (see Fig. 87). In the dorsal cord the posterior columns are almost completely degenerated. There are usually small normal areas still preserved in the posterior external and the most anterior portions. In the cervical cord (see Fig. 88) the so-called columns of Goll are chiefly affected, together with the prolongation of the fibers from the root-zones of the lumbar cord, and also the "lateral root-areas"—that is, those portions in the columns of Burdach where fibers enter directly from the posterior nerve-roots, and from which fibers may be traced farther into the gray matter of the posterior cornua; but the so-called posterior external areas, and also two little antero-lateral areas, remain entirely, or at least for a long time, free from the disease. Figures 89 and 90 show how the first beginnings of the disease are localized in the posterior columns. These were drawn from preparations from a case examined by us in the very first stage of the disease. A system of very fine fibers, entering through the posterior roots, is also frequently affected, even very early. They branch outward immediately after the entrance of the roots, and here occupy a small but very sharply defined territory at the point of the posterior cornua, between the posterior and lateral columns (Lissauer).

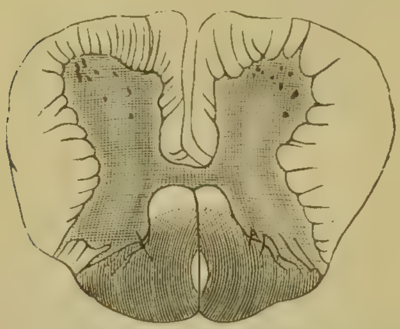

Fig. 87.—Transverse section through the lumbar region in locomotor ataxia. The diseased portions of the posterior columns are shaded.

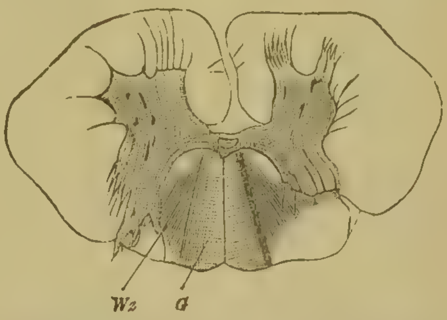

Fig. 88.—Transverse section through the cervical region in locomotor ataxia. *G.* Columns of Goll. *Wz.* Root-zones.

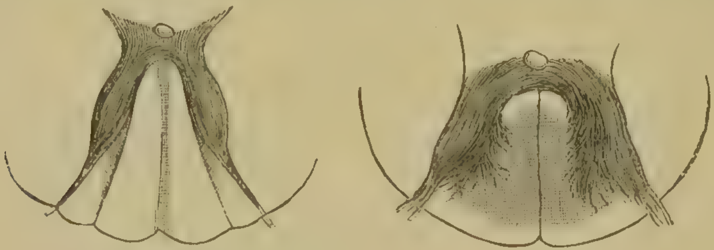

Fig. 89. Dorsal region. Fig. 90. Lumbar region.

Transverse section through the posterior columns of the cord in beginning locomotor ataxia.

We must state, with reference to the participation of the gray matter in the disease, that the posterior cornua, as we have already said, are also found considerably affected, which is explained mainly by the atrophy of the posterior root-fibers which enter them directly. It also can not appear strange that the medullated fibers found in Clarke's columns seem very much reduced in number, since they are also direct processes of the posterior root-fibers. The cells of the columns of Clarke remain normal.

On the other hand, the peripheral processes of the posterior root-fibers are not wholly spared. At any rate, in advanced tabes we can also make out in the larger peripheral nerve trunks, such as the sciatic, and probably still more in the finer branches of the sensory nerves, a number of degenerated fibers, at least of centripetal fibers (Déjérine, Oppenheim and Siemerling, and others). At present we can make no definite statement as to which part of the conducting tract the degeneration here begins in, or how far primary and secondary atrophies are to be separated; but it now seems most probable that the peripheral degenerations in tabes occupy an independent position, since, besides the changes in the peripheral sensory nerves, pronounced degenerative conditions sometimes occur in the trunks of certain cranial nerves, notably in the optic and oculomotor nerves, and more rarely in the vagus, acoustic, etc.

It is most remarkable that the changes described are found in almost precisely the same manner in all cases, that the same portions of the spinal cord are always chiefly affected, while certain other portions constantly remain free; that the disease is very exactly limited, and is precisely symmetrical in the two halves of the cord. This condition is explained only by the assumption that in tabes certain systems of fibers are always affected—that is, fibers which belong together in their anatomical and physiological aspect. Since fibers of different functions are manifestly diseased, as the symptoms of tabes show, we must regard the trouble not as a simple affection but as a combined sytemic disease, the more so as we often find certain cerebral nerves, as above stated, affected at the same time (*vide infra*).

The form of the disease consists of a primary degenerative atrophy of the nerve-fibers, and a corresponding secondary increase of the connective tissue. The gray color of the posterior columns is due to the loss of the medullary sheaths. Since the destruction of the nerve-fibers advances but very slowly, we never find more than a few fatty granular cells (see page 617). In old cases we find numerous corpora amylacea, whose origin and significance are still unknown. The thickening of the pia mater is a secondary and insignificant phenomenon.

We will state below the little that we know as to the precise relations between the anatomical lesions and the clinical symptoms of tabes, and we will also mention some other rarer anatomical changes in the disease.

Clinical History.—A disease which has as its basis so definite and strictly limited an anatomical change as is the case with tabes dorsalis would also be expected to give a very characteristic clinical picture. This supposition is entirely correct, and there are few diseases which can be diagnosticated, even in their earliest stages, with as much certainty as tabes. This fact is explained only by regarding tabes as a systemic disease, in which certain systems of fibers are always affected, while others are as constantly spared by the disease. The difference between individual cases of tabes lies, therefore, less in the symptoms themselves than in their intensity, their duration, and the order of their occurrence. In this regard, however, the differences in the clinical pictures are extremely varied, so that, even with a comparatively great personal experience, we often see new combinations of symptoms and also peculiarities in their course.

For the majority of cases we may sketch the following general description of the disease, in which it is better to divide the whole course into several stages; but, of course, this division can have only a schematic value.

Tabes dorsalis begins, as a rule, with a stage of initial symptoms, which develop very gradually and insidiously, and which may be of a very varying duration. The most characteristic symptoms of this stage are those of sensory irritation, most frequently in the form of the so-called lightning-like, "lancinating" pains in the lower extremities. They are sometimes very severe, but at other times only of slight intensity, and are comparatively little noticed by the patient, who regards them as "rheumatism." Many patients have a feeling of numbness and tingling in the tips of the fingers, especially of the ring and little fingers, and there is often a pronounced girdle sensation in the trunk. In some cases, too, neuralgic and migraine-like pains in the head may appear in the early stages.

Besides these symptoms of sensory irritation, which may often be for years the only symptoms of which the patient complains, two objective symptoms appear very early, which are of the greatest importance in the diagnosis of incipient tabes: the disappearance of the patellar reflex, first discovered by Westphal, and the reflex immobility of the pupils (Argyll Robertson). The absence of the patellar reflex is the most constant of all the known symptoms of tabes, and it is found so early that we can hardly ever decide with exactness upon the time of its occurrence. The reflex immobility of the pupil—that is, the failure of the pupil to contract to light, while the changes on accommodation may be perfectly retained—is, indeed, not so constant as the failure of the patellar reflex, but still it is quite frequent. If all three symptoms—lancinating pains, absence of patellar reflex, and immobility of the pupils—are present at the same time, the diagnosis of tabes is absolutely certain, even if all other symptoms are wanting, because this peculiar combination of three such apparently heterogeneous symptoms is seen in this disease alone.

Among the rarer initial symptoms we shall also learn to recognize diplopia, caused by paralysis of certain ocular muscles, loss of vision, from optic atrophy, and certain disturbances of cutaneous sensibility, such as analgesia. Sometimes disturbances in micturition appear quite early, while in other cases, however, gastric crises (*vide infra*) are the first symptom which the patient notices.

After this first stage of the disease has lasted for a very varying period, from a few months to two or five or even twenty years, the second stage begins; this we usually term the ataxic stage of tabes.

The beginning of this stage is recognized by the appearance of disturbances of gait. The gait becomes more difficult and more uncertain, and there are certain peculiarities which we will describe more fully later. Careful examination shows that the disturbance in gait is due not to a paresis of the muscles but to a disturbance of co-ordination, ataxia of the lower extremities. This symptom usually increases very slowly, until it reaches a degree where the patient can walk only with effort, and finally can not walk at all. There is often later, but almost always not for years, ataxia of the upper extremities.

Besides the persisting symptoms of the first stage, there are often now more marked disturbances of sensibility, as well as ataxia. The patient has a feeling as if he were walking on wool, felt, or similar substances. If he closes his eyes there is great swaying of the whole body—"Romberg's symptom." Physical examination of the sensibility often shows a marked loss of tactile sense, of sensibility to pain, or other disturbances (*vide infra*). A loss of muscular sense is especially frequent. The disturbances of micturition, such as incontinence, gradually become more marked, and very often cystitis gradually develops. This stage may also last for years. Sometimes the disease seems to stand still, frequently even manifest improvement is seen, but then the condition becomes worse again.

The third stage, the terminal stage of the disease, develops if the patient has not previously succumbed to an intercurrent disease. The symptoms are the same as in most of the other chronic diseases of the spinal cord. The patient gradually becomes more and more wretched and helpless, and finally is confined almost wholly to his bed. The ataxia is very marked, and sometimes even paresis develops, which may increase to an actual paralysis of the legs. In these cases, which are by no means frequent, we are right in calling the third stage of tabes the "paralytic stage." A severe pyelo-cystitis usually develops, bedsores appear, and death finally frees the patient from his lamentable condition.

We must now complete this briefly sketched picture of the disease by a more careful description of the single symptoms.

1. Disturbances of Motility in the Extremities.—The typical motor symptom of developed tabes dorsalis is the disturbance of co-ordination, the ataxia (see page 542). This is almost always seen in the lower extremities first. If we have the patient describe a circle in the air with his foot, while lying on his back we notice the irregularity, the "excursion" of the movement. It is still better to tell the patient to touch the knee of one leg with the heel of the other foot. We see then that the leg moved is often carried beyond the point designated several times before it reaches it. The ataxia is often noticeable, even in throwing one

leg over the other, as the leg raised makes much too great and too "throwing" a movement.

The alteration of the gait is very characteristic—the ataxic gait, from which we can often perceive the patient's disease at the first glance. If the patient sits down and tries to get up again to walk, there is difficulty in rising. He separates his legs to find a firm point of support, he takes a stick to help himself if he can, and he often gets the proper balance to keep himself erect only after several attempts. The gait itself is straddling, and the legs are raised abnormally high and set down with a stamp. If we have the patient turn rapidly or make a proper military "about face," the uncertainty of movement is still more marked. These methods of testing are therefore especially suitable for ascertaining the first beginnings of ataxia. Most patients always walk with a stick and control the movements of their legs by keeping their eyes fixed on the floor as they walk. This control is particularly necessary when the sensibility of the legs, especially the muscular sensibility, is diminished at the same time.

The disturbances of sensibility are also the sole reason for Romberg's symptom mentioned above—namely, the swaying with the eyes shut, especially when the patient puts his feet together. This phenomenon has often been classed with ataxia, but it depends merely upon the defective control of the muscular movements, which are necessary to preserve the equilibrium, as a result of the impaired sensibility of the skin of the soles of the feet and that of the muscles themselves. If this control be supplied by the eyes, the swaying is insignificant, but it at once becomes more marked if the control by the eyes is lost. From a like reason it is much harder for most ataxics to walk in the dark than by daylight.

If the ataxia be very marked, the patient can finally keep on his legs no longer. Walking is wholly impossible. The ataxia can then be made out very plainly from the different movements of the legs in bed. The throwing movement, the excess of innervation, is almost always most prominent.

If ataxia of the upper extremities occur in the course of the disease, it is easily recognized if the patient tries to take hold of some definite object, such as his ears, or if he brings the tips of his two forefingers together from a certain distance, or if he does fine work with his hands, such as writing or sewing. The movements are irregular and uncertain, and the excursions are marked. If there be at the same time any sensory disturbance in the arms, the anomaly in their movements is still greater with the eyes shut.

There has been much written and much dispute as to the cause of the ataxia in tabes dorsalis, although at present we have not attained perfect clearness and unity. There are three principal theories, or, more properly, groups of theories, which have been advanced up to the present time to explain the ataxia. According to the first theory (Jaccoud, Cyon, Benedikt), ataxia depends upon a disturbance in the reflex activity of the spinal cord. According to the second theory (Leyden and others), ataxia is the result of the sensory disturbance in tabes, "sensory ataxia"; and finally, according to a third theory (Friedreich, Erb), in ataxia we have a lesion of definite "co-ordinatory fibers," which run centrifugally, and preside over the co-ordination of motion. The exact place where these fibers run is not definitely asserted. If Charcot places these fibers in the external portions of the posterior columns, in the "cuneate fasciculi," it does not agree with the above theory, because centrifugal fibers probably do not pass through this part at all.

It would be an impossible task for us to try here to estimate these theories exactly and critically. The main reason why it is at present impossible to give an incontrovertible explanation of the occurrence of ataxia lies in the fact that we are not yet in a condition to know and to analyze exactly the process of normal co-ordination of motion; for manifestly every theory as to the causes of ataxia must begin with the processes involved in the co-ordination of normal movements. If we try to get a clear idea of this, the most essential point seems to us to be that co-ordination of motion is not a congenital function, but a power of our organs of motion learned by practice. The movements of little children who are learning to walk are ataxic, and even in later life it often happens that we have to learn how to perform certain complicated and difficult movements. We can get no other idea of this learning how to co-ordinate than that it takes place by the aid of the constant action of controlling and correcting impressions coming from the periphery—that is, centripetal—but we must bear in mind that these actions are mainly unconscious. The surer we become in the execution of the movements, the more the regulatory influence of the centripetal irritations falls into the background, without ever wholly disappearing. In these cases we must not consider, by any means, merely the irritations which are brought to the central organs from the skin of the parts moved; but we should consider, just as much or even more, those irritations which are due to the varying tension and position of the deeper parts, the muscles, the fasciæ, the ligaments, and the articular surfaces. Even the special organs of sense, particularly the eye, under some circumstances, assist materially in regulating motion.

According to this, a disturbance of co-ordination must take place when the regulating influences themselves either are absent or have lost their activity—that is, when the possibility of a successful transmission of these influences to the motor apparatus is absent. We do not know exactly which of these two conditions is realized in tabes. Perhaps both are concerned. Several circumstances may be cited to favor the theory of a loss of centripetal irritations in tabes: the disturbances of sensibility that can often be made out, the absence of the tendon reflexes, the undoubtedly diminished muscular tonus, etc. All these symptoms are certainly not in themselves the causes of ataxia, but still they are noteworthy facts, because they generally point to the actual loss of centripetal irritations. A second theory has perhaps still more support. According to this, the transmission of the regulatory centripetal irritations to the motor apparatus is disturbed in tabes. It harmonizes completely with the fact that the degree of ataxia in tabes does not run at all parallel to the disturbance of conscious sensibility. There are doubtless cases where there is a good deal of ataxia, while the sensibility—that is, the conscious perception of sensory impressions—is practically undisturbed. On the other hand, there are several cases recorded in literature in which, in spite of marked anæsthesia, there was no ataxia. In these cases the regulatory influence of the irritations from the anæsthetic parts was certainly absent, but it could be replaced by the control of the organs of the other senses, especially the eye; for as long as the completely anæsthetic patient has his eyes open he can walk well, but as soon as he shuts his eyes he can no longer stand a moment, and falls at once. In these cases, then, a regulation of motion by the eyes is still possible; there is no special ataxia. In genuine ataxia, motion remains unco-ordinated in spite of the control sought from the sensations of sight; which can be explained by the fact that the influences coming from the eyes, which regulate motion, are no longer of value, because their transmission to the motor apparatus has become impossible. Nevertheless, the eye has a certain unmistakable influence on the movements of ataxics. As soon as the patient shuts his eyes all the movements become much more uncertain and lack any control, so that the patient's judgment as to the degree of his movements, when there is at the same time cutaneous and muscular anæsthesia, is entirely lost.

We may suppose that the point where the transmission of centripetal impressions to the motor apparatus for the purpose of co-ordination of motion takes place is only in the gray matter, and that it takes place only by the interposition of the ganglion cells. We must thus assume that ataxia, so far as it depends upon any disturbance of transmission, is due anatomically to a lesion of the gray matter (posterior cornua ?); although, of course, we do not exclude the idea that the loss of centripetal, unconscious irritations, independently of a lesion of the centripetal fibers running in the posterior roots into the cord itself, may have an influence upon the occurrence of ataxia.

These brief glances at the conditions to be considered in the question as to the origin of ataxia may suffice to give the reader a preliminary survey of the most important points, and an incitement to further reflection upon this interesting subject.

Ataxia is the chief motor disturbance in tabes. The crude strength of the muscles may be perfectly normal, and it is chiefly a service of Duchenne's to have made clear for the first time the principal distinction between ataxia and paralysis. He showed that ataxics, who can no longer walk a step alone, can nevertheless exert the greatest strength with their legs. We have ourselves treated a teacher of gymnastics who, in spite of the most marked ataxia of the arms, had still so much strength in them that he could support himself in bed on his arms and keep his whole body, with his legs extended, in the air.

It sometimes happens, however, that even the crude strength disappears in tabes, and that the muscles become paretic. We have stated above that even a complete paraplegia may finally develop in the course of the disease. In these cases we find, on anatomical examination, that the process is no longer confined to the posterior columns, but that there is also a systemic degeneration of the lateral pyramidal motor tracts in the lumbar cord. The rare paralyses of certain motor nerves have another significance. They have usually been noted at the beginning of the disease, and affect the radial, peroneal, accessory, etc. They are probably due to changes in the affected peripheral nerves, but as a rule they are transitory, and, in our opinion, are to be put in the same category with tabetic oculomotor paralyses (*vide infra*).

We may add, finally, that slight symptoms of motor irritation, slight twitchings in the muscles, especially in the fingers, are not uncommon, but they are noticed only when the attention is especially directed to them. It is not certainly known how they arise; in our opinion, they are of reflex origin.

The condition of the muscles on passive motion is very characteristic. We notice in most cases a very striking flaccidity of the limbs, so that there is hardly any muscular resistance to be felt. We have to do, as it seems, with a diminution of muscular tonus, whose cause is not yet quite clear; but since there are many reasons for believing that the normal muscular tonus is of reflex origin, we are led to think of a connection between the absence of muscular tonus and the other reflex disturbances in tabes, such as the absence of the tendon reflexes.

The electrical excitability of the nerves and muscles, as we may also note here, remain perfectly normal in uncomplicated tabes.

2. Disturbances of the Cutaneous and Muscular Sensibility.—As we have already said, tabes begins, in the great majority of cases, with symptoms of sensory irritation, which usually persist in the later course of the disease also. Besides the simple paræsthesia—the feeling of tingling and numbness in the legs, and sometimes, too, a similar feeling which appears quite early in the upper extremities (especially often, as we have said, in the ulnar region)—the tabetic pains are remarkably characteristic of the disease.

The intensity of the pains differs very much in different cases; but we see a complete absence of them extremely rarely. The patient's attention is often first called to his slight and infrequent pains by direct questioning; but in some cases the severe pains are a constant distress to him. The pains most characteristic of tabes are the lightning-like, "lancinating" pains, which shoot like neuralgic pains for some distance along the course of the nerves. They often come on in very severe paroxysms, and are absent at other times. There are also boring, stabbing pains, which are fixed at one point and have their seat especially in the vicinity of the joints; and finally "constricting pains," which are felt most frequently in the back and loins. The well-known "girdle feeling" of tabetics—that is, the sensation of a band tightly encircling the trunk, or a tight, "drawn-together" pressure on the lateral portions of the trunk—belongs to the latter form of symptoms of sensory irritation. The girdle feeling is manifestly due to irritative processes in the region of the lower dorsal or upper lumbar nerves. Since it is comparatively frequent, and often appears quite early, it also has a certain diagnostic significance.

The tabetic pains also begin in the legs, corresponding to the almost constant beginning of the symptoms of the disease in the lower extremities; but later on quite analogous pains sometimes appear in the arms, and in very advanced cases we have also observed pains in the region of the occipital nerves and of the trigeminus. On the other hand, neuralgic pains in the face, especially in the region of the frontal nerve, or in the occiput, or even migraine-like attacks, also occur, even in the initial stage of tabes, as we have ourselves observed. In some cases the lancinating pains in tabes may be accompanied by the appearance of an eruption of herpes—a condition which at any rate is extremely rare.

Usually much later than the pains appears also a diminution of sensibility which can be made out objectively. As a rule, it may be stated that in most, but not in all, cases of tabes the sensibility does not remain normal; although more marked anæsthesia never appears until the more advanced stages of the disease.

The form of the disturbances of sensibility varies extremely, and no disease furnishes so many opportunities for the study of interesting details in the region of anomalies of sensation as tabes dorsalis. Our knowledge of the occurrence of partial paralyses of sensation especially is very largely based on the examination of tabetic patients. The tactile sense suffers in most cases of tabes, but we can usually make out only a certain blunting of it. Only when the disease is far advanced does the patient cease to feel a light touch on the skin. The sense of pain is also often abnormal. We sometimes see a pronounced analgesia, but in other cases there is a very decided sensibility to pain, in spite of a defective tactile sensibility. It is a very frequent symptom that, at a pin-prick, the patient has at first only a slight and not a painful sensation, and that a few seconds later, especially if the prick persist, he suddenly winces and says he feels a decided pain. With it there is usually a reflex contraction in the affected leg. We generally speak of this symptom as a "delayed conduction of painful sensations" or "delayed reflex"; but it seems to us that the symptom has not yet been analyzed carefully enough, and especially that it has not been properly separated from the after-sensations, which are also very common in tabes. It often happens that after a single prick, tabetic patients report at varying intervals five or six or more painful after-sensations.*

If the first sensation is not painful in these cases, it may happen that the patient may first say "Now" at a prick, and soon after "Ow," when he first feels the pain (the "double sensation" of Naunyn, Remak, and others). Fischer has termed a peculiar disturbance of sensibility occurring in tabes, polyæsthesia; the patient asserts, when examined with an æsthesiometer, that he feels several (four or five) points, although he was touched with but one.

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* If, while the patient's eyes are shut, we prick the leg and the arm or neck as nearly as possible at the same time, the prick on the leg would necessarily be felt much later than that on the arm, if there is a delayed conduction of sensory impressions from the leg; but we have never as yet been able to make out this condition with certainty.

Disturbances of the sense of pressure, and still more that of temperature, are also quite frequently found, especially as partial paralyses of sensation, when the sensibility is otherwise well preserved. We should note particularly the occurrence of partial anæsthesia of the sensibility to heat or cold. On the other hand, the special sensations of temperature may sometimes be pretty well defined, while in other respects there is quite a high degree of anæsthesia.

The considerable anomalies of the muscular sense, which are often to be made out in more advanced cases, have a special interest (see page 509). If the patient shuts his eyes, he is often entirely unaware of the situation and position of his limbs. He makes a false report as to the direction and extent of passive motions.* If the muscular sense in the arms be disturbed, and we put the arms into any unusual position, the patient has considerable trouble in bringing the hands together with his eyes shut. He gropes about in the air with his arms until he accidentally touches one arm with the other hand, and then he feels down this to the hand. The action of ataxia and muscular anæsthesia are accordingly combined in these cases, but we can not possibly consider the former as a result of the latter; for there are doubtless cases of ataxia—we have ourselves carefully examined such with regard to this question—in which the perception of motion and position is perfectly normal in spite of the existence of ataxia. The disturbance of movements willed, by the loss of muscular sense, is noticed only when the eyes are shut. With the eyes open the control by the sense of sight supplies the lack of muscular sensibility. Pitres has lately described peculiar attacks of muscular rigidity, and attacks of a decided feeling of fatigue in the muscles, apparently coming on spontaneously in incipient tabes ("*crises de courbature musculaire*").

Only in rare and far-advanced cases is there finally a complete anæsthesia of the lower, and exceptionally of the upper extremities. We then see also at times disturbances of sensibility in the region of the trigeminus, in the skin of the face. These disturbances are probably connected with a degeneration of the sensory ascending root of the trigeminus, which degeneration has already been observed several times at the autopsy (Westphal).

It is very hard to judge as to the dependence of all these sensory disturbances mentioned upon the anatomical changes of tabes. In many cases both the initial pains, and in part the anæsthesias, especially if they have their chief seat in a single nerve territory (ulnar, etc.), are to be referred to disease of the sensory peripheral nerves. The severer pains, however, are perhaps due chiefly to morbid processes in the posterior roots, while with a high degree of anæsthesia we are usually right in assuming that there is a pronounced atrophy in the posterior cornua.

3. Disturbances of the Reflexes.—The cutaneous reflexes show no constant changes in tabes. They are usually approximately normal, but sometimes they are diminished, especially if there be at the same time a marked disturbance of sensibility.

The absence of the tendon reflexes, especially of the patellar reflex, is, however, an almost constant sign of tabes, and is a sign of the highest diagnostic value. As we have already said, the disappearance of this reflex is one of the earliest symptoms of the disease, and is therefore of the greatest significance in the diagnosis of initial tabes. There are certainly cases of tabes in which the patellar reflex may persist for a long time, in spite of the development of many other morbid symptoms. These, however, are only very rare exceptions, which do not disprove the rule, and do not at all contradict our general views as to tabes. In individual cases the affected fibers, which serve to set free the reflex, may be spared for a long time, just as any other characteristic symptom of the disease may under some circumstances occasionally be absent. Concerning the precise anatomical cause of the disappearance of the patellar reflex, it can be due only to a degeneration in the centripetal portion of the affected reflex arc—that is, only in the fibers which belong to the territory of the posterior roots. It is in accordance with this that disease of the middle portion of the posterior columns in the lumbar cord (that is, the root-zones, see Fig. 89) always seems to be accompanied by a failure of the patellar reflex. The direct mechanical irritability of the muscles, especially of the quadriceps, is almost always retained in tabes.*

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* We can describe different letters and figures in the air with the patient's extremities, and try whether they can be correctly recognized with the eyes shut.

4. DISTURBANCES IN THE EYE AND THE OTHER ORGANS OF SPECIAL SENSE.—The authority for regarding tabes as a combined systemic disease arises from the frequency with which certain cerebral symptoms, as well as the spinal, are found in it.

The symptoms in the eyes deserve the first attention. We find disturbances in the pupils, of course not in all cases, but still in the great majority of them. The pupils are often very much contracted—" spinal myosis"—and show no trace of contraction to light, although the well-known changes in the pupils are very manifest upon any variation of the accommodation—dilatation of the pupils with approximately parallel axes of vision on looking at distant objects, and contraction of the pupils with the most marked convergence of the eyeballs on fixing a near object. We give this phenomenon, whose precise anatomical cause is not yet known, the name of reflex immobility of the pupils with retained mobility on accommodation (Argyll Robertson pupils). It is not at all necessary, however, that there should also be a myosis, for we not very infrequently find the pupils quite dilated or unequal, although we find there is reflex immobility. As we have already said, the immobility of the pupils is often a very early symptom, so that it also has a diagnostic importance. In other cases we find inequality of the pupils and, as a rare symptom, a striking change in the size of the pupils, so that first one and then the other pupil is the larger.

The paralyses of the ocular muscles in tabes are also very interesting. They are usually unilateral, but sometimes bilateral, and often appear even at the beginning of the disease, so that diplopia may be the first subjective symptom of which the patient complains. In every sudden oculomotor or abducens paralysis, coming on without any other cause, we must think of the possibility of an incipient tabes. It is remarkable that these paralyses in many cases disappear permanently and entirely after some time; but sometimes they remain, as we have repeatedly seen, especially in a case with bilateral abducens and unilateral oculomotor paralysis, and also in a case with almost complete bilateral oculomotor paralysis. In the autopsies on such cases we find the trunks of the affected nerves and their nuclei markedly atrophic. It seems to us very probable, however, that the temporary ocular paralyses in tabetic patients depend upon changes in the peripheral nerves of the ocular muscles.

The third complication in the eyes in tabes is optic atrophy. It occurs in about ten or fifteen per cent. of all cases, and is usually an initial symptom, at a time when the absence of the tendon reflexes, which may usually also be noticed, is the only thing, except this, to render the diagnosis of the disease possible. The patient complains of diminution of vision, and the power to distinguish colors, especially green, disappears quite early. On objective examination, we find, besides this anomaly in the color sense, usually a limitation of the field of vision, and the beginning gray degeneration of the optic nerve can easily be made out on ophthalmoscopic examination. The affection sometimes makes little halts and slight apparent improvements, but it usually ends with complete blindness. The optic atrophy more rarely first appears in the later stages of the disease, when all the other symptoms are already fully developed.

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* [From autopsies on cases of combined sclerosis, where the symptoms very closely resembled those of tabes, but where the patellar reflex persisted until near the close of life, Westphal thinks that the patellar reflex disappears only when a special region in the posterior columns is involved in the disease. This region lies at the junction of the dorsal and lumbar cords, between the posterior cornu and a line running parallel with the posterior fissure, and starting from the angle seen in the posterior cornu at the site of the substantia gelatinosa.—TRANS.]

Auditory disturbances are much rarer than those of sight, but they also occur. The cause, in at least a part of the cases, is an atrophy of the acoustic nerve. Symptoms are also frequently seen which resemble those of Ménière's disease—tinnitus, vertigo, and deafness.

Changes in the senses of taste and smell have been observed only in a few cases.

5. DISTURBANCES IN THE BLADDER, THE RECTUM, AND THE SEXUAL ORGANS.—Difficulty in emptying the bladder is an almost constant symptom in the later stages of tabes. It sometimes, indeed, appears very early. The patient feels the desire to urinate more frequently, there is often a slight involuntary micturition, and at other times there is retention of urine, sometimes coming on quite suddenly; in advanced stages there is frequently complete incontinence. A cystitis very often develops as a result of all these disturbances, which may be the starting-point of severe cysto-pyelitis and pyelo-nephritis, and thus be the cause of death.

Persistent constipation is also a very frequent symptom of tabes, the reason for which is perhaps to be sought in the defective reflex excitation of intestinal peristalsis. The constipation may in many cases give rise to great distress to the patient, since it sometimes provokes very painful sensations in the loins and abdomen. The coccyodynia, which sometimes occurs in tabes, has already been mentioned (see page 528). Incontinence of fæces occurs quite rarely in the last stages of the disease.

A diminution in the sexual functions is found almost constantly in advanced cases. The loss in power is also often one of the initial symptoms.

6. SYMPTOMS IN THE INTERNAL ORGANS.—We see, not very infrequently, in tabes certain symptoms in the internal organs which are in part very characteristic, and which, at any rate, are based upon disturbances of innervation. The most important and the most frequent are the so-called "gastric crises." These almost always come on suddenly and paroxysmally, and consist of an extremely severe cardialgic pain, which is accompanied by violent vomiting. The patient also feels very wretched, and there is often at the same time palpitation, acceleration of the pulse, vertigo, etc. The attacks last about two or three days. In many patients they are repeated every few months. As we have said, the gastric crises may appear very early. We ourselves know cases where, in consequence of severe gastric crises, a severe gastric affection has been falsely diagnosticated, in the beginning of the disease. Attacks of diarrhœa, "intestinal crises," usually not associated with pain, have also been repeatedly observed.

We term attacks of severe dyspnœa "laryngeal crises." These probably depend upon a (reflex ?) spasm of the glottis, and may attain a very alarming degree. They are also associated with a severe spasmodic nervous cough. Paralysis of the laryngeal muscles (crico-arytænoids) has also been observed. We may assume changes in the vagus-accessory nucleus, or in the vagus or recurrent itself (Oppenheim), as the anatomical cause of all these symptoms.

In a few cases "renal crises" ("*crises nephritiques*") have also been described. They consist of severe attacks of pain, like renal colic. French authors also describe "urethral crises" and "*crises clitoridiennes*," the paroxysmal appearance of voluptuous feelings with a vaginal secretion in women, in the beginning of the disease.

We may mention, finally, that we sometimes see in tabetic patients a constant and very great frequency of the pulse, 100 to 120 a minute. We also frequently see the combination of tabes with aortic insufficiency which has been mentioned by some authors. The precise connection between the two affections is probably dependent upon previous syphilis.

7. Trophic Disturbances.—In many cases of tabes trophic disturbances are entirely absent. We have already spoken of the occasional appearance of an eruption of herpes, with severe lancinating pains. In some cases a marked exfoliation of the epidermis has been seen, and also a falling out of the hair and nails. Sometimes there are small hæmorrhages, apparently spontaneous, into the skin or into the visible mucous membranes, especially into the conjunctiva, as we have seen in several cases.

The peculiar joint affections which occur in tabes, and were first accurately described by Charcot as "*arthropathies tabétiques*," have a greater interest. (See Fig. 91.) The affection is situated most frequently in the knee- and hip-joints, more rarely in the ankle- and shoulder-joints. It is usually bilateral, even if it be more marked on one side than on the other. We sometimes find abundant serous effusions, so that the knee-joint in particular may swell up in monstrous fashion; but we find especially a high degree of arthritis deformans, with marked atrophy of the ends of the bones, and with the formation of many osteophytes. Spontaneous dislocations and fractures also occur. An affection of the anterior gray cornua, which Charcot suspected to be a cause of the articular affection, could not be made out in a case examined by us anatomically. We do not believe that the theory of an exclusive "nervous-trophic disturbance" can explain the origin of the tabetic joint affections. Perhaps we have to do with syphilitic joint affections, and perhaps with those of some other more fortuitous (traumatic ?) origin, which are rather a complication than a symptom of tabes. We believe, however, that the unusual intensity and the peculiar form of the anatomical lesion are indeed directly connected with tabes, and are due chiefly to the anæsthesia of the articular surfaces. We saw a case, a short time ago, where an affection of the knee-joint developed in a comparatively very early stage of tabes, which up to that time had not been diagnosticated at all. As the patient felt no pain at all in his knee, he nevertheless hunted most vigorously through a whole autumn, until finally an extremely severe swelling of the knee-joint and an actual dislocation of the leg ensued.

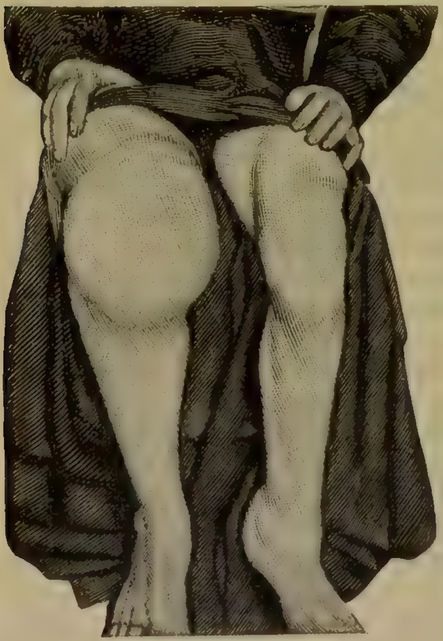
Fig. 91.—Tabetic arthropathy of the right knee and left ankle. Personal observation.

The muscles preserve their normal state of nutrition, except as they take part in a general emaciation. Charcot described a case of a combination of tabes with genuine progressive muscular atrophy, in which the autopsy showed a degeneration of the anterior gray cornua in the spinal cord besides the atrophy of the posterior columns. The first report upon a unilateral atrophy of the tongue, which sometimes develops quite early in tabes, is due to the same observer. Nothing definite is known at present as to the origin of this peculiar complication.

In conclusion, it is worthy of note that cases of "*mal perforant du pied*" (ulcerations on the heels or between the toes) have been repeatedly observed in tabes.

8. Cerebral Symptoms.—Besides the frequent important disturbances on the part of certain cerebral nerves, such as the optic and oculomotor, which have already been mentioned, we must mention here the relation between tabes and progressive general paralysis (*q. v.*). On the one hand, the symptoms of tabes are often present in the course of general paralysis, so that the autopsy shows a typical degeneration of the posterior columns (Westphal); and, on the other hand, it also happens that the whole process begins with a tabes, which may exist alone for years without any mental symptoms, and then only at the close do the symptoms of paralytic dementia, delusions of grandeur, dementia, etc., appear.

The complication of tabes with hemiplegia repeatedly occurs. The latter depends upon a cerebral hæmorrhage or an embolic or thrombotic softening, so that it is doubtful whether the two affections have a real connection or are merely a chance combination. It seems to us worthy of note that in two such cases we saw scarcely any contracture develop in the paralyzed limbs.

Course and Prognosis.—Although most of the characteristic symptoms of tabes develop in almost all cases, still the order and the intensity of their onset vary greatly. We have already briefly described the general type of the disease which most frequently comes under observation, and many other peculiarities in its course have been mentioned from time to time.

We have stated that the initial period is usually characterized, apart from the symptoms that can be made out only objectively, such as absence of the patellar reflex and reflex immobility of the pupils, by the lancinating pains; that these may differ very much in intensity; and that the duration of this first stage may vary between a few months and ten or twenty years. The optic atrophy, the ocular paralyses, gastric crises, vesical disturbances, etc., were mentioned as rarer initial symptoms. The passage from the first stage to the second—the stage of ataxia—is sometimes very gradual, but in some cases very rapid and sudden. We have repeatedly seen such changes, with a sudden change for the worse in the condition. If the previous symptoms were slight, the patient dates his disease from this point, and says that he was quite suddenly broken down by some cause, and that since then he has not been able to walk at all, or else only with difficulty. In such cases there is often slow improvement following the sudden change for the worse in the condition, which, of course, is not permanent.

No rules of general value can be given as to the further advance of the disease, the invasion of the arms by the ataxia, or the occurrence of the rarer symptoms, such as the joint affections, etc. Almost every individual case affords its idiosyncrasies, since one group of symptoms is often especially prominent, while another is entirely absent or developed only to a slight degree. On the whole, however, we can almost always make out a gradual, even if a very slow, advance in the disease. New symptoms appear, the old ones increase, the general condition becomes worse, until finally the last stage of the disease comes on.

Recovery from tabes occurs only very rarely, if at all. The treatment of the affection may indeed cause improvement, delay the course of the disease, and alleviate single symptoms, but the prognosis is always to be regarded as unfavorable, although many patients, especially under favorable external conditions, may lead a tolerable existence for years.

Diagnosis.—There is scarcely any other disease of the spinal cord whose diagnosis can in most cases be made with so great certainty and such comparative ease. Since tabes is a combined systemic disease, it affords a definite combination of symptoms such as can occur under no other conditions. The diagnosis, therefore, is to be made not from any one single symptom, but only from the combination of all and from the whole course of the disease.

The diagnosis of initial tabes is especially important. In every case of obstinate "rheumatic" pains, or similar pains in the lower extremities, we should think of the possibility of tabes, and examine the tendon reflexes and the pupils. The combination of characteristic pains, absence of the patellar reflex on the two sides, and reflex immobility of the pupils, usually makes the diagnosis almost perfectly certain; two of these symptoms, especially if the reflex immobility of the pupils be one, make it at least very probable. Ocular paralyses, temporary ptosis, or temporary diplopia, may be very important for the diagnosis. With these symptoms, too, we should never forget to think of the possibility of tabes, and to look for the other characteristic symptoms. Finally, we may recall the fact here that the disease may begin with an optic atrophy, and that early gastric crises may simulate a gastric affection, early disturbance in micturition a vesical trouble, or swelling of the joints a joint disease, until careful examination of the other symptoms explains the true nature of the disease.

In the fully developed ataxic stage of tabes the diagnosis is almost always easy, and often can be made at the first glance. The history, the characteristic ataxic gait, the swaying with the eyes shut, the absence of the reflexes, etc., make the diagnosis certain. The diagnosis may be more difficult if we happen to see the patient for the first time in the final stage, when actual paralysis has set in, when a complicating hemiplegia has arisen, etc. In such cases we must lay stress on the development of the disease and find out what characteristic tabetic symptoms —pupillary symptoms, absence of patellar reflex, remains of ataxia, or pains—can now be discovered. With proper attention and knowledge of the case, the diagnosis can even then almost always be made correctly.

Vertebral affections are to be mentioned first of the diseases which may be confused with tabes. These also cause, under some circumstances, lancinating pains and an absence of the patellar reflex, as a result of compression of the spinal roots; but in these cases the later course of the disease is entirely different, apart from the changes in the vertebral column and the absence of other characteristic symptoms of tabes. The same holds true of certain deep-seated tumors in the neighborhood of the spinal cord. We have already said that in rare cases a multiple sclerosis may offer symptoms similar to those of tabes. In these cases the chief stress in regard to diagnosis is to be laid on the *ensemble* of symptoms and their development. It is of greater practical importance that certain toxic nervous diseases may have a great similarity to tabes. Chronic alcoholic neuritis has already been spoken of in this connection (see page 583). In these cases, however, the reflex immobility of the pupils and the disturbances of the bladder are usually absent, while atrophic paralysis may develop later, which never happens in tabes. The ætiological factors are also, of course, to be taken into account.

Finally, it may be mentioned here that we have twice seen a group of nervous symptoms in workmen who have worked many years in tobacco factories, which resembled tabes in so many points that we might term it "nicotine tabes." The morbid symptoms, which resemble tabes, consist of painful sensations, absence of the patellar reflex, contracted pupils with reflex immobility, and uncertainty of gait; but the whole type of the disease is distinguished from tabes by a peculiar tremor, by a marked increase of the cutaneous reflexes, especially in the lower extremities, etc.

Treatment.—The tedious course of tabes demands that the physician have at hand a choice of remedies and methods of treatment which he can vary according to the predominating circumstances, either to obtain a certain amount of improvement by a new way of attacking the disease, or at least constantly to kindle the patient's hope and courage anew.

If syphilis be a possible ætiological factor, we regard it as entirely justifiable to prescribe first an antisyphilitic treatment: inunction of fifty to seventy-five grains (grm. 3–5) of mercurial ointment a day, with iodide of potassium internally. In very many cases this, of course, does no brilliant service—at times the disease has even been noticed to grow worse under inunction—but sometimes we see distinct improvement. The earlier the treatment is begun, the more is to be expected of it. At any rate, we should try whether we can not check the further advance of the disease by a methodical and continuous antisyphilitic treatment. When symptoms implying loss of function (*Ausfallssymptome*) have already appeared, we can not, of course, cause them to disappear, for iodine and mercury can not restore fibers in the posterior columns that have already been destroyed.

If the antisyphilitic treatment be not indicated, or have been unsuccessful, electricity and balneo-therapeutics or hydro-therapeutics deserve the most confidence.

The electrical treatment consists chiefly of the passage of the ascending constant current through the spinal cord. The currents must not be too strong, and the sittings should take place daily, or every other day. Erb recommends placing the medium-sized cathode in the vicinity of the sympathetic, and the large anode close to the spinous processes on the other side of the vertebral column, moving it at intervals from above downward. This procedure lasts about four or five minutes for each side. We also obtain good results symptomatically when there are severe pains, vesical weakness, etc., by peripheral galvanization. If we find painful points on the vertebral column, as is rarely the case, they should be especially treated with the stabile anode. Lately, the treatment recommended by Rumpf with the faradic brush has been used several times with good results. This consists in brushing the skin of the back and the extremities with a strong current for five or ten minutes. Every form of electrical treatment, in order to obtain results, must be kept up for months.

Hydro-therapeutics, used rationally, have often resulted in considerable improvement in tabes, although they may cause much mischief. Hot baths, especially vapor-baths, are often followed by a rapid change for the worse—a fact which, unfortunately, we can often observe where vapor-baths have been prescribed for patients at the beginning of their disease "for rheumatism." Continuous wet packs and severe rubbings are also often accompanied by unfavorable results. Tepid half or full baths, however, of 75° to 86° at most (20°–24° R.), associated with gentle rubbing of the skin, often do good service. Wet compresses, laid about the abdomen or the legs at night, often have a favorable influence on the pains. In general, it is a good plan to send well-to-do patients in summer to a water-cure establishment which is conducted by an experienced director and well managed, but the necessary procedures may also be undertaken at home.

Of the baths whose use is recommended in tabes, Oeynhausen-Rehme has the greatest reputation, and the best results to show. Many tabetics, of course, come back from Rehme just as they went; but, in advising a bath, Rehme is always the first to be considered. We may refer to what was said on page 626 as to the establishment of artificial Rehme baths. The baths in Nauheim have a very singular composition. The indifferent thermal baths, Teplitz, Wildbad, and Ragatz, formerly much in favor, have at present lost their reputation in tabes. Mud baths and iron baths—Pyrmont, Driburg, Cudowa, Elster, Franzensbad—may sometimes act favorably.

Besides the methods of treatment so far mentioned, there are still a number of internal remedies, the use of which seems sometimes to be of advantage. The chief ones to be mentioned are nitrate of silver, first recommended by Wunderlich, one-sixth-of-a-grain pills (grm. 0·01), at first three, gradually increasing to six a day, before meals; and ergotine,* one-grain pills (grm. 0·05), three to six a day; we may also try iodide of potassium, phosphorus, arsenic [chloride of aluminium, two to four grains (grm. 0·1–0·25), thrice daily], etc. All these remedies, especially the two first named, may be used for a long time, and, with interruptions, even for years.

Finally, we must mention here nerve-stretching, usually stretching of the sciatics, which for a short time was practiced on many tabetic patients as a result of a somewhat too sanguine recommendation on the part of Langenbuch. Since, however, experience has taught us that nerve-stretching, in spite of some apparent successes, scarcely ever exerts a permanent favorable action, and is also not wholly without danger, the operation has been almost entirely given up in tabes. It may still be tried in those cases where we have unusually severe attacks of pain in the region of definite nerves.

[Of late years Charcot has advocated suspension in the treatment of tabes. The patient is suspended in a Sayre apparatus from one to three minutes daily or every other day. It is not without danger, and it should not be employed if any spastic symptoms be present. It has not met with the success at first claimed for it, but in a few cases it seems to relieve the pain and the ataxic gait.—K.]

Symptomatically, the same remedies are to be considered as were mentioned in the treatment of chronic myelitis. We try to alleviate the pains by narcotic embrocations and bandaging the legs. Of internal remedies, sodic salicylate and antipyrine sometimes undoubtedly alleviate and shorten the pains. In bad cases morphine is indispensable. We try to remove the constipation by prescriptions as to diet, or by mild cathartics, such as the bitter waters, tamarinds, and rhubarb, and by enemata. Morphine is the best remedy in the gastric and laryngeal crises. Cystitis and bedsores must be treated according to the rules in general use.

In regard to the patient's general manner of life, we must warn him against any physical or mental overexertion, prescribe a prudent but strengthening diet, and enjoin good air, a country residence in summer, or perhaps the Alps or sea air. The earlier we get the patient under treatment, the more persevering and careful should we be in our treatment, because then we can still hope for success. In old and far-advanced cases we must confine ourselves to a purely symptomatic treatment.

APPENDIX.

HEREDITARY ATAXIA. FRIEDREICH'S FORM OF TABES.

A peculiar and rare disease, which has a certain similarity to tabes, was first described by Friedreich under the name of "hereditary ataxia." The affection almost always occurs in several children of the same family, and develops in youth, somewhere between twelve and eighteen years of age. The female members of the family are decidedly more often affected than the male. A stage of initial pains is usually absent. The disease begins with a pronounced ataxia of the legs, which usually very soon passes to the arms. The tendon reflexes disappear in most cases, but the sensibility of the skin and muscles remains perfectly intact, a fact which may properly be regarded as evidence for the independence of ataxic disturbances from anomalies of sensibility. The vesical functions also remain completely normal for a long time. Disturbances of vision have not been observed up to the present time, but in the further course of the disease a peculiar disturbance of speech arises, which is probably due to a disturbance of co-ordination in the muscular movements of the lips and tongue necessary in speaking. Friedreich has also sought to interpret the nystagmus which appears by the hypothesis of an "ataxic nystagmus." The disease lasts for a very long time, for many years, and finally leads to complete paralysis, contractures, and atrophy of the paralyzed muscles.

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* There is only an apparent contradiction in the fact that, in spite of the occurrence of an "ergotine tabes" (*vide supra*), ergotine is also used as a remedy against tabes. It may very well be that the same remedy, which in large doses causes certain systems of fibers to atrophy, may in smaller doses have some favorable (irritating) action on the same system of fibers; but we must always be cautious in the use of ergotine.

The anatomical examination of the spinal cord has so far shown in all cases a combined fascicular disease of the posterior and lateral columns. In the case reported by Kahler and Pick this disease could be demonstrated as a combined systemic disease. It affected the lateral pyramidal tracts, the lateral cerebellar tracts, the fundamental bundle of the posterior columns, and the columns of Goll. F. Schultze has lately taken the same views in regard to the anatomical lesion.

The disease is incurable; at least all the attempts at treatment up to the present time have been unsuccessful.

ATAXIC PARAPLEGIA.

(Combined Postero-lateral Sclerosis.)

[This name has been given by Gowers to a chronic degenerative disease of the spinal cord which involves both the posterior and lateral columns. It differs from Friedreich's ataxia in that it is not a family disease, and that it usually attacks persons in middle life, men being affected more frequently than women. Unlike true tabes, syphilis is only rarely a factor in the ætiology. The various alleged causes of chronic myelitis have been supposed to have an influence in producing this affection, especially exposure to cold, trauma, and sexual excess. In Italy, pellagra has been known to produce it. Chronic lead poisoning may be of influence.

As has been said, the pyramidal tracts and the postero-median columns are chiefly affected, but the degeneration is not limited absolutely to these systems of fibers.

The symptoms, as may be inferred from the distribution of the sclerosis, are a combination of those of spastic paraplegia and true tabes. There is weakness of the legs combined with ataxia. Rigidity is a late symptom. Pain is occasionally present, but it is much less marked than in true tabes. Sensory disturbances are rarely met with. The knee-jerk is usually exaggerated, and there may be ankle clonus. If, however, Westphal's tract be involved (*vide supra*), the knee-jerk is lost. The bladder and rectum may become affected later. Eye symptoms are not common. The affection may be associated with general paralysis (*q. v.*).

The affection is slowly progressive. The diagnosis is based on the co-existence of ataxia and paraplegia, and the absence of sensory and ocular symptoms. Transition forms more closely resembling chronic myelitis may also occur. The treatment is that of chronic myelitis.—K.]

CHAPTER VII.

AMYOTROPHIC LATERAL SCLEROSIS.

AMYOTROPHIC lateral sclerosis is a disease perfectly sharply defined both in its clinical and its pathological aspects, and in the majority of cases it may be diagnosticated with great certainty, even during the patient's life. For the first accurate knowledge of it we must thank Charcot, who published in 1869, in company with Joffroy, his first observations upon such cases, and who in 1874 was able to give quite a complete description of the disease; but an exact knowledge of amyotrophic lateral sclerosis was first made possible by Flechsig's investigations upon the course of the paths of conduction in the spinal cord. According to these, it may be stated with perfect certainty that the affection is to be regarded as a systemic degeneration of the pyramidal tract throughout its whole extent, or at least in certain portions of it, combined with atrophy of certain nerve-nuclei in the medulla oblongata. It is still entirely unknown to us what causes bring on the disease of these nerve-fibers and the cells belonging to them. In individual cases we can not usually make out any definite ætiological factor at all. Severe physical exertion is sometimes claimed to be a cause for the disease. It occurs chiefly in persons in youth and middle life, between twenty-five and forty-five. The male sex seems to be decidedly more prone to the disease than the female.

Pathological Anatomy.—In the typical cases of amyotrophic lateral sclerosis, which come to autopsy in the last stage of the disease (initial cases have not yet been examined anatomically), we find in the spinal cord a degeneration or "sclerosis" of both pyramidal tracts, and a considerable atrophy of the corresponding large ganglion-cells in the anterior gray cornua, especially in the external portion. The degeneration of the pyramidal tract is to be made out either in the two lateral columns alone, or, if there be an anterior pyramidal tract also, in one or both anterior columns (see page 535 and Figs. 66 and 67). It occupies mainly the same area on cross-section of the cord as the region of the pyramidal tract; the boundaries of which tract have been determined by the distribution of secondary descending degeneration (*vide infra*), and by the results of the history of development. Beginning in the lowest part of the lumbar cord, it may be traced upward to the pyramids of the medulla, and sometimes, but not always, still farther through the pons and the crura to the internal capsule. In some cases examined recently by Charcot and Marie the degeneration extended even to the central convolutions, whose large motor ganglion-cells also showed a pronounced atrophy. Toward the lateral cerebellar tract the disease of the pyramidal tract in the cord is very sharply defined, but in the anterior portions of the lateral columns other bundles of fibers are often affected. The whole extent of the disease in the lateral columns is therefore usually greater in amyotrophic lateral sclerosis than in secondary descending degeneration. Of the greatest clinical importance, however, is the already-mentioned co-existing disease of the motor ganglion-cells in the anterior cornua of the spinal cord and the disease of certain nerve-nuclei in the medulla oblongata, especially the hypoglossus nucleus, the vagus-accessory nucleus, etc. From these cells, which are inserted into the motor tract, the degeneration passes on toward the periphery, in the affected nerve-trunks (hypoglossus, etc.), or in the anterior roots belonging to them. It is difficult to make out atrophied fibers in the peripheral nerves, and up to the present time they have not always been looked for with sufficient care, but it can scarcely be doubted that the affected motor fibers, which are processes of the atrophied ganglion-cells, are likewise to be found in a condition of degeneration. Finally, the muscles show a considerable atrophy, as is plainly manifest even during the life-time of the patient. Their volume is much diminished; many muscles (for details *vide infra*) finally almost wholly disappear, so that in their place there is little left but connective tissue and fat. In the other muscles we find, besides a number of normal fibers still preserved, many very small fibers, and also some which have lost their tranverse striation and show a granular or fatty degeneration. The nuclei of the sarcolemma are usually increased, and the interstitial fat tissue is often, but not always, abundantly developed.

We accordingly see that the anatomical basis of amyotrophic lateral sclerosis is a more or less completely isolated disease of the great motor cortico-muscular conducting tract from the center to the periphery. The process is to be regarded as a simple degenerative atrophy. Fiber after fiber becomes diseased and atrophies. We do not know where the process begins, whether at one definite spot, whence it extends upward and downward, or whether the fibers throughout their whole extent, with the corresponding ganglion-cells and muscular fibers, are attacked at the same time. Perhaps different possibilities are to be regarded in these cases, which may explain the many variations in the clinical course. At any rate, the different portions of the system may become diseased in a varying order, and the further extension of the disease may vary in its rapidity. The spinal and the bulbar diseases are perfectly analogous to each other and are co-ordinated. Both affect portions of the same system; one belongs to the muscles of the extremities, the other to the muscles of the face, tongue, etc. The nerve-nuclei in the medulla are to be regarded as precisely analogous to the anterior gray cornua. The destruction of the nerve-fibers is always the primary process, the increase of the interstitial connective tissue and the slight changes in the vessels are a secondary, accidental process.

Besides the typical cases of amyotrophic lateral sclerosis, there are also, quite rarely, combined and transitional forms. Besides the degeneration of the pyramidal tract, an affection of the posterior columns and a degeneration of the lateral cerebellar tract have also occasionally been found.

Clinical History.—In all typical cases the clinical symptoms give a perfectly characteristic type of disease, limited strictly to the motor sphere, corresponding to the perfectly systemic anatomical lesions just described.

The first signs of the disease almost always begin in the arm. The patient notices a difficulty in working, and becomes easily tired. The weakness of the arm gradually increases, and finally, usually some months later, involves the other arm. A wasting of certain muscles, which gradually increases and becomes more extensive, is now often noticed by the patient. About six months or a year later, symptoms appear in the lower extremities. The gait becomes stiff and uncertain, the patient gets tired more easily, and quite a marked tremor of the legs often comes on, apparently spontaneously.

If we now examine the patient carefully, the type of the disease is usually perfectly plain. We notice, first in the upper extremities, a very pronounced and more or less extensive muscular atrophy. This is usually most marked where it begins—namely, at the thenar and hypothenar eminences. The interossei are also plainly atrophied, and the muscles on the extensor side of the forearms. The flexors of the hand and the fingers remain intact longer. In the upper arm the triceps and the deltoid are usually the most atrophied, and later, and to a less degree, the biceps and the muscles of the shoulder. We find a functional disturbance of the muscles—a paresis—corresponding to the degree of atrophy. The functional capacity depends upon how much muscle is left, and only with a complete disappearance of the muscle is there a complete loss of the corresponding motion, but a marked paresis can sometimes be noticed in muscles which are not yet much atrophied. The electrical excitability in the muscular fibers still preserved is normal. The strength of contraction of the muscle irritated by the faradic current is therefore proportional to the amount of muscular substance still present. In the much-atrophied muscles the effects of irritation are finally very slight, and then we can always make out a distinct reaction of degeneration in the degenerated muscular fibers that are still left, especially in the ball of the thumb. We can scarcely ever make out with certainty a loss of excitability in the nerve-trunk, probably because here a greater number of normal fibers are always preserved.

The examination of the tendon reflexes is very important. They are invariably much increased, even from the early stages of the disease. We obtain vigorous reflex contractions from a gentle blow on the tendons of the biceps and the triceps, and on the lower ends of the bones of the forearm. These are of diagnostic importance, because they never occur in this way in ordinary "progressive muscular atrophy"—that is, that disease in which the degeneration extends merely from the muscles to the motor ganglion-cells in the anterior cornua, while the lateral motor-tracts remain free (*vide infra*). In the later stages of the disease marked contractures in the arms and hands sometimes, but not always, develop. The sensibility of the skin and deeper parts, however, remains absolutely normal.

The first morbid symptoms usually develop in the lower extremities some months later than in the arms. The pure spastic symptoms are here remarkably prominent, while the muscular atrophy is late in its development, and is but slight. The legs become stiff, and oppose considerable muscular resistance to attempts at passive motion, but the crude strength of the muscles is decidedly below the normal. There is an obvious paresis, although there is, as it seems, hardly ever a complete paralysis of the legs; and the disturbance of motion, at any rate, is considerably increased by the spastic symptoms (see a later chapter). These symptoms depend mainly upon the great increase of the tendon reflexes. The patellar reflex is very vigorous, and we often find a marked and persistent ankle clonus. The patient can still walk quite a distance, but the gait is, of course, difficult and laborious. The patient walks with short, slow, dragging steps—the spastic-paretic gait. The sensibility also remains absolutely normal in the legs. The cutaneous reflexes show no striking changes. Disturbances in micturition are also entirely absent. The bowels may be somewhat costive, but are otherwise normal.

After the condition has lasted for some time (a year or two) in this form—muscular atrophy and increased tendon reflexes in the upper extremities and spastic paresis in the lower—and has slowly grown worse, bulbar symptoms come on in the third and last stage of the disease. The speech gradually becomes more indistinct, and there is difficulty in swallowing. If we examine closely we find the lips atrophied, so that puckering the mouth, whistling, etc., are difficult. We also notice a decided atrophy of the tongue. Its surface is uneven, and we notice more or less marked fibrillary twitchings of single muscular bundles. The sensibility is also normal here. We sometimes find a vigorous masseter reflex (jaw-jerk) on striking the lower jaw, analogous to the increased tendon reflexes in the extremities. If the patient have trouble in taking food, from difficulty in swallowing, the state of the general nutrition soon becomes worse. Respiratory disturbances are usually the final immediate cause of death, if an intercurrent disease, such as inhalation pneumonia, etc., does not previously put an end to the patient's melancholy condition.

The picture of amyotrophic lateral sclerosis just sketched accords very well with the pathological lesions. As the degeneration affects the main motor tract exclusively, the clinical symptoms are also limited entirely to the domain of motility. The associated affection of the anterior gray cornua explains the occurrence of muscular atrophy, while the degeneration of the lateral columns must be made answerable for the paresis, independent of the atrophy, and for the spastic symptoms. The increase of the tendon reflexes, whose reflex arc passes through the anterior cornua, forces us to suppose that the disease in the lateral columns precedes the degeneration in the anterior cornua, as this increase is seen especially in the lower extremities; for manifestly there can be no longer any reflex in muscular fibers whose special ganglion-cells are already atrophied. The increased reflexes are seen only in muscles which are composed, at least in part, of normal fibers. The bulbar symptoms are dependent upon the degeneration of the nerve-nuclei in the medulla.

The **diagnosis** of the disease is usually easily made. Its typical course, the muscular atrophy with co-existing increased tendon reflexes, the complete absence of sensory or vesical disturbances, and the final appearance of bulbar symptoms, are chiefly to be considered in diagnosis. Confusion may arise from the fact that tumors or myelitis may for a long time have a similar localization, as in the gray matter of the cervical cord, and therefore provoke analogous symptoms; but in such cases the later course is almost always different, and thus a subsequent diagnosis can be made correctly.

The **prognosis** of amyotrophic lateral sclerosis must be regarded as absolutely unfavorable. The disease advances slowly but unceasingly, and usually leads to death in a few years. Only in a few cases, occurring in early youth (Seeligmüller), does the disease seem to come to a standstill.

We can therefore expect but slight results from **treatment.** At most we can perhaps check the advance of the disease by an electrical treatment, kept up with very great patience and perseverance.

CHAPTER VIII.

PROGRESSIVE (SPINAL) MUSCULAR ATROPHY.

Preliminary Remarks and Pathological Anatomy.—Few diseases of the spinal cord have undergone such different conceptions and significations in the course of time as progressive muscular atrophy. The reason for this lies chiefly in the fact that its main symptom, the progressive atrophy of the voluntary muscles, may be found in many totally distinct diseases, and hence has given rise to constant confusion and mistakes. If, at the present time, we read the older and even a part of the newer literature on this subject, we find everywhere a mixture of different cases, not at all of the same disease, and only the latest accurate clinical and anatomical methods of investigation have enabled us to bring at least some order out of this chaos.

Except for a few earlier observations, Duchenne and Aran, in 1849 and 1850, gave the first good description of progressive muscular atrophy. The French observers, therefore, at present speak of the disease, in distinction from other similar affections, as "*atrophie musculaire progressive, type Duchenne-Aran.*" A short time after, in 1855, Cruveilhier, on the ground of a positive lesion on autopsy, first pronounced the opinion that a disease of the gray matter in the spinal cord was to be regarded as the special anatomical cause of the disease. Since then a tedious dispute has been carried on, and even in part kept up to the present time, as to whether, in fact, the disease has its seat in the spinal cord, or rather in the muscles themselves—a dispute which was necessarily without results for a long time; the more because the actual pathological basis was very scanty, and because the data of examinations completely contradicted one another, owing to the confusion between different forms of morbid processes which did not belong together at all. The spinal nature of the disease was proven especially by the investigations of Lockhart Clarke, and Charcot, while in Germany Friedreich in particular has of late asserted its myopathic origin.

In our opinion there can no longer be any doubt* of the fact that there is a perfectly clearly defined disease, whose chief clinical symptom consists of a very slow but constantly progressing atrophy of the muscles, following a certain type, while anatomical examination shows a degenerative atrophy, not only of the affected muscles, but also of the corresponding peripheral nerve-fibers, anterior root-fibers, and motor ganglion-cells in the anterior cornua of the spinal cord. We are therefore right in separating this disease, as a "spinal form of progressive muscular atrophy," from those cases where there is also the development of an independent progressive atrophy of the muscles, but where the affection always remains to the last confined to the muscles, and never invades the motor nerves and the spinal cord. These last-named cases form the pure muscular atrophies, which correspond to the types of the "hereditary or juvenile muscular atrophy or pseudo-hypertrophy" (see the appendix to this chapter). The spinal form of progressive muscular atrophy, with which we have to do here, is undoubtedly nearly allied to "amyotrophic lateral sclerosis," described in the previous chapter; but although in the latter the whole pyramidal motor tract may be degenerated, and the lateral pyramidal tracts in the cord are in particular always affected, in "progressive muscular atrophy" the degeneration, as we have said, is confined to that portion of the motor conducting tract which extends from the ganglion-cells of the anterior cornua to the muscular fibers themselves. The further central prolongation of this tract, however—that is, the lateral pyramidal tract—remains perfectly normal. It is very improbable that this difference in the extent of the anatomical localization is the principal distinction between the two diseases named. In their ætiological relations the two diseases, and also progressive bulbar paralysis (*vide infra*), are identical. The clinical distinctions due to the different anatomical localization are, however, of sufficiently marked prominence to justify, at least provisionally, a special description of progressive muscular atrophy and of amyotrophic lateral sclerosis. [In the great majority of cases which have come to autopsy, changes have been found in the lateral columns, so that an anatomical distinction between the three affections can hardly be maintained. Hence Schultze suggests the name of motor tabes as a general term for the three.—K.]

The precise anatomical lesion in progressive (spinal) muscular atrophy is as follows:

In the spinal cord, most marked usually in the cervical cord, we find the anterior gray cornua very small; the ganglion-cells have wholly or largely disappeared, and those remaining are atrophied; and the neuroglia is changed to a fine fibrous tissue, sometimes studded with spider-cells; but the lateral columns, especially the pyramidal tracts—that is, the portion of the motor conducting tract central to the ganglion-cells of the anterior cornua—are perfectly normal. The anterior roots and the affected motor fibers in the peripheral nerves are also atrophied, although in the nerve-trunks the discovery of degenerated fibers mixed with many other normal fibers is not perfectly easy. In the muscles the atrophy is, of course, still more noticeable on a post-mortem examination than on examination during life. The muscles most affected are reduced to small, pale, and flabby bundles, in which fat and connective tissue outweigh the proper muscular tissue. On histological examination, we find in many fibers a simple atrophy—that is, a very considerable diminution, but with the transverse striation still retained; but in many other fibers we run across the signs of a degenerative atrophy—fatty and waxy degeneration of the muscular fibers, a splitting longitudinally or transversely, etc. The interstitial connective tissue is always increased, the muscular nuclei are increased in number, and we often find quite a deposit of fat between the fibers which are still preserved.

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* To the cases already published we can add a recent observation of our own, on a case of progressive muscular atrophy, involving the upper extremities chiefly, with almost complete atrophy of the ganglion-cells in the anterior cornua, although it was not followed by a corresponding co-existing degeneration of the lateral pyramidal tracts.

So far the actual lesion. In comprehending it, the only difficulties arise from the questions as to the mode of development, and as to the reciprocal dependence of the different disturbances. Is the atrophy of the anterior cornua to be regarded as primary, and the atrophy of the nerves and muscles as a secondary descending degeneration; or does the process begin in the muscles, and extend upward from them to the spinal cord; or, finally, do we have to do with an approximately simultaneous degeneration of the whole motor portion affected? These are questions to which at present no definite answer can be given. Many reasons seem to us to favor the belief that the degenerative process begins in the last terminal branches of the motor nerves, and from these ascends gradually to the spinal cord. This, however, is not yet proven, and possibly the starting-point of the disease and its further extent may differ in different cases.

Ætiology and Clinical History.—Progressive muscular atrophy is an extremely slow and chronic disease from the beginning. We often can not find any ætiological factors which seem to favor its development, but in a good many cases the first symptoms follow an immoderate muscular exertion. Thus we see the first signs of muscular weakness coming on, for example, after continued thrashing, hard washing, and similar severe physical toil. A hereditary predisposition is reported as common by most observers, but it is at present certain that most cases of this "hereditary muscular atrophy" belong, not to the spinal, but to the myopathic form (*vide infra*). It also seems to us to be very probable that of most of the other ætiological factors reported their number is to be explained, for the most part, only by erroneously reckoning other forms of atrophic processes with genuine progressive muscular atrophy. We refer especially to the alleged origin of this disease from injuries, from acute diseases, such as typhoid and diphtheria, or from syphilis.

The disease begins, by far the most frequently, in the upper extremities, and especially, as it seems, in the right arm, but sometimes in the left, or in both arms at once. As a rule, it begins with an atrophy of the short muscles of the thumb and of the hypothenar eminence, which is accompanied by a corresponding disturbance of function. Any other symptoms, especially disturbances of sensibility, paræsthesia, or pain, are usually entirely absent. The atrophy usually affects the abductor pollicis brevis first, and then the opponens and the adductor. We notice very early the characteristic sinking in and flattening of the ball of the thumb, and the abnormal position of the thumb, which is persistently approximated to the second metacarpal bone—the "ape hand." At the same time, or a little earlier or later, the interossei begin to atrophy, which is recognized by the sinking in of the interosseous spaces and the increasing incomplete extension of the terminal phalanges of the fingers. The atrophy of the lumbricales causes a visible flattening of the hollow of the hand. If the disturbance in the function of the interossei have reached a certain degree, the same claw-like position of the fingers appears, as we have already learned to recognize in ulnar paralysis (see

Fig. 78), due to the contracture of the antagonist of the interossei, the extensor communis digitorum.

In the further progress of the disease the atrophy extends either to the muscles of the forearm, or, what is not at all rare, it jumps over these and attacks the muscles of the shoulder, usually the deltoid first. In the forearm it is usually the muscles on the extensor side which are first attacked, the abductor and extensor longus pollicis, and only later the supinators, flexors, etc. In the upper arm the deltoid almost always atrophies first, and then the biceps, while the triceps may remain intact for a comparatively long time. Sooner or later the muscles of the trunk are often added to the list, the trapezius first usually, then the pectorals, the rhomboidei, and the latissimus dorsi. The disturbance of function caused by the atrophy of all these muscles is readily apparent from what was said in the chapter on the different forms of paralysis. In advanced cases the arms hang down laxly by the two sides of the trunk. Only with the greatest difficulty, if at all, can the patient do anything with them, either dress or undress; but he sometimes learns to help himself, at least in some degree, by throwing movements, by bending his body to meet anything, by using his mouth in taking hold of things, etc. Quite rarely the atrophy finally attacks the muscles of the neck. The severest respiratory disturbances may be excited by an implication of the diaphragm and the other muscles of respiration.

The time which elapses before the gradual appearance of the more marked disturbances of function is almost always very long. Years may elapse before the atrophy extends from the little muscles of the hand to the other muscles of the arm. In the muscles of the legs the first signs of atrophy almost always develop very late if at all. The arms are often perfectly useless when the patient can still walk for hours. Of course there are some exceptions to this rule. In the arms themselves, too, the process does not always develop in the way described above. Thus we not very rarely see the affection begin in the scapular muscles or the deltoid, and from this point the atrophy involves the muscles of the hand or of the upper arm. The muscles of the trunk, the pectorals, and the muscles of the back, are much more rarely the starting-point of the disease, and in only a very few cases have we been able to make out that the disease began in the legs. It is worthy of note that in such abnormal cases the muscles first affected were sometimes, but not always, subjected to extraordinary demands in carrying loads, etc.

Besides the atrophy, and the loss of function that runs parallel to it, we must mention some other changes in the muscles. The fibrillary muscular twitchings are often very striking. A constant tremor and wave of the muscle may be provoked by them. In some cases they are weak and only rarely noticed. They usually become vigorous if one irritates the muscle mechanically by a blow. The electrical excitability of the diseased muscles varies, inasmuch as it depends entirely upon the number of normal fibers still preserved in the muscle. Since the atrophy affects only one fiber after another, the faradic and galvanic excitability decrease gradually, and are lost completely only when the greatest part of the muscle is destroyed. On careful testing, we can then, however, almost always make out a decided reaction of degeneration in single muscles that are much diseased, especially in the form of the so-called partial reaction of degeneration: the excitability of the nerves is retained, but the contractions in the muscles themselves seem very slow, and the anodic closure contractions (AnSZ) predominate (see page 552).

In many cases there is an increase of the fatty tissue at the same time with the atrophy of the muscular substance, which often makes it very hard to judge of the atrophy; but the loss of function in the muscles, the diminished electrical excitability, and the peculiar soft feeling which atrophied muscles have when covered with fat, reveal the true condition of things. Other trophic disturbances in the skin are usually entirely absent, but they are sometimes seen. In a few cases a spontaneous pemphigoid formation of bullæ has been observed, especially in the hands. The skin sometimes becomes thick and fissured, and the nails become brittle, grooved, and greatly curved. The coldness and cyanosis of the skin sometimes seen are due perhaps to vaso-motor disturbances, but at any rate we must also consider the disturbance of circulation which is due to the lack of muscular movements.

The examination of the tendon reflexes is of great diagnostic importance. While they are invariably decidedly increased in the upper extremities in amyotrophic lateral sclerosis, they are entirely absent in genuine progressive muscular atrophy, a condition which is easily explained by the atrophy of the motor ganglion-cells belonging to the reflex arc, or by the atrophy of the centrifugal motor fibers. Since no degeneration of the lateral pyramidal tract precedes this atrophy, it goes without saying that the preceding increase of the tendon reflexes, characteristic of amyotrophic lateral sclerosis, is also absent. In the lower extremities the patellar reflex is retained as long as the disease spares the legs, but it is not increased. If the atrophy involve the legs, the patellar reflex is usually lost.

In distinction from all these manifest disturbances in the motor region, the sensibility of the skin and deeper parts remains perfectly preserved. There are also never any morbid symptoms on the part of the bladder or rectum.

In many cases the affection finally invades the muscular region innervated from the medulla; the symptoms of "progressive bulbar paralysis" (*vide infra*) are added to those of progressive muscular atrophy. This combination of spinal and bulbar disease appears, as we have previously shown, as a rule, in amyotrophic lateral sclerosis, and even after the disease has lasted a comparatively short time. In genuine progressive muscular atrophy the bulbar symptoms usually develop, if at all, only after the disease has gone on for years. Then the speech begins to become indistinct, from the atrophy of the tongue, swallowing is difficult, and the patient finally succumbs to increasing inanition or to respiratory disturbances. In their principal characteristics the muscular atrophy of the extremities and the bulbar symptoms are precisely analogous phenomena, inasmuch as the nerve nuclei in the medulla have precisely the same significance for the muscles of the tongue, the pharynx, and the face, as the anterior gray cornua of the cord have for the muscles of the trunk and the extremities. In many cases, however, bulbar symptoms do not develop at all, since the patients die before then of some intercurrent disease.

Diagnosis.—The diagnosis of progressive muscular atrophy can easily be made if we confine ourselves strictly to the definition of the disease, and do not confuse it with other affections in which the muscular atrophy is only a symptom which under some circumstances may have an entirely different origin: muscular atrophies in extensive diffuse myelitis, in tumors, and in the formation of cavities in the cord, in multiple neuritis, as a result of articular affections (see the chapters on acute and chronic inflammations of the joints), etc. We should consider especially the typical course of the affection in most cases of genuine progressive muscular atrophy, its beginning in the upper extremities, the small muscles of the hand, or more rarely the muscles of the shoulder and upper arm, its slow advance, the peculiar "individualization" of the atrophy—that is, the affection of some muscles while other neighboring muscles remain completely normal; and, finally, the absence of all disturbances of sensibility or of the sphincters. Progressive muscular atrophy is doubtless nearly allied to amyotrophic lateral sclerosis, but the latter is distinguished by its more rapid course, and especially by the increase of the tendon reflexes due to the affection of the lateral columns, and the corresponding appearance of spastic symptoms in the legs. The differential diagnosis between the spinal and the myopathic, juvenile muscular atrophy, will be described in the appendix to this chapter.

The **prognosis** of progressive muscular atrophy is to be regarded as absolutely unfavorable. The disease appears comparatively benign only in its frequently very slow advance, since it may last for ten or fifteen years, or even longer. As we have already said, the fatal termination at last appears from some intercurrent disease, or as a result of the final appearance of dangerous bulbar symptoms, paralysis of deglutition and respiration.

The results of **treatment** are accordingly very slight. An electrical treatment, continued for months and years with very great persistence, can alone produce a little improvement or check the advance of the atrophy somewhat. Temporary improvement may also sometimes be attained by a methodical massage of the muscles and a rationally conducted gymnastic treatment. In other respects, the treatment must be symptomatic. [Gowers claims that daily subcutaneous injections of strychnine, gr. $\frac{1}{100}$ to $\frac{1}{40}$ (grm. 0·0005-0·002) often arrests the disease.—K.]

APPENDIX.

THE PRIMARY MYOPATHIC FORMS OF MUSCULAR ATROPHY.

(*Hereditary or Juvenile Forms of Muscular Atrophy. Pseudo-hypertrophy of the Muscles. Pseudo-hypertrophic Muscular Paralysis.*)

Besides the spinal form of progressive muscular atrophy, just described, there are also morbid conditions of the muscles, which develop exclusively in the muscles themselves, independently of any discoverable affection of the spinal or peripheral motor nerve-tracts, and which also lead to a very considerable atrophy and a corresponding disturbance of function in them. Besides this anatomical distinction, which is still more sharply pronounced in the special form of the disease of the muscles (*vide infra*), an essential clinical distinction between the spinal and the myopathic forms of muscular atrophy is shown by the fact that, with few exceptions, the myopathic form appears in youth, and often even in childhood, and that it very often attacks several members of the same family. We may therefore imagine that a congenital defective predisposition of the muscular system is the chief cause of myopathic muscular atrophy.

The form of this class of muscular diseases longest known is the so-called pseudo-hypertrophy of the muscles, a disease in which the actual atrophy of the muscular fibers is in part so concealed by an increase of the interstitial fatty tissue that the atrophied muscles show even an increase of volume. Griesinger in 1864 gave the first accurate description of this condition in Germany, while Duchenne first called attention to the disease in France, and in 1868 was able to give a very complete description of the clinical aspect of the disease. M. Eulenburg and Cohnheim, by the first careful anatomical examination of the nervous system, had already before this (in 1866) made out that its condition was perfectly normal.

Of late, however, we have reached the opinion that juvenile myopathic muscular atrophy need not always appear wholly in the form of lipomatous pseudo-hypertrophy, but that it may develop partly, if not exclusively, as simple muscular atrophy with a considerable loss of volume in the muscles. Erb in particular has lately described a number of cases, which have led him to establish a second special form of juvenile muscular atrophy. In fact, we can not deny that we can establish different "types" of myopathic muscular atrophy on the ground of certain peculiarities; but it is shown by increasing experience, as Erb himself has already stated, that the individual types do not differ in principle, but that they may run into one another in various ways. In what follows, the separate description of the chief types known at present is to be regarded as separate only in a clinical sense. We hold firmly, however, to the general distinction between these types and the spinal form of muscular atrophy. The constant confusion of these two forms of disease has been the main cause of the errors that obtained until a short time ago in regard to all the questions with reference to this subject. It is still advisable at present, however, especially from didactic reasons, to describe the myopathic forms directly after the spinal forms.

1. PSEUDO-HYPERTROPHY OF THE MUSCLES (*Lipomatosis luxurians muscularis progressiva* of Heller; *Atrophia musculorum lipomatosa* of Seidel).—Pseudo-hypertrophy develops almost invariably in childhood, somewhere between the ages of five and eight years. It very often depends upon a pronounced hereditary predisposition, since several children of the family are affected by the disease in the greater part of the cases. More rarely we can make out the same disease in the patient's antecedents. The male sex is decidedly more disposed to the disease than the female. Sometimes, but not always, we also find in the affected families some disposition to a nervous taint, such as hysteria, epilepsy, feeblemindedness, anomalies of the skull, etc.

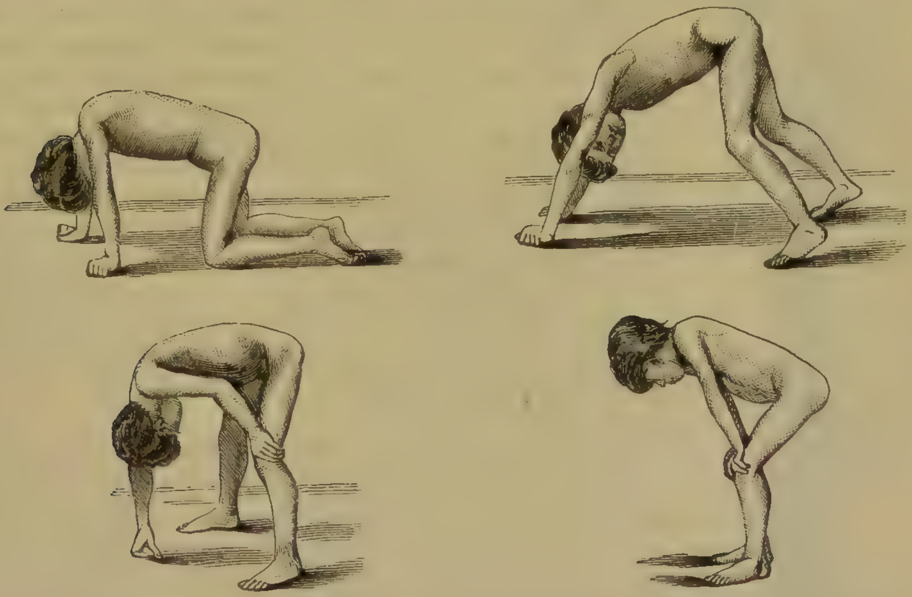

FIG. 92.—Positions of a child with hereditary (pseudo-hypertrophic) muscular atrophy, on rising to an erect attitude. (From GOWERS.)

The disease begins gradually and almost always without a special exciting cause. The parents notice that the child, who had been previously perfectly well and strong, becomes insecure on his legs, so that he can no longer jump or go up-stairs as well as he used to do. This points to the first characteristic peculiarity wherein pseudo-hypertrophy differs from progressive muscular atrophy. It begins, with rare exceptions, in the muscles of the trunk, especially in the muscles of the back and loins, and in the muscles of the lower extremities, especially in those of the thigh. While the arms and hands are still perfectly normal, walking constantly grows more and more difficult, and the gait very soon assumes so characteristic a type that from this alone the diagnosis can often be made at the first glance. The gait becomes waddling, the belly appears very prominent, the vertebral column is arched forward in the lumbar region in marked lordosis, and the whole upper part of the body is balanced on the legs. The legs are raised slowly and with difficulty, and the toes usually droop from paresis of the dorsal extensors. The child's movements, when he tries to raise himself from the floor or pick up any object, are very characteristic, and are alike in almost all cases. Since it is impossible to raise the trunk, the child usually gets on all fours first, and then gradually straightens himself up by leaning his arms on his knees (see Fig. 92). Later on disturbances of motion appear in the upper extremities also, and in general they are very similar to those to be described more fully in the next type.

If we examine the patient more closely we shall usually find at the first glance an extraordinary increase in the volume of single muscles (see Fig. 93). The calves are disproportionately thick, and sometimes the thighs also; the arms are affected later, especially the deltoids, the triceps, etc. This increase of volume is caused by an abnormal interstitial development of fat, "pseudo-hypertrophy." Hence the muscles do not feel firm, but soft and spongy. It is by no means rare, however, that, besides the pseudo-hypertrophy in some muscles, a genuine atrophy develops in others, with a pronounced loss of substance and without any coexisting development of fat. This is seen especially in the upper extremities. Finally, there seems to be in addition even a genuine muscular hypertrophy. In several cases we have seen a marked increase in volume in the muscles of the calves, which were capable of quite an extraordinary display of strength. In such cases, however, there is, probably, in our opinion, a sort of compensatory hypertrophy, since the muscles that are still able to work are exerted immoderately.

Fibrillary twitchings of the muscles can only very rarely be plainly noticed, which is probably connected with the form of atrophy (*vide infra*). Electrical examination shows a diminution of excitability corresponding to the atrophy and to the increased deposit of fat, but never reaction of degeneration. This is a fact of great importance because it agrees with the anatomical condition of the diseased muscles, and is in remarkable contrast to the occurrence of reaction of degeneration in spinal muscular atrophy. The sensibility remains perfectly normal, and also micturition and defecation; the patellar reflex was absent in some of the cases examined by us. It is noticeable that the skin, especially in the legs, very often shows a peculiar bluish marbled coloring. Bulbar symptoms probably never occur. The intelligence in most cases is perfectly normal, but it sometimes happens that children with hereditary muscular atrophy at the same time show manifest signs of mental or even of moral weakness.

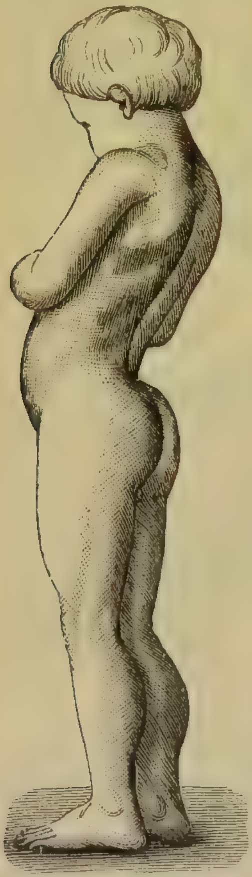
FIG. 93.—Pseudo-hypertrophy of the muscles. (From DUCHENNE.)

The disease advances very slowly but without remissions. Finally the patient can not walk at all; he is confined to the bed, and becomes more and more helpless. Death usually ensues from some intercurrent disease, but sometimes from insufficiency of the respiratory muscles.

The anatomical lesions in all cases of genuine pseudo-hypertrophy, whether hereditary or arising in childhood, which have thus far been carefully examined (Charcot, F. Schultze, Berger, and others), have been completely negative with regard to the nervous system. Except for accidental and insignificant complications, the spinal cord, and especially its anterior gray matter, have been perfectly normal. In the muscles microscopic examination shows a very considerable increase of the interstitial connective tissue, and especially of the fatty tissue between the single fibers of the muscle. This may sometimes be seen, even during the patient's life, by harpooning or excising little pieces of muscle. The fibers themselves have not undergone fatty degeneration, and they show but very little degenerative atrophy, but everywhere their transverse striation is manifest. Some of them are perfectly normal in volume, others decidedly smaller, and some are even actually hypertrophied (compensatory hypertrophy ?). We see, then, that the anatomical lesions of the muscular disease are essentially different from the purely degenerative changes in the muscles in spinal muscular atrophy.

2. Erb's Form of Juvenile or Hereditary Muscular Atrophy.—This form also begins almost always in youth, before the age of twenty, but, as a rule, somewhat later than the form associated with pseudo-hypertrophy. It is occasionally or even very often hereditary or generic (that is, occurring in families), and the female members of the family are often attacked by this form, while pseudo-hypertrophy is seen especially in boys. The disease is also, like pseudo-hypertrophy, sometimes seen to begin in the back and legs, but quite frequently the shoulders and upper extremities are first attacked. There is also a remarkable regularity in the choice of the muscles attacked. According to Erb, the following muscles are almost constantly diseased in the trunk and the upper extremities: the pectoralis major and minor, the trapezius, the latissimus dorsi, the serratus magnus, the rhomboidei, the sacro-lumbalis and longissimus dorsi, and later the triceps. The following, however, almost always remain normal: the sterno-mastoid, the levator anguli scapulæ, the coraco-brachialis, the teres major and minor, the deltoid, the supra-spinatus and infra-spinatus, and, in distinction from what was especially stated in regard to spinal muscular atrophy, the small muscles of the hand. The muscles of the forearm, too, except the supinator longus, remain intact for a long time, if not entirely. In the lower extremities the atrophy attacks chiefly the glutæi, the quadriceps, the peronei, and the tibialis anticus, while the sartorius and the muscles of the calf are usually spared for a long time. Fibrillary twitchings in the affected muscles are generally absent, and there is never any reaction of degeneration.

The disturbances of function due to this condition are self-evident, so that a full description of them may be omitted. The arms usually suffer first, as we have said. The marked projection of the scapulæ, from the paralysis of the serratus, is especially characteristic. The gait soon becomes waddling, as in pseudo-hypertrophy, and, finally, walking is utterly impossible. The whole course of the disease is always very chronic. Erb describes cases in which the disease has existed from twenty-three to thirty-eight years. Bulbar symptoms are as rare as in muscular pseudo hypertrophy. It is of significance that the diaphragm may atrophy, and the consequent respiratory disturbance may be the cause of death.

If we have an opportunity to examine the diseased muscles anatomically, we find that the interstitial development of fat is entirely absent. The connective tissue is somewhat increased, and its nuclei are more numerous than in healthy muscles. In the beginning of the disease some of the muscular fibers are hypertrophied, but simply atrophied fibers predominate. The number of muscular nuclei is considerably increased. We often see vacuoles in the individual fibers. The more the muscular substance wastes the more abundant becomes the connective tissue.

3. The Infantile, usually Generic Form of Myopathic Muscular Atrophy with Implication of the Facial Muscles. (Duchenne, Landouzy and Déjérine; see Fig. 94.)—Duchenne had already observed that in children there is a form of muscular atrophy which begins in the muscles of the face; but his statements were almost forgotten until, a few years ago, Landouzy and Déjérine called atten-

tion anew to this subject, and demonstrated, contrary to previous belief, that implication of the facial muscles is by no means an unusual symptom. Indeed, the disease very often begins in the facial muscles; the eyes can no longer be completely closed, and the movements of the mouth in whistling, laughing, and talking become impaired. From the sinking in of the cheeks, the drooping of the under lip, etc., there develops a very characteristic type of countenance ("*facies myopathique*"), by which the practiced eye can recognize the affection at once. As the disease advances the muscles of the shoulder and upper arm are usually first affected, sometimes the muscles of the back, the thigh, etc. The muscles of mastication, the internal ocular muscles, and the muscles of the forearm and hand almost always remain normal. A slight permanent contracture of the biceps is often present and is characteristic. The selection of muscles usually affected is almost precisely the same as in the previously described juvenile muscular atrophy. Fibrillary contractions and reaction of degeneration are almost always absent. We therefore believe that the two forms are not sharply to be separated, but that they are closely allied to each other. The implication of the facial muscles was in former times probably often overlooked. In order to detect it it is the best plan to have the patient close his eyes when the head is bent backward; we can then readily detect the fissure left between the eyelids (insufficiency of the orbicularis oculi). Sometimes the defective development and functions of the facial muscles seem to be a congenital condition, which may remain as such, or to which sooner or later an actual progressive muscular atrophy is added. We have ourselves recently made the especially interesting observation that sometimes we find in the relatives (brothers or sisters) of patients with pronounced myopathic muscular atrophy, signs of a congenital weakness of development of the facial muscles, such as incomplete closure of the lids, inability to whistle, etc., without any disease which progresses further.

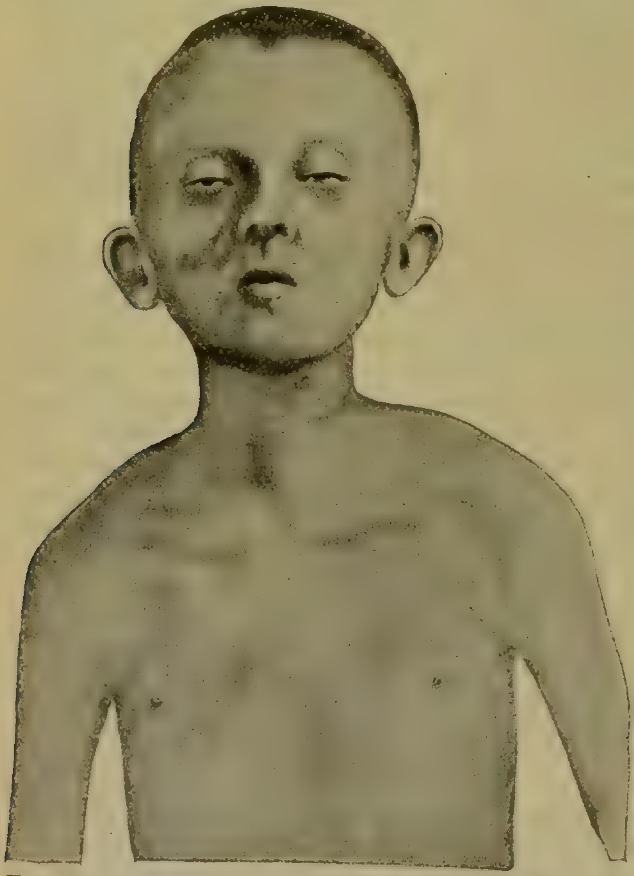
FIG. 94.—Juvenile myopathic muscular atrophy in a ten-year-old child, with marked implication of the facial muscles. Inability to close the eyes or move the lips. Atrophy of the pectorals, etc. (From the Erlangen Medical Clinique.)

That all three of the forms just described of juvenile atrophy and pseudo-hypertrophy are, at bottom, probably identical diseases, follows not only from all the other similarities mentioned, but also from the fact that sometimes one of the children affected in the family displays rather the type of pseudo-hypertrophy, another the type of simple juvenile atrophy. We also find various transitions between the different forms. Thus, for example, pseudo-hypertrophy in the legs may be associated with simple atrophy of the arms. The affection may also begin in the arms, while later the facial muscles are also affected. We do not know what causes produce the great increase of fat-tissue in a part of the cases. Even in the cases without the interstitial development of fat the atrophy of the muscles is essentially simple, not degenerative, and the motor nerves and spinal cord remain normal, at least according to all present experience. Erb groups the two forms together, under the name of "*dystrophia muscularis progressiva.*"

The differential diagnosis between the myopathic and the spinal muscular atrophies is not difficult, if we pay attention to the juvenile or generic character of the former, and also to its characteristic localization, affecting the extensors of the spine and leaving free the small muscles of the hand, etc., and to the absence of fibrillary twitchings and of reaction of degeneration in the muscles.

The treatment can scarcely ever be expected to give permanent results, but sometimes, even in juvenile muscular atrophies, some considerable improvement has been obtained by a very persistent electrical treatment and massage of the muscles.

[Charcot and Marie, and, independently of them, Tooth, have described a form of progressive atrophy, the "peroneal type," which begins in the peroneal muscles, or perhaps in the intrinsic muscles of the foot. It occurs usually in families, and begins in the second half of childhood, seldom after the age of twenty. Males are most frequently affected. There is some fibrillary twitching of the affected muscles, and reaction of degeneration may occur. The muscular atrophy may lead to club-foot. Vasomotor and sensory disturbances have been noted. It is rather apt to follow acute diseases, especially measles. The pathology is still obscure, but many writers are disposed to think it due to a peculiar form of chronic peripheral neuritis. Hence it is sometimes spoken of as progressive neuritic atrophy.—K.]

CHAPTER IX.

THE SO-CALLED SPASTIC SPINAL PARALYSIS.

(*Primary Lateral Sclerosis. Spasmodic Tabes Dorsalis.*)

In the year 1875 Erb, and soon after Charcot, called attention to a clinical form of spinal paralysis by no means rare, which is characterized "by a gradually increasing paresis and paralysis, usually advancing slowly from below upward, with muscular tension, reflex contractions and contractures, with a marked increase of the tendon reflexes, with complete absence of sensory and trophic disturbances, of vesical and sexual weakness, and of any cerebral disturbance." Both observers agreed in assuming a "primary symmetrical sclerosis of the lateral columns" as the anatomical cause of this condition.

The numerous observations published in the following years have shown that the type of disease just briefly described is, in fact, often to be met with, and may easily be distinguished from the other forms of spinal paralysis. The hypothesis as to its anatomical basis has not yet been confirmed, however, since, in all the cases that have come to autopsy so far, other anatomical changes were found instead of the primary lateral sclerosis supposed to be the only lesion; but it can not be denied that, besides other lesions, of course, a disease of the lateral columns has been repeatedly made out in such cases; and that this was certainly not without significance for the occurrence of the group of symptoms in question. It is also not at all impossible that there may be an isolated systemic disease of the lateral columns, especially of the pyramidal tracts, without any disease of the gray matter or of other portions of the cord at the same time, which disease is closely connected with amyotrophic lateral sclerosis in particular, and is a further link in the chain of primary diseases of the motor conducting tract; but, as we have said, a definite case of isolated disease of the spinal pyramidal tracts, without invasion of the anterior gray cornua, has not yet been known.

In what follows we will first describe the clinical peculiarities of spastic spinal paralysis, and then add the enumeration of its anatomical causes, as far as they are known at present. Hence we mean by "spastic spinal paralysis" only a group of symptoms which is so often observed that, from practical reasons, it is advisable to give it a short and unprejudiced name.

Type of Spastic Spinal Paralysis.—Two symptoms predominate in the picture of spastic spinal paralysis: motor paresis, and the increase of the tendon reflexes—the patellar reflex, and the ankle clonus. The former—we are speaking at present only of the spastic paralysis of the legs, which is by far the most frequent and the most clearly marked—is found in various degrees, from a simple weakness of movement to a complete and more or less extensive paralysis. It is the second symptom, however, which gives to the disturbance of motion its characteristic feature of spastic paralysis. If the increase of the tendon reflexes be very considerable, the reflex contractions come on even upon the stretching and pulling of the tendons, which is excited by the weight of the limbs or by any active or passive movements. The reflex muscular tension opposes any attempt at motion. The muscles feel rigid and firm, and the legs are often found in almost permanent contracture and extended, with the feet in plantar flexion. If we try to flex the leg passively at the knee, or if we try to extend the foot dorsally, we find it almost impossible to do so. The more rapidly and suddenly we try to produce the motion, the more marked is the muscular resistance, which can scarcely be overcome. If, however, we go to work very slowly and cautiously, and avoid any sudden tension of the tendons, we can almost always flex the leg without special trouble. If we put the patient on the edge of the bed, the legs do not hang down laxly, but they usually fall at once into a vigorous extensor tetanus, since the weight of the leg puts the quadriceps into contraction by the tension on the ligamentum patellæ. A convulsive, reflex tremor of the whole leg often comes on at once, similar to ankle clonus. If we examine the patient while in the bath we find the spasms decidedly less, because in the water the influence of the weight of the limb is absent.

Active motion, as is easily explained, must also be impaired from the inhibitory action of the reflex spasms. The degree of disturbance of motion is thus increased, and the paresis often seems greater than is really the case. The influence of the muscular tension is especially manifest in the gait of the patient. As long as walking is still possible, we notice very plainly that it is rendered difficult, not only by the muscular paresis, but also by the stiffness of the legs. The patient walks with short and difficult steps, the legs are scarcely flexed at the knee, and the feet are scarcely raised at all. The feet "stick to the floor" and are slowly slid forward, and there is a marked tendency to walk on the toes from the contraction in the muscles of the calves. The weight of the body alone presses the feet downward. We term this very characteristic form of gait the spastic-paretic gait.

The increase in the tendon reflexes may also exist without the presence of any special motor paresis of the muscles at the same time. Since, however, in this case, the motion is not a little influenced by the constant spasms, a disturbance of motility may be counterfeited, which might be called "spastic pseudo-paralysis," or, more properly, pseudo-paresis. In these cases the muscular strength is almost normal, and the patient can walk for quite a long time. Nevertheless, all his movements are stiff and difficult, and the gait shows all the peculiarities of the pure spastic gait. The steps are not very short and they follow one another quite rapidly, but the legs remain perfectly stiff and are scarcely raised from the ground, and the patient walks almost entirely on his toes. In the house the patient walks with a noisy shuffle, and in soft sand we can see the furrows drawn by the feet as they slide along the ground.

Although we are doubtless justified in referring the spastic condition mainly to the increase in the tendon reflexes, we must also add that sometimes symptoms of direct motor irritation may occur—single rapid or slow contractions, for which we can not make out a reflex origin. In some muscles there seems sometimes to be a state of constant tonic irritation; thus we often see the great toes in a permanent state of marked dorsal extension. If the patient bend his knee, a marked dorsal extension of the foot is added as an associated movement. Although the symptoms mentioned are the characteristic positive features of "spastic spinal paralysis," yet, in the original sense of the word, the affection is also characterized by the fact that certain other spinal symptoms, especially disturbances of sensibility, disturbances in micturition and defecation, ataxia, muscular atrophy, and other trophic symptoms, are entirely absent. Only when such was the case have Erb and Charcot claimed that a special anatomical cause must lie at the bottom of the peculiar group of symptoms. In fact, the cases in which we happen to see the type of genuine spastic spinal paralysis without any other symptoms are not very rare. It develops slowly and gradually, without known cause, and usually in patients in youth and middle life. One leg is first affected and then the other. The muscles of the trunk and of the arms are sometimes added to the list later on, and in the arms we find a paresis with a decided increase of the tendon reflexes and without any disturbance of sensibility or any muscular atrophy. This type of disease, however, only very rarely remains in its purity—at least, according to present experience. Sooner or later other symptoms are mixed with it, and in those cases, which so far have come to autopsy, the anatomical lesions are by no means always of the same sort.

Pathological Lesions.—As we have already said, Erb and Charcot originally advanced the hypothesis that the anatomical basis of spastic spinal paralysis is to be found in a sclerosis of the lateral columns. This opinion is well-founded, inasmuch as the picture of the symptoms of spastic spinal paralysis manifestly recalls amyotrophic lateral sclerosis in many of its relations. In both diseases we find symptoms limited exclusively to the motor sphere, and an increase of the tendon reflexes. The only distinction is in the muscular atrophy, whose anatomical cause is undoubtedly to be found in the atrophy of the anterior gray cornua. If we imagine the pyramidal tract exclusively affected, without co-existing disease of the gray matter, the result must be the picture of "spastic spinal paralysis." This line of reasoning, the justification of which must to-day be acknowledged, has, however, not yet been entirely confirmed by facts; but we have learned to recognize a chain of circumstances under which, at least at times, the symptoms of spastic spinal paralysis may arise.

We must first mention that cerebral changes, especially chronic hydrocephalus, may sometimes simulate the type of spastic spinal paralysis. Except for some anomalies in the skull, special cerebral symptoms may be entirely absent, while the motility of the legs (and arms) is diminished, and the tendon reflexes are so decidedly increased that the symptoms of spastic paralysis may result. R. Schulz and we ourselves have made such observations.

The following changes are also to be considered :

1. Transverse myelitis in the upper dorsal or the cervical cord. This sometimes shows a remarkable symmetry in its distribution for some time, and a predominant localization in the lateral columns, while the posterior columns remain comparatively free. Hence, as we can easily understand, the result is a paralysis of the legs with greatly increased tendon reflexes, but with normal sensibility. Tumors of the cervical cord may more rarely cause similar appearances.

2. *Compression of the Spinal Cord.*—A gentle compression of the cord in the cervical or dorsal region is followed, as we have seen, by paresis and increase of the reflexes, but not by sensory disturbance. We can understand that, if no manifest cause of compression can be made out, a primary affection of the cord may simulate the symptoms of spastic spinal paralysis.

3. Multiple sclerosis may also frequently show such a localization of its nodules as to be followed by paresis and spastic symptoms without sensory disturbances. The case diagnosticated by Charcot himself as "spasmodic tabes dorsalis" turned out on autopsy to be multiple sclerosis.

4. In a case observed by us, with almost the entire and pure type of symptoms of spastic spinal paralysis, the autopsy showed a hydromyelus with co-existing degeneration of the lateral columns.

5. Spastic paralysis has been sometimes observed to come on after acute diseases, but the reports of autopsies in such cases are at present wanting.

6. Finally, we will here briefly mention the combined systemic disease of the pyramidal tract, the lateral cerebellar tract, and the columns of Goll, in adults, described by us. In these cases we find a gradually increasing paralysis of the legs, and later of the arms, with increased tendon reflexes, spastic symptoms, and sensibility almost perfectly normal. Later on, however, vesical disturbances arise, which are probably to be referred to the disease of the columns of Goll. The occurrence of true ataxia in the legs, with weakness and increased tendon reflexes, seems to be not unusual; but lancinating pains, marked disturbances of sensibility, and pupillary changes are always absent, so that the morbid picture is always very materially different from that of tabes dorsalis. Further observations must give us more definite knowledge as to the frequency and the possibility of diagnosis of this apparently especially limited form of spinal disease. Minkowsky has also recently reported a case in which anatomical examination showed nothing but primary degeneration of the pyramidal and cerebellar tracts in the two lateral columns as the cause of a spastic spinal paralysis. In this and in some similar cases, which come very close to Erb's and Charcot's theoretical postulate, syphilis was perhaps to be regarded as the special cause of the disease.

Diagnosis.—The symptomatic diagnosis of spastic spinal paralysis is easy to make, with attention to the description given above. We must always, however, be very cautious at present in our anatomical diagnosis. Only the further course of the disease can give us data whereby we can first consider the morbid conditions mentioned above.

Prognosis.—The prognosis of most cases which show the symptoms of spastic spinal paralysis is just as unfavorable as most of the other diseases of the spinal cord, but we must always bear in mind that many of these cases run a very slow course. The disease seems to stand perfectly still for a long time, the symptoms are less severe than in other spinal diseases, there are no pain and no incontinence, and sometimes we have seen manifest improvement and even a few recoveries, but such cases at present lack definite anatomical proof.

Treatment.—The treatment of spastic spinal paralysis in general agrees with that of chronic myelitis (*vide supra*). Galvanic treatment usually gives comparatively the best results. We must also mention especially that prolonged warm baths often act well against the spastic symptoms. They may last for half an hour to an hour and a half, and should be of a temperature of 90°, or at most 95° (26°–28° R.). The legs are more flexible and more movable after them. Among internal remedies we may try nitrate of silver and ergotine. If there be a suspicion of syphilis, for which we should always look carefully, it is an absolute necessity to employ inunction, and to prescribe iodide of potassium internally.

CHAPTER X.

ACUTE AND CHRONIC POLIOMYELITIS.

1. Spinal Paralysis of Children.

(Acute Poliomyelitis in Children.)

Ætiology and Pathological Anatomy.—In children there is quite frequently a definite and well-characterized form of paralysis, for the first accurate knowledge of which we must thank Jac. von Heine in 1840. Although Heine later, in 1860, expressed the opinion that a disease of the spinal cord formed the basis of the paralysis, the first actual confirmation of this opinion was furnished later by Prévost and Vulpian, Charcot and Joffroy, and others, so that at present we are justified in exchanging the old term "essential paralysis of children" for the name of "spinal paralysis of children."

As the name indicates, the affection occurs chiefly, if not exclusively (*vide infra*), in children, and is most frequent in the earlier years, somewhere between one and four. An exciting cause, such as taking cold, has hardly ever been made out. The children are almost always perfectly healthy * previously, and come of healthy families without any neuropathic predisposition. The whole course of the disease makes the hypothesis very probable that we have to do with an acute infectious disease—with an infectious process, which first causes a general infection of the body, and then is localized chiefly in a circumscribed portion of the spinal cord. It also, perhaps, bears some relation to the nature of the disease as just signified that most of the cases occur in warm weather. With regard to this, it is worth mentioning that a short time ago we observed that in a little village within a few days three children fell ill with acute poliomyelitis.

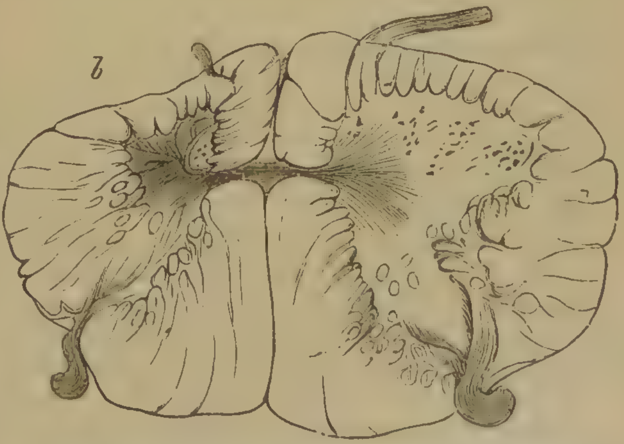

Fig. 95.—Section through the cervical enlargment in anterior poliomyelitis: the left anterior column is very much contracted and is without ganglion-cells. (From Charcot and Joffroy.)

With reference to its anatomy, the disease may be defined as an acute inflammation, which affects chiefly a definite extent of the anterior gray matter of the spinal cord, usually attacking only the anterior gray cornu of one side; yet it does not always limit itself strictly to this, but it may involve the white matter in the vicinity somewhat, although, of course, to a lesser extent than the gray matter. Although fresh cases have so far been examined in very scanty numbers, still we can sometimes make out clearly the remains of inflammation in the older centers of disease. The ordinary lesion in old cases, which is most frequently found, consists of a considerable atrophy of one anterior cornu, which is changed to a dense sclerosed tissue, often pierced by dilated and thickened vessels, and which contains scarcely a single normal ganglion-cell. If the paralysis affect one arm, the corresponding anterior cornu in the cervical enlargement is atrophied (see Fig. 95); if the leg be paralyzed, the process is seated in the lumbar enlargement. In bilateral paralysis we must think of an affection of both anterior cornua at the corresponding level of the cord.

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* The paralyses arising after acute diseases—such as measles, scarlet fever, smallpox, etc.—are perhaps partly of spinal origin, but they can not be identified with the idiopathic spinal paralysis of children.

This inflammation of the anterior cornu, the poliomyelitis, is to be regarded as the primary center of disease. From this point, as in every severe lesion of the motor ganglion-cells there situated, there develops a secondary degeneration, which, extending to the periphery, affects the corresponding anterior roots, and later their appropriate motor nerves and the muscles supplied by them. In the paralyzed muscles and nerves we accordingly find a marked pure degenerative atrophy, such as we have learned to recognize in severe peripheral paralyses.

Although at present the spinal origin of the atrophic paralysis of children is regarded as sufficiently certain, we would not deny that some authors, especially Leyden, have assumed a peripheral origin for some cases—that is, a primary neuritis, without a material implication of the spinal cord. In fact, it does not seem impossible that the same morbid ætiological factor, which we have supposed to be infectious, may exceptionally be localized chiefly in a peripheral motor nerve. In the chapter on cerebral paralysis of children, which is by no means very rare, we shall see that a manifestly closely allied acute process in children—one, perhaps, even ætiologically identical—may also develop in the motor regions of the cortex cerebri.

Clinical History.—The disease almost always begins suddenly. A child who was previously perfectly well and lively is all at once attacked with severe fever, often reaching 105° or 106° (40°–41° C.), which is associated with quite severe general symptoms even from the beginning. The child complains of headache, and sometimes of pain in the loins and in the limbs, and is decidedly stupid and somnolent. Very often still more marked cerebral symptoms develop: complete loss of consciousness, single twitchings in the face or the extremities, or general convulsions. The eclamptic attacks, turning of the eyes, and clonic contractions in the face and the extremities, often appear even at the beginning of the disease. The whole of the initial symptoms, whose intensity varies very much in the different cases, sometimes last only a very short time—a day or two—although they often continue for a week or two. Indeed, we even know cases in which, as the mothers have assured us, the children are said to have "lain in spasms," almost uninterruptedly, even for four or five weeks before the beginning of the paralysis—that is, before it became noticeable. On the other hand, however, it may happen that the initial symptoms, especially the severe cerebral symptoms, are entirely absent or only intimated.

After the initial period of the disease just described has passed away, the parents usually notice that the child is attacked by a more or less extensive paralysis. If its development can be followed more closely, we always find that it spreads rapidly, often in single spurts which rapidly follow one another, so that it usually reaches quite a great extent in a short time. Either both legs, or the legs and one arm, or all the extremities, and even the muscles of the trunk, are affected; but the paralysis scarcely ever remains permanently as extensively distributed as at first; it is reduced much more rapidly, and soon draws back to a definite muscular region, which remains permanently paralyzed. In some cases the paralysis may even entirely disappear, but as a rule a complete paralysis is left in one extremity, or at least in a portion of it; most frequently in one leg, especially in the peroneal muscles; somewhat more rarely in the arm, chiefly in the deltoid; sometimes in both legs; or, very rarely in spinal paralysis, in one arm and leg on the same side or on opposite sides. Meantime the child's general health has been completely restored. He is well and vigorous, has an excellent appetite, never shows any permanent cerebral disturbance—only the painless, flaccid paralysis, the inability to use the affected extremity, is left behind. In the following weeks and months a further, slower advance in the improvement in the power of motion often becomes noticeable, but, as a rule, a permanent and more or less complete paralysis of certain muscles remains.

In regard to the more intimate peculiarities of this remaining paralysis, it may invariably be characterized as a flaccid atrophic paralysis. A marked atrophy of the paralyzed muscles shows itself a few weeks after the beginning of the paralysis. This atrophy gradually advances further and may finally attain the highest degree. The atrophy is often, but not always, partly concealed by a more abundant development of fat tissue. The changes in the electrical excitability of the paralyzed nerves and muscles come on still more rapidly than the visible atrophy. Since we have to do with a pure degenerative atrophy of nerve and muscle, as follows from the anatomical basis of the disease, a pronounced reaction of degeneration must necessarily develop in the affected parts. Duchenne found that usually the faradic excitability of the nerves and muscles is completely lost after a week or two. On galvanic examination, we can at first detect an increase of excitability in the muscles with a predominance of slow anodic closure contractions (AnSZ), while later, after two or three months, the galvanic excitability also sinks very considerably; but the muscular contractions preserve their qualitative peculiarities characteristic of reaction of degeneration (see page 551). Very often the whole affected extremity remains backward in its growth, so that later the bones may show a shortening of several centimetres. The parallel between the muscular atrophy and the stunted growth is not, however, present in all cases, as Volkmann, in particular, has stated.

Passive motion of the paralyzed extremity is at first, and even later, perfectly free, except for the contractures that finally set in (*vide infra*). Many joints are so flaccid that we can actually make flapping movements with the paralyzed limbs and give them the most extraordinary positions. The tendon reflexes are invariably completely absent in the paralyzed extremities, and so almost always are the cutaneous reflexes—a condition which may sometimes be of diagnostic significance. The skin often shows certain trophic disturbances; it feels cool and has a cyanotic appearance. Its sensibility, however, is completely retained in all cases. Micturition is sometimes a little disturbed at the beginning of the disease, but in most cases this disturbance completely disappears later.

After the paralysis has existed for a long time, certain secondary contractures almost always develop in the paralyzed parts, which are in part of a very characteristic type. In the legs especially the "paralytic club-foot" (*talipes varo-equinus*) is a condition long known. It is due to the fact that, from the paralysis of the peronei muscles and of the tibialis anticus, the point of the foot constantly droops, and that a contracture is gradually developed in the antagonistic muscles of the calf, whose points of insertion are permanently approximated. In paralysis of the muscles of the calf there arises, on the other hand, a moderate degree of calcaneus from the contracture of the antagonists. In the arms and in the vertebral column, in paralyses of the spinal muscles, the most manifold and sometimes very considerable contractures and deformities may also arise, the chief cause of which is always to be referred to the contracture of unparalyzed antagonists and to external mechanical conditions, such as weight and pressure.

In conclusion, if we simply compare the type of disease sketched with its anatomical cause, the general agreement of the two may at once be seen. The affection of the anterior gray cornu must have as a result a paralysis with a subsequent atrophy and reaction of degeneration, in which the reflexes must be lost by the destruction of the reflex arc, but the sensibility must remain perfectly normal from the persistence of the sensory conduction (the posterior columns and the posterior gray cornua), and the vesical functions must also remain normal. The subsequent paralysis is the result of the destruction which the morbid process, in itself completely ended, has caused in the spinal cord.

Diagnosis.—The diagnosis of the spinal paralysis of children is almost always easy to make and certain if we hold strictly to the definition and peculiarities of the disease, and do not reckon as spinal paralysis every paralysis appearing in a child. We should consider chiefly the acute beginning, the subsequent flaccid paralysis with atrophy and reaction of degeneration, with the loss of the reflexes, but with retained sensibility. If we observe these features, we are sufficiently protected against confusion with cerebral diseases and other diseases such as spondylitis, hereditary muscular atrophy, or spastic spinal paralysis.

Prognosis.—It is not impossible, but it is not yet proven, that many of the cases where children die speedily with convulsions are to be regarded as the initial stage of acute poliomyelitis. If, however, the first stage of the disease be past, the prognosis as regards life is entirely favorable, since the rest of the child's physical development is no further affected in any way. The prognosis as regards the complete restoration of the disturbance of function is, however, much more unfavorable. What has not recovered in the first weeks or months usually remains paralyzed for the whole life. Nevertheless, this experience should not restrain us from persevering in treatment, at least in the first years, since sometimes a very noticeable improvement in the functions of the paralyzed parts can thus be procured.

Treatment.—If we have an opportunity, even during the initial stage of the disease (when, of course, the diagnosis can not usually be made with certainty), to attack the disease by our treatment, we may prescribe cold compresses or an ice-bag to the head, and eventually, where there is high fever or great stupor, a tepid bath with cool affusions. We are but rarely led to try local bloodletting by leeches behind the ears or on the temples, where there are signs of marked cerebral hyperæmia. Internally we usually prescribe a mild "intestinal derivative," such as half a grain or a grain (grm. 0·03–0·05) of powdered calomel every two or three hours, infusion of senna, etc.

After paralysis appears, we can expect the most success from electrical treatment, kept up consecutively for months, and, with interruptions, for years. We put a large, broad electrode on the vertebral column at the spot which corresponds to the place of the lesion in the spinal cord—on the cervical vertebræ in paralysis of the arm, and the lower dorsal in paralysis of the leg—while the other electrode serves for peripheral application to the paralyzed nerves and muscles. In this way we apply a moderately strong constant current, reversing it occasionally, for two or three minutes, partly stabile and partly by passing the cathode, or eventually the anode, slowly over the paralyzed muscles and nerves. We may also employ occasional interruptions and changes of the current. Duchenne has also found persistent treatment by the faradic current of advantage. The sittings should take place three or four times a week, and later even oftener, if possible.

Besides electrical treatment, methodical gymnastic exercises of the muscles, that can still be moved somewhat actively, may be of distinct advantage. Regular and persistent massage of the muscles is also to be recommended in the later stages. In practice we can not avoid prescribing certain embrocations, such as spirits of camphor, spirits of mustard, or spirits of formic acid. Passive motion is very important to guard against contractures, and to improve the already existing deformities. In regard to the further details of orthopædic treatment, which is of great importance, we must refer to the appropriate special works on surgery and orthopædics.

The use of baths, of brine or ferruginous waters, is to be recommended, although, of course, they must not be overvalued. They may be given at home. If circumstances permit sending the child to a bath during the summer months, we should chiefly consider the brine baths at Reichenhall, Kreuznach, Kösen, and Colberg; the acid salines at Rehme, Nauheim, and Soden; and eventually, with weak and anæmic children, the use of the iron baths at Driburg, Pyrmont, or Schwalbach. Good results are sometimes obtained at the indifferent thermal baths at Teplitz, Wildbad, Ragatz, or Gastein, but these must be used only with caution. We also obtain good results, especially with older children, at the cold-water cures.

Very little is to be expected from the use of internal remedies. Iodide of potassium and strychnine are recommended, the latter in the form of subcutaneous injections, $\frac{1}{60}$ to $\frac{1}{20}$ grain (grm. 0·001–0·003) daily.

In old cases, where there is no longer any hope of further improvement of the paralysis worth mentioning, the treatment may be limited to keeping up and strengthening the patient's general condition as much as possible by proper food and good air.

2. Acute Poliomyelitis of Adults.

(*Acute Atrophic Spinal Paralysis of Adults.*)

Although it had been believed for a long time that the form of acute atrophic spinal paralysis, just described, occurred only in children, later observations by Moritz Meyer, Duchenne, Erb, F. Schultze, F. Müller, and others, have established the fact that precisely analogous cases of disease may also develop, although decidedly less frequently, in adults, especially in young persons under thirty. There is no longer any doubt of this fact, especially if we consider an undoubted anatomical lesion found by F. Schultze. We have, however, already stated that for a long time we have regarded the diagnosis of acute, and, as we shall soon see, of chronic poliomyelitis also, as too readily made, and that certainly very many of the cases diagnosticated and published as poliomyelitis are to be classed as primary neuritis (see page 582). Since we know that primary degenerative processes may develop acutely and subacutely in the motor nerves, and that these also lead to an atrophic paralysis, a greater part of the teaching on poliomyelitis needs new and careful revision in order to exclude what does not belong to it.

The type of acute poliomyelitis of adults, so far as it has been established by definite observations, which at present are not numerous, is, of course, not materially different from the type of the spinal paralysis of children.

We often can not make out any ætiological conditions; sometimes exposure to cold, overexertion, etc., seem to favor the development of the disease. Cases are seen more frequently in the male sex than in the female.

The disease likewise begins with quite severe initial symptoms, fever, headache, somnolence, delirium, and vomiting, which may last from a few days to a week or two. The violent spontaneous pains which are very often reported as occurring in the loins, the back, and the extremities, usually belong probably to those cases in which a primary neuritis, but not a poliomyelitis, is the chief anatomical lesion. After the end of this stage the paralysis appears. This develops with varying distribution, usually in single spurts, but always rather rapidly. The paralyzed muscles are perfectly flaccid, the cutaneous and tendon reflexes are wholly absent, and very soon a pronounced atrophy and reaction of degeneration appear, while the sensibility and the vesical and sexual functions remain normal.

The distribution of the paralysis shows certain peculiarities, which must be briefly described here, since they can be studied much better in adults than in children. The paralysis may be very extensive, it may affect all four extremities, or it may occur in the form of paraplegia, or even of monoplegia. In the extremities we very often find certain groups of muscles paralyzed, to which E. Remak first called attention. Since the muscles that are paralyzed at the same time are not supplied by the same peripheral nerves, but usually are connected in their functions, we may suppose that the corresponding ganglion-cells in the anterior cornua of the spinal cord also lie together, without regard to the distribution of their peripheral processes in the different motor nerves. Thus, for example, it is worthy of note that, in paralysis of the crural region, the sartorius often remains entirely free; that in the leg the tibialis anticus, on the one hand, and the peronei and the extensor digitorum on the other, may be separately diseased; that in the forearm the supinator longus, supplied by the radial nerve, remains free, while all the other muscles on the extensor side of the forearm are paralyzed ("forearm type" of E. Remak); and that, on the other hand, the supinator may be paralyzed alone or together with the biceps, brachialis anticus, and deltoid ("upper-arm type" of E. Remak). This latter form of paralysis is said to correspond to a lesion in the cord at the level of the fourth and fifth cervical roots, the forearm type to a lesion at the level of the eighth cervical and first dorsal roots. According to Kahler and Pick, the center for the muscles of the calf lies at the level of the fourth and fifth dorsal roots. Ferrier and Yeo, in their experiments on monkeys, by irritation of the anterior motor spinal roots have obtained results which, for the most part, agree very well with the observations on men (*vide supra*, page 624).

In regard to diagnosis in the future, especial attention must be paid to the distinction between poliomyelitis and neuritis. The greatest stress is to be laid on the initial pains and any other slight disturbances of sensibility. In other respects, the course of the two diseases is so similar that we can indeed imagine that they are closely allied in their ætiological relations, and exhibit merely different forms of localization of the same (probably infectious) morbid agency. Some observations also seem to favor the theory that transitional forms may occur with a coexisting primary lesion of the cord and of the peripheral nerves.

The prognosis is not wholly unfavorable, as sometimes a complete recovery has been observed, although only after months. Of course, it is not certain whether these cases were not multiple neuritis. On the other hand, however, the same permanent paralyses as in spinal paralysis of children may be left, with atrophy and contractures.

The treatment follows precisely the same rules that we have mentioned in the spinal paralysis of children. The internal or subcutaneous use of ergotine may be added on the recommendation of some physicians. F. Müller recommends a solution of two and a half drachms of ergotine (grm. 10) with a third of a grain of sulphate of atropine (grm. 0·02) in five drachms of water (grm. 20), of which he injects seven to fifteen minims twice a day.

3. SUBACUTE AND CHRONIC POLIOMYELITIS.

(*Subacute and Chronic Atrophic Spinal Paralysis. Paralysie générale spinale antérieure subaigue* [Duchenne]).

While the anatomical basis of acute poliomyelitis in adults is still lacking in proof, our anatomical knowledge of the occurrence of a subacute and chronic poliomyelitis, in the sense of the term given it by various authors, is still completely defective. Confusions with multiple neuritis are also undoubtedly very common here, and the diagnosis is not incontestable in all the cases published under the name of "subacute poliomyelitis." Therefore we will limit ourselves to reproducing here briefly the picture of the disease at present described under the above name, while we especially repeat that a certain and accurate confirmation of its anatomical basis must be left to the future.

In the cases classed under this heading a paralysis, first of the two legs and somewhat later usually of the two arms, develops in a comparatively short time—in the course of some days, or weeks at most. It usually has no special cause or any severe initial symptoms. The patient complains at first of weakness in the legs; he can no longer walk, and is confined to the bed. A short time later the same disturbances appear in the arms, and lead to a more or less complete paralysis. The patient often feels some slight paræsthesia in the affected parts, but in general the sensibility remains perfectly normal. The paralyzed muscles are often quite sensitive to pressure (neuritic symptoms ?). Soon after the paralysis an equally extensive atrophy develops, and a distinct loss of electrical excitability, running parallel to it; which passes over into a partial or, in all severe cases, a complete reaction of degeneration. The cutaneous and tendon reflexes are very much diminished and often entirely lost. The bladder and rectum, however, remain intact, and bedsores never develop. We sometimes notice a striking diminution of the sweat secretion. In rare cases the muscles of the neck, the lips, the tongue, and the pharynx are attacked by the disease.

After the paralysis has reached its greatest extent there is usually a cessation. The condition remains stationary for months sometimes, and then a gradual improvement begins, which may go on to complete recovery; but often, of course, the recovery remains incomplete, so that the patient has a more or less marked disturbance of function for life. The "middle form of chronic poliomyelitis" described by Erb, in which there is only a partial reaction of degeneration in the paralyzed muscles, almost always gives a good prognosis. Those rare cases, however, in which the muscles of deglutition and respiration are involved, may have an unfavorable termination, although even then the possibility of an improvement is not to be entirely excluded.

Anatomical lesions, which confirm the hypothesis of a subacute (inflammatory ?) affection in the anterior cornua of the cord ascending from below upward, are to be found, as we have said, only in an extremely small number of cases, and in part of them even they are not entirely trustworthy. A short time ago Oppenheim published an undoubted case of chronic poliomyelitis, which ran its course to a fatal termination in three years. There was finally paralysis and atrophy of the muscles of all four extremities without a trace of sensory disturbance. The anterior cornua of the cord were throughout markedly diseased, while only very slight changes were found in the peripheral nerves. Clinically, the forms of disease belonging in this category are well characterized, and are easy to diagnosticate with proper attention and knowledge. Further investigations must be made as to its anatomical basis and its relations to acute poliomyelitis and the primary neuritides.

As follows from the above description, the treatment is by no means fruitless, and electrical treatment especially may produce the most complete and rapid regeneration of the affected parts.

CHAPTER XI.

ACUTE ASCENDING SPINAL PARALYSIS.

(*Paralysis ascendens acuta. Landry's Paralysis.*)

In the year 1859 Landry described a disease under the name of "*paralysie ascendante aigue*," which is chiefly characterized clinically by the fact that first the lower and soon after the upper extremities, and finally a number of the muscular regions supplied by the medulla, are attacked by a rapidly advancing paralysis, while the sensibility and the functions of the bladder and rectum remain normal. In many cases the disease terminates fatally. Examination of the nervous system has so far, however, shown no lesion which can be regarded with certainty as the anatomical cause of the disease. Considering the continued and quite numerous observations of the disease, it seems questionable, at any rate, whether we can establish a uniform anatomical basis for it. The diversity of many symptoms (*vide infra*, the condition of the reflexes, the condition of the electrical excitability) points rather to the fact that the seat of the disturbance is not always the same. Nevertheless, we can not doubt the clinical resemblance of most cases, and we must regard it as possible that the same cause of disease does not always need exactly the same localization to provoke the disease. We may very well recognize the ætiological unity of "acute ascending paralysis," without claiming that all cases also agree completely in the clinical anatomical details.

General Symptomatology.—Acute ascending paralysis attacks chiefly persons previously strong and healthy in youth or middle life, somewhere between twenty and thirty-five years of age. Some cases have also been seen in children and older people. The disease seems to be more frequent in men than in women.

The affection almost always begins with certain prodromata. These consist of general malaise, moderate febrile symptoms, headache, loss of appetite, and quite frequently of dragging and tearing pains in the back and the extremities. After these symptoms have lasted some days, or more rarely some weeks, during which they are comparatively slight, or so severe that many patients are already confined to the bed, there usually comes on quite suddenly, or sometimes more gradually, a paresis, first of one but very soon of the other leg, which rapidly increases, and usually in a few days leads to an almost complete motor paraplegia.

The paralysis is flaccid in almost all cases. The legs may be moved passively without any muscular resistance, and the muscles show neither active nor reflex tension. Their electrical excitability remains perfectly normal in many cases, but there is sometimes a rapid loss of faradic muscular excitability. It is not yet proven whether complete reaction of degeneration occurs. The reflexes, both cutaneous and tendon reflexes, seem to be diminished or wholly lost in the majority of cases, but some exceptions to this rule have been known.

Sensibility is sometimes perfectly intact, but slight alterations do occur, and quite rarely there may be even marked anæsthesia. At times a noticeable delay of sensation is observed. We find no changes in the nerves of special sense. There is occasionally a slight œdema in the legs, which is perhaps to be regarded as a vaso-motor disturbance. The marked sweating, from which many patients suffer, is also worthy of mention. The bladder and rectum in most cases are not at all affected, or they present merely slight and temporary disturbances.

A short time after the legs are attacked the arms also begin to be paretic. A marked motor weakness appears first in one, then in the other arm, and this may also increase to almost complete paralysis. The sensibility, the reflexes, and the electrical excitability show conditions like those in the lower extremities. The muscles of the trunk are also affected at the same time as, or still earlier than, the arms. The patient can no longer sit up in bed, turn on his side, or make similar movements. In some cases a paralysis of the muscles of the neck has also been observed.

The third and last stage of the disease is characterized by the appearance of respiratory disturbances and bulbar symptoms. Manifest signs of a beginning respiratory paralysis appear; the respiration is labored and difficult, the movements of the diaphragm grow less, and the paroxysms of coughing are weaker. Disturbances in swallowing, difficulty in articulation, and paresis of the soft palate and the lips may set in. In a few cases a facial paralysis and disturbances of the ocular muscles have been observed. The condition grows worse acutely, and, as we have said, in many cases death ensues.

Besides the symptoms thus far mentioned, referable to the nervous system, we find certain other symptoms in almost every case, which are less striking, but yet of greater significance in judging of the disease. The first of these is fever. The temperature is usually elevated from the beginning; it may temporarily show quite a considerable increase, up to 104° (40° C.), and later it varies somewhere between 100° and 102° (38°–39° C.), but between times it may even sink to normal. Of the internal organs the spleen shows the most frequent changes. It is usually swollen moderately, but still it is manifestly swollen. There is also sometimes a slight albuminuria.

In the cases with a fatal termination the whole duration of the disease is sometimes only a few days, and as a rule a week or two, or rarely more. Fortunately, however, all cases do not terminate fatally. The disease may come to a standstill at any time, even if the most threatening symptoms be present. Then the paralysis shows no further advance, the disturbances present disappear, and recovery ensues after a course of several weeks. It is, of course, usually quite a long time before the patient again feels himself in possession of his full powers.

Pathological Anatomy and Pathogenesis.—If we consider the whole picture of acute ascending paralysis, the idea is necessarily forced upon us that we have to do here with an acute infection of the body, with a predominating localization in the motor nervous system, an opinion which was first expressed by Westphal. The beginning of the disease with general malaise corresponds perfectly to the prodromal stage of many other acute infectious diseases. The fever, the acute splenic tumor, and the occasional albuminuria can also scarcely be explained in any other way, according to our present views, except by the above hypothesis.

The anatomical examination has, of course, as yet brought no absolute proof of this theory. A notable case, published by Baumgarten, in which many rods, like the bacilli of splenic fever, were found in the spinal cord, is at present wholly unique; but the completely negative anatomical lesions in many cases seem to point to the fact that we must look for the cause of the severe nervous symptoms chiefly in the disturbance of function excited by a toxic (infectious) influence. We have already signified that the point of attack of the infectious agent need not always be precisely the same. The condition of the reflexes and the rapid loss of electrical excitability, in connection with the pains at the beginning, seem to justify the hypothesis that the disturbance sometimes has its chief seat in the peripheral motor nerves, that the disease then exhibits the most acute form of infectious "multiple neuritis" (*vide supra*). More accurate anatomical investigations directed to this point will perhaps procure some positive support for this theory. In other cases, however, the motor portions of the spinal cord, the lateral columns, and the anterior gray cornua, are perhaps chiefly affected. This idea is supported by the occasional discovery (R. Schulz and F. Schultze, Von den Delden) of an acute myelitic affection in the parts named.

Diagnosis and Prognosis.—In every paralysis of the lower extremities beginning acutely and accompanied by general symptoms and fever we must consider the possibility of an acute ascending paralysis, but only the further course of the disease can decide the question. Inasmuch as only a well-characterized clinical group of symptoms is meant by the above term, the diagnosis is always easy to make, with attention to the peculiarities given above. It is more difficult, however, to decide accurately whether the case corresponds rather to the type of an acute multiple neuritis or to the type of an acute ascending spinal paralysis. We can judge as to this point only by careful attention to the single symptoms, especially the condition of the sensibility (pains, anæsthesia), of the reflexes, and of the electrical excitability.

The prognosis must at first be made with great reserve, and we must especially bear in mind the possibility of a rapidly fatal termination. If the first acute stage pass off fortunately and there be a decided cessation in the extension of the symptoms of paralysis, the prognosis is quite favorable, for we may then expect that the patient will be completely restored.

Treatment.—We can not be certain whether an energetic "derivative treatment" is of advantage in the beginning of the disease. Dry cups along the vertebral column are recommended, and even the use of the hot iron to the back. We would hardly advise the latter. It may be recommended, however, to prescribe an inunction with mercurial ointment, thirty to forty-five grains a day, as in antisyphilitic treatment. Of internal medicines we may give iodide of potassium or ergotine. It also seems to be a good plan to begin galvanic treatment early, galvanism to the spine and peripherally. If threatening attacks of respiratory insufficiency come on, electrical excitement of the phrenic nerve and of the respiratory muscles sometimes affords relief to the patient.

If the symptoms be arrested, electrical treatment and the use of baths may do most to hasten convalescence.

CHAPTER XII.

NEW GROWTHS OF THE SPINAL CORD AND OF ITS MEMBRANES.

Pathological Anatomy.—Tumors of the spinal cord are rare. The commonest primary new growth is the glioma, which probably arises from the neuroglia, and is a cellular and vascular tumor. We often find in gliomata secondary softening (the formation of cavities, see the following chapter) and hæmorrhages. The tumor is situated most frequently in the cervical or upper dorsal cord, and may have a considerable longitudinal extent, and a transverse diameter of several centimetres.

Of other new growths in the spinal cord we may mention solitary tubercles, syphilomata, and myxomata (myxo-sarcomata).

In the spinal meninges have been found sarcomata, fibromata, lipomata, myxomata, and syphilomata. A carcinoma arising from the vertebræ may also reach the spinal meninges by direct invasion. Marked signs of compression, and the consequent secondary degenerations, often show themselves in the spinal cord at the point where a new growth is situated in the meninges.

We know practically nothing of the ætiology of new growths in the spinal cord. It is merely worthy of note that in the cases of glioma of the spinal cord observed, an injury, such as a fall on the back, etc., very often preceded the appearance of the first symptoms.

Symptomatology.—A general description of the tumors of the spinal cord can not be given, since, of course, the individual symptoms must differ in almost every case, according to the seat and the extent of the new growth.

In tumors of the meninges the symptoms of compression of the cord are often quite prominent. In the beginning we notice pronounced "root symptoms"—that is, shooting pains, stiffness, paræsthesia, anæsthesia, etc. Later on the results of the compression of the cord show themselves: motor weakness, which may increase to a complete motor and sensory paraplegia. We can not here go more fully into the details. They follow of themselves from attention to the general laws to be considered for localization of lesions in the spinal cord.

In tumors of the spinal cord marked symptoms of sensory irritation are usually absent at first. A complicated type of spinal disease gradually develops, in which all those symptoms may be present in a single case which we have learned to recognize more exactly in the description of diffuse chronic myelitis. In fact, the differential diagnosis between tumor and transverse myelitis is often impossible, but certain peculiarities in the type of disease are sometimes present, which at least turn our suspicions to the possibility of a tumor. Among them especially is the early asymmetry of the symptoms on the two sides. Since a tumor may at first be confined to one half of the spinal cord (which scarcely ever happens in myelitis), the signs of a unilateral lesion of the spinal cord (*vide infra*, Chapter XV) are often observed in tumors in a more or less pronounced fashion. A certain change in the symptoms, improvements, and new and quite sudden changes for the worse, are sometimes noticed, a circumstance which is probably to be referred to a change in the fullness of the vessels, or to hæmorrhages in the substance of the tumor. The diagnosis of a tumor of the spinal cord, however, can be made best only with a certain probability. The decision as to the seat and the extent of the tumor is based upon precisely the same rules as in the diagnosis of the different forms of myelitis. We can hardly ever predict anything definite as to the kind of tumor.

The prognosis of tumors of the spinal cord is utterly unfavorable. The course of the disease is often protracted for several years, but the final termination is always fatal, from general weakness, cysto-pyelitis, and bedsores. The treatment is purely symptomatic, and is the same as in chronic myelitis. If there be a suspicion of a previous syphilis, we must try inunction and the internal exhibition of iodide of potassium. [In a few cases, where the growth has not involved the substance of the cord, it has been successfully removed by surgical interference.—K.]

CHAPTER XIII.

THE FORMATION OF CAVITIES AND FISSURES IN THE SPINAL CORD.

Pathological Anatomy and Pathogenesis.—The abnormal formation of cavities in the spinal cord either arises from a dilatation of the central canal (hydromyelus), or it develops outside of the central canal, and near it (syringomyelia).

The cases of pure hydromyelus are recognized by the fact that the cavity is found in the middle of the cord, corresponding to the position of the central canal, and that its walls are covered by cylindrical epithelium. Slight degrees of hydromyelus, in which the dilated central canal has a diameter of a millimetre, or a millimetre and a half, are quite frequently found. The dilatation usually extends over only a portion of the spinal cord. Higher degrees of hydromyelus, where the central canal is dilated to a diameter of half a centimetre, or a centimetre, are much rarer. In such cases the substance of the cord suffers from the internal pressure on it.

In regard to the origin of hydromyelus, following Leyden's example, we may consider anomalies of development in the formation of the central canal to be a cause in at least a part of the cases. Certainly only exceptionally do we have a process of stasis, as Langhans has found in some cases, which may have its origin in an increased pressure in the posterior fossa of the skull, from tumors, etc.

As to most cases of syringomyelia, however, the discoveries of Westphal, Simon, and F. Schultze leave scarcely a doubt but that they arise from a destruction of proliferated masses of neuroglia. We have the formation of a central glioma, probably arising usually from the ependyma of the central canal itself, or from its vicinity, with a secondary disintegration and the formation of a cavity. In these cases we can make out the newly formed masses of neuroglia about the cavities, either proliferating or disintegrating. The cavity is usually situated quite close to the center, and extends most frequently into the substance of the posterior columns. In its longitudinal extent it may involve a great part of the cord.

Clinical Symptoms.—We can not give a uniform picture for the formation of cavities in the spinal cord, since the symptoms, of course, must vary very much, according to the seat and the extent of the change. Slight dilatations of the central canal may run their course entirely without symptoms. In the cases of extensive cavity formation, with much damage to the surrounding substance of the cord, there usually arise a severe and complex array of spinal symptoms, whose correct interpretation can hardly ever be made with certainty during the patient's life. If the posterior columns and posterior cornua be chiefly involved in the cavity formation, the results of the disturbance of function of those parts are especially prominent. In the celebrated case of general anæsthesia which Späth and Schüppel have described, a very extensive syringomyelia was found in the spinal cord on autopsy. In other cases there exists a form of disease which resembles spastic spinal paralysis, or there develops a composite spinal affection in which the symptoms of unilateral lesion are to a degree prominent. In all such cases the diagnosis can be made with a certain probability by excluding other chronic morbid processes in the cord. A very slow course, extending over years and years, is especially worthy of consideration.

In one class of cases the symptoms of syringomyelia are more characteristic, and here, with due attention and knowledge, we can, in fact, often succeed in making a correct diagnosis. By many observations by F. Schultze, Kahler, and others, it has been proven that central gliosis with secondary cavity formation very often begins in the cervical cord, and gives rise to so characteristic a type of disease that the process may often be diagnosticated with great certainty during the patient's life. The morbid symptoms appear in the upper extremities. A gradually increasing muscular atrophy sets in, with a corresponding loss of strength and power to work; this is often precisely like true spinal progressive muscular atrophy in the small muscles of the hand, the forearm, the deltoid, etc. With this are next associated peculiar disturbances of sensibility, chiefly in the domain of the temperature sense (anæsthesia to heat and cold) and of the pain sense (analgesia), while the tactile sensibility for a long time remains normal. Trophic disturbances are often observed and are very interesting; among them are atrophy of the finger-tips, thickening of the joints, changes in the nails and skin, etc. Felons and similar inflammations, from which the patients often suffer, are either connected with these trophic disturbances or are due to external injuries, such as burns as a result of analgesia, etc. Anomalies of the sweat secretion have been repeatedly observed. The lower extremities remain for a long time normal; later spastic and paretic symptoms may come on in the legs.

With this type of disease, which we can usually distinguish sufficiently well from the very similar amyotrophic lateral sclerosis, progressive muscular atrophy, multiple neuritis, etc., and which has something very characteristic, as we can confirm from our own experience, we can make the diagnosis of syringomyelia of the cervical cord. The onset of other clinical symptoms corresponds, of course, to the further slow progress of the anatomical process.

The prognosis is, of course, always unfavorable, but the course is very slow, and there are long-continued apparent cessations of the disease.

The treatment is purely symptomatic, and follows the same rules as in chronic myelitis.

[MORVAN'S DISEASE.—*Analgesic Paresis with Panaritium.*—Morvan, a Breton physician, has described an affection which seems quite common in a small district in Brittany, some twenty cases having occurred in a population of 50,000. One or two cases have been reported in America. It is characterized by severe pains at the outset, paresis with analgesia, and the formation of panaritia (felons), which are usually painless. There is also tactile and thermal anæsthesia. The presence of tactile anæsthesia is considered the distinction between it and syringomyelia, but more recent observations indicate that the trouble is merely syringomyelia associated perhaps with neuritis.—K.]

APPENDIX.

SPINA BIFIDA.

(*Hydrorrhachis. Myelocele. Meningocele.*)

We give the name of spina bifida to a congenital fissure-formation on the posterior side of the vertebral arches, due to anomalies of development, and associated with a hernia-like protrusion of the sac of the dura. The most frequent seat of the malformation is in the sacral and lumbar regions. Only rarely is the tumor so great as to hinder the birth of the child. Children afflicted with spina bifida are usually born normally, and only after delivery do we find the tumor in the sacral region; its size may be from that of a small nut to that of the fist or larger. The skin above the tumor is sometimes entirely normal, but in other cases very tense and reddened. If we have an opportunity to examine the tumor carefully anatomically, we usually find beneath the skin the protruded sac of the dura, and beneath it the arachnoid. Only rarely is the dura also fissured, so that the sac is formed exclusively of the arachnoid. It is filled with a clear fluid which is precisely identical with the cerebro-spinal fluid. In rare cases there is also a dilatation of the central canal, hydromyelus; then the substance of the cord is atrophied to a greater or less extent, and the central canal communicates directly with the cavity of the spina bifida. In other cases the condition of the cord is normal; sometimes its lower end is adherent to one spot of the sac. We must refer to the text-books of pathological anatomy in regard to the many further details of the anatomy and the history of development.

In regard to the clinical symptoms of spina bifida, the condition of most children at first is perfectly normal, apart from the malformation. The tumor itself usually feels tense. If we exert pressure on it with the hand, we can often force part of its contents back into the vertebral canal. This causes an increase of the cerebral pressure, and we notice, besides the lessening of the spina bifida, a marked expansion of the fontanelles, and also the appearance of somnolence, contractions, and changes in the pulse and respiration, which demand a speedy interruption of this rather dangerous experiment. If such symptoms do not appear at all, we can conclude that the sac is completely constricted and closed.

Only rarely, however, does the child's condition remain normal later on. The tumor usually shows a slow growth, and the results of pressure on the spinal cord or on the cauda equina gradually appear. Paralysis, anæsthesia, vesical disturbances, bedsores, etc., develop, and these symptoms finally lead to death. Still more frequently the sac bursts, or its walls inflame, and this becomes fatal from the onset of a purulent meningitis.

The prognosis of most cases of spina bifida is accordingly to be regarded as unfavorable unless we succeed in curing the disease by surgical treatment. Recovery has been brought about in many cases by methodical compression of the sac, and by puncture, with evacuation of the fluid and a subsequent injection of a solution of iodine to obtain an obliteration of the sac; but, on the other hand, the operative treatment of spina bifida is attended with many dangers, such as meningitis, so that we can note frequent bad results as well as favorable ones. We can not here go into the details of the surgical methods for the cure of spina bifida; they can be found in full in the text-books of surgery.

CHAPTER XIV.

SECONDARY DEGENERATIONS IN THE SPINAL CORD.

ALTHOUGH the secondary degenerations in the spinal cord are chiefly interesting merely from an anatomical point of view, we must briefly describe them, because, in the first place, a clinical significance has been ascribed to them in certain quarters, and also because the study of secondary degenerations has been the starting-point of all our present knowledge as to the systemic diseases of the spinal cord.

1. *Secondary Degeneration in the Spinal Cord after Cerebral Lesions.*—We already know (compare page 538) that every lesion of the great motor ganglion-cells in the anterior cornua of the spinal cord, and every permanent break in conduction in the motor nerves themselves, is followed by a secondary degeneration of the peripheral portion of the motor fibers. We assume as the reason for this, as we have seen, a "trophic influence" of the said ganglion-cells on the motor fibers arising from them, so that the latter degenerate when the conduction of that trophic influence is interrupted, or when the trophic ganglion-cells themselves are destroyed. Precisely analogous conditions exist for the first great portion of the motor conducting tract, the lateral pyramidal tract, from the cortex cerebri to the anterior cornua of the spinal cord. The great ganglion-cells of the motor portion of the cortex cerebri also exert a trophic influence on the motor fibers arising from them, which extends to the motor ganglion-cells of the spinal cord. If there be disease situated in the motor portion of the cortex cerebri itself, or in any part of the motor tract in the brain (the motor fibers of the corona radiata, the internal capsule, the crus, or the pons), by which disease the conduction is interrupted—if there be disease there, we repeat, a secondary descending degeneration of the motor fibers ensues in the whole portion below, down to, but exclusive of,

the anterior cornua of the gray matter. This secondary descending degeneration of the pyramidal tract is found correspondingly in the pyramid of the same side on which the focus of disease in the brain is situated. From this point we can trace the main part of the degeneration farther down the lateral column of the spinal cord on the opposite side (secondary degeneration of the lateral crossed pyramidal tract) (see Fig. 96), while in many cases besides we find a slighter secondary degeneration in the anterior column of the spinal cord on the same side (secondary degeneration of the anterior uncrossed pyramidal tract). As we know from Flechsig's investigations, the relative amounts of the crossed lateral fibers, and the anterior fibers that remain uncrossed, vary in individual cases within certain limits. In the cases where no anterior pyramidal tract exists—that is, where all the motor fibers pass over to the lateral column of the opposite half of the spinal cord in the decussation of the pyramids—of course a descending degeneration in the anterior column is wholly wanting. We must add, however, that in some cases a small number of fibers seem to proceed uncrossed in the lateral column, so that accordingly we may also have a slight secondary descending degeneration in the lateral pyramidal tract of the same (affected) side (Pitres).

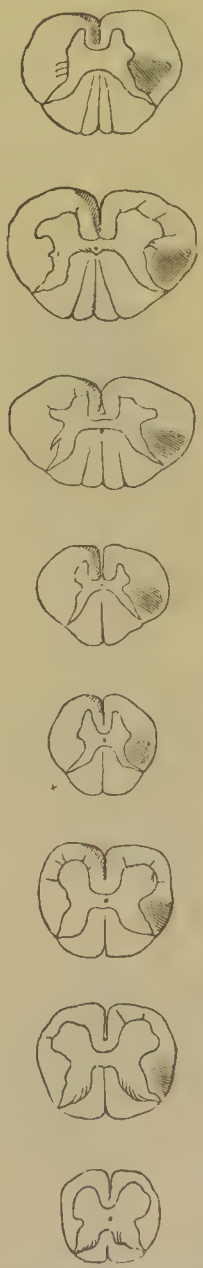

Fig. 96. — Secondary descending degeneration of the pyramidal tracts in a primary lesion of the left half of the cerebrum. The lateral pyramidal tract of the right half of the cord is degenerated down to the lowest part of the lumbar region (1-8); the anterior pyramidal tract of the left half of the cord is degenerated to the beginning of the lumbar enlargement (1-6).

2. *Secondary Degenerations in the Spinal Cord in Transverse Affections of the Spinal Cord itself.*—If a lesion be situated in any part of the spinal cord, by which more or less of its transverse section is affected, the interruption of conduction in these fibers is also followed by the appearance of secondary degenerations which may be made out both in a descending and in an ascending direction (see Fig. 97). It is most frequently transverse myelitis, compression of the spinal cord, and tumors of the cord, which give rise to secondary degenerations. The latter, however, of course, are never due to the sort of lesion, but only to its seat, and to the interruption of conduction caused by it.

The secondary descending degeneration affects the pyramidal tract in a fashion precisely analogous to that which we have also learned to recognize in secondary degenerations after cerebral lesions; but since the primary affection usually affects the pyramidal tract on the two sides, the descending secondary degeneration of course develops in both lateral pyramidal tracts, and also in the anterior pyramidal tracts, if they exist below the point of lesion.

The secondary ascending degeneration, developing upward from the primary point of disease, affects two systems of fibers, the so-called columns of Goll (the internal portion of the posterior columns), and also at the same time the lateral cerebellar tracts* on the periphery of the lateral columns and external to the lateral pyramidal tracts. Both systems of fibers mentioned, whose conduction is in a centripetal direction, must accordingly receive trophic influences from more peripheral ganglion-cells. The connection of the columns of Goll with the gray matter (spinal ganglia? posterior cornua?) is not yet accurately known. The fibers of the lateral cerebellar tracts, however, are certainly connected with the cells of the columns of Clarke. If these also be destroyed by any process in the lower dorsal and upper lumbar cord, an ascending degeneration of the lateral cerebellar tracts develops, which may be traced upward into the restiform body. The further course of the fibers to the cerebellum is not yet certainly known.

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* The area of the ascending degeneration of the "lateral cerebellar tract," as may be seen in the picture, shows at its anterior end a marked expansion. Possibly this anterior portion of the degenerated fibers corresponds to another system (Bechterew).

Although no clinical significance at all can be attributed to secondary ascending degeneration, the theory first advanced by the French observers (Charcot and others) prevails almost universally, that secondary descending degeneration causes definite clinical symptoms. The secondary contractures and the increase of the tendon reflexes in the paralyzed limbs, occurring in hemiplegia, are especially referred to this. We shall see in a later section that this theory is by no means proven, and is even improbable, so that, in our opinion, the secondary descending degeneration also has no material clinical significance.

3. *Secondary Degeneration in the Spinal Cord after Injuries of the Cauda Equina.*—After injuries of the cauda equina—for example, after fractures or caries of the lower lumbar vertebræ and of the sacrum, in new growths in this region, etc.—a secondary ascending degeneration occurs in the spinal cord, if an actual solution of continuity of the fibers have existed for a long time, which depends exclusively upon the lesion of the affected posterior root-fibers. This is accordingly limited to the posterior columns of the spinal cord, and in its distribution it shows a great resemblance to the condition of the degeneration in tabes dorsalis. In the lumbar cord the greater portion of the posterior columns is degenerated, with the exception of a little median zone and the most anterior portion (compare Fig. 87). The degeneration grows smaller upward, and finally limits itself in the cervical cord to the region of the columns of Goll. Thus this condition again affords a proof of the correctness of the statements advanced by Singer, Kahler, and others, that the columns of Goll form, at least in part, the prolongation of the fibers from the root-zones of the lumbar cord (compare page 510).

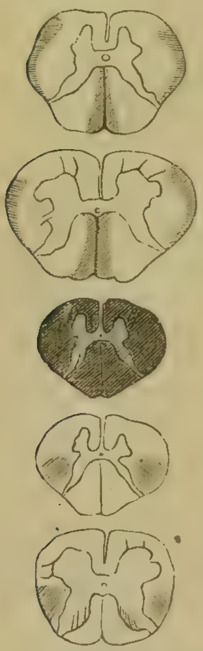

FIG. 97.—Secondary ascending and descending degeneration in a transverse affection of the upper dorsal region. The columns of Goll and the direct cerebellar tracts are degenerated upward. The lateral pyramidal tracts are degenerated downward.

CHAPTER XV.

UNILATERAL LESION OF THE SPINAL CORD.

(*Brown-Séquard's Spinal Paralysis.*)

UNILATERAL lesion is not a definite disease of the spinal cord, but a peculiar group of symptoms, which occurs whenever an interruption of conduction is produced by any affection in one lateral half of the spinal cord. Since the symptoms in these cases were first carefully studied clinically and experimentally by Brown-Séquard in particular, we often call the type of disease in question "Brown-

Séquard's paralysis." We see this paralysis most frequently and in its purest form in injuries of the spinal cord. Almost perfectly exact sections of one lateral half of the spinal cord are often produced by stabs from a knife, a sword, etc. Inflammatory processes, compression, and especially tumors of the cord, may also, during a certain period of their course, cause the symptoms of a more or less sharply defined unilateral lesion.

The peculiar condition of the symptoms in unilateral lesion is easily explained by a consideration of the course of the fibers in the spinal cord. In the accompanying diagram (see Fig. 98) the motor fibers from the anterior roots are marked *v*, the sensory fibers from the posterior roots, *h*. As we have already said, the sensory fibers, *h*, pass at once into the opposite half of the spinal cord, and accordingly decussate with the corresponding sensory fibers of the other side. The motor fibers, *v*, however, pass upward uncrossed on the side they enter the spinal cord, especially in the lateral column. If now, for example, there be situated on the right side of the spinal cord at *a* a lesion, causing a section of one half the cord, the conduction of those motor fibers which come from the right side is interrupted, as well as the conduction of those sensory fibers which come from the left side. From this it follows that there must be a motor paralysis on the same side of the body as the lesion in the spinal cord, and a sensory paralysis (anæsthesia) on the other side of the body. If the affection be situated in the dorsal or lumbar cord, the leg on the corresponding side is paralyzed, and the leg on the other side is anæsthetic. If the lesion be situated in the cervical cord, above the entrance of the nerves for the upper extremities, the arm and the leg on the side of the lesion are both paralyzed (spinal hemiplegia), while the arm and the leg on the other side are anæsthetic, but their motility is normal.*

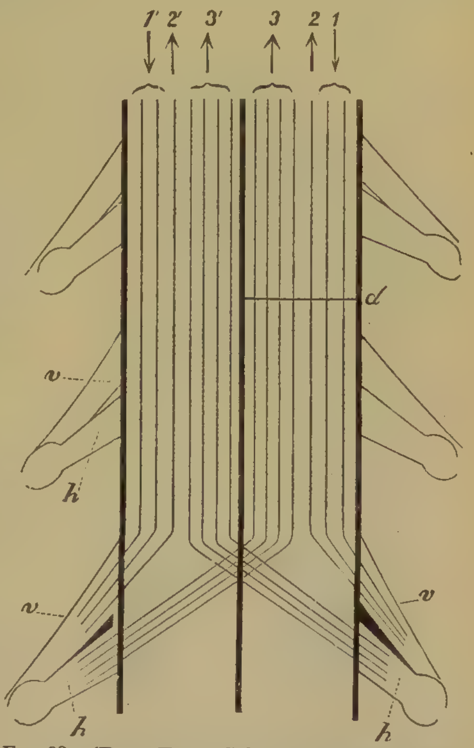

FIG. 98.—(From ERB.) Schematic representation of the course of the main tracts in the cord, represented for a single pair of roots. *v*. Anterior roots. *h*. Posterior roots. 1. Paths for motor and vasomotor conduction. 2. Paths for muscular sense. 3. Paths for cutaneous sensibility on the right. 1′, 2′, 3′. The same paths on the left. The arrows indicate the direction of physiological conduction.

On more careful examination, further conditions of physiological interest appear. The sensibility on the side of the motor paralysis is usually not only normal, but there is even a pronounced hyperæsthesia for all, or at least for some, of the forms of irritation. Slight pricks are very painful, and tickling the soles of the feet is felt with abnormal strength. The muscular sense alone (the feeling for passive motion) is a noteworthy exception, since it is usually markedly diminished on the paralyzed side. We can explain this fact only by Brown-Séquard's theory that the fibers for the muscular sensibility (see 2 and 2′ in Fig. 98), in distinction from all the other sensory fibers, run their course in the spinal cord uncrossed, just like the motor fibers.

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* [More recent investigations by Mott render this theory of immediate decussation somewhat doubtful, and unilateral lesion may not always present this group of symptoms.—K.]

Above the hyperæsthetic territory in the skin we usually find a small anæsthetic zone (Fig. 99, *b*), and above this at times again a small hyperæsthetic strip (see Fig. 99, *c*). The anæsthetic zone is easily explained. It corresponds precisely to the level of the lesion in the spinal cord—that is, to those sensory fibers coming from the same side, which are immediately involved as soon as they enter the cord; but a satisfactory explanation is entirely lacking for the appearance of the hyperæsthesia on the paralyzed side, and for the origin of the uppermost small hyperæsthetic zone.

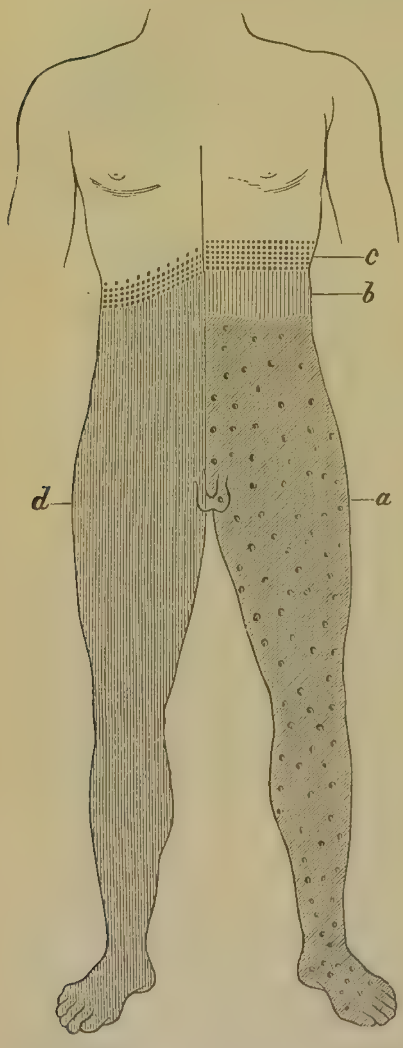

FIG. 99.—Schematic representation of the chief symptoms in unilateral lesion of the left dorsal cord. (After ERB.) The oblique shading signifies motor and vasomotor paralysis; the vertical shading signifies cutaneous anæsthesia; the dots signify cutaneous hyperæsthesia.

The reflexes, especially the tendon reflexes, are usually increased on the paralyzed side. There is often a vigorous ankle clonus, a symptom which must be explained by the loss of the reflex inhibitory influences coming from above. Finally, we often find on the side of the lesion the signs of a vasomotor paralysis, especially a marked rise in the cutaneous temperature of even 2° (1° C.), or more.

On the anæsthetic side, however, in pure cases, the motility and also the muscular sense are perfectly normal, in distinction from the other forms of sensation. Anæsthesia is not always complete, but it sometimes affects only single qualities of sensation to a greater or less degree. Thus we have often seen partial paralyses of the temperature sense, especially partial anæsthesia to cold. Above the anæsthetic region we also find frequently a small hyperæsthetic zone (see Fig. 99, *c*). The reflexes are usually normal, or only a little increased.

Of the other spinal symptoms we have still to mention the almost invariable disturbance of micturition and defecation, neuralgic pains, now more on one side and now more on the other, muscular atrophy, changes in the electrical excitability, etc. All these symptoms are not characteristic of the unilateral lesion as such, and are always easily explained in any given case from the localization of the disease. We must also mention that the symptoms of unilateral lesion may not be perfectly pure, but we can often recognize only a few prominent features of them.

We need add nothing as to the prognosis and treatment of unilateral lesion, because, of course, they are governed entirely by the form of the primary disease.

IV.—The Diseases of the Medulla Oblongata.

CHAPTER I.

PROGRESSIVE BULBAR PARALYSIS.

(*Glosso-labio-laryngeal Paralysis.*)

DUCHENNE in 1860 described for the first time with completeness the symptoms of a disease to which Wachsmuth has since given the name of progressive bulbar paralysis. Duchenne did not, however, recognize the true seat of the disease, and it was not till 1870 that Charcot in France, and E. Leyden in Germany, were enabled to confirm the suggestion of Wachsmuth that the lesion is a progressive degeneration and atrophy of the nuclei in the medulla oblongata. Since then our knowledge of the disease has grown rapidly, both from the clinical and the anatomical standpoints; and Kussmaul and others have thoroughly investigated its relations to two other closely allied forms of disease—amyotrophic lateral sclerosis, and progressive muscular atrophy.

Ætiology.—We have scarcely any certain information about the cause of the disease. Heredity seems of slight importance. In some cases its origin is ascribed to catching cold, emotional excitement, traumatic influences, and excessive bodily exertion. Perhaps it is sometimes occasioned by excessive use of the muscles to which the disease is chiefly confined, as in playing on wind-instruments; but in many instances no possible cause can be found. Men seem somewhat more liable to be attacked than women. The disease hardly ever appears till middle or old age—that is, after thirty-five.

Clinical History.—The symptoms are almost always very slow in their development. There may be mild premonitory symptoms—such as painful sensations in the back of the neck. Then there is a very gradual appearance of difficulty in articulation. Many words are pronounced indistinctly. The first trouble is noticed especially with letters in the utterance of which the tongue plays an essential part: E, R, L, S, G (hard), K, D, T, and N.[*] It is easily seen that the derangement is not aphasic. There is no forgetting or confounding of the words or letters; but the innervation of the tongue has become impaired. Long before the ordinary movements of this member are visibly embarrassed, the patient has lost the ability to make those more delicate manipulations of it which are essential to normal speech. This disturbance of articulation is termed alalia or anarthria.

By the time this has become somewhat marked, it is usually possible to detect, on close examination, that the tongue is beginning to atrophy. It seems flabby, thin, and less rounded. Here and there its surface presents furrows and depressions; and often the individual fasciculi exhibit active fibrillary contractions. Just as in progressive muscular atrophy, the impairment of motion usually keeps equal pace[†] with the atrophy. The greater the atrophy, the less is the mobility. Finally it becomes quite impossible to project the tongue from the mouth or move it from side to side. The tongue lies flat and limp on the floor of the mouth. Its surface is often diversified with furrows and depressions, containing much desquamated epithelium or the like. Evidently any great impairment of motility in the tongue hinders not only speaking, but also chewing and swallowing. The organ can no longer bring out such portions of the food as get between the cheeks and the teeth, nor can it push the bolus backward within the grasp of the pharyngeal constrictors.

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[*] [Except as otherwise specified, the letters and words used as examples here and later on are to be given the ordinary English pronunciations.—TRANS.]

[†] At the commencement of the disease the paralysis may possibly seem greater than the atrophy, so far as the latter can be detected. Nor would it be impossible for a primary lesion of the nuclei of nerves to result in a paralysis before the secondary descending degeneration had become completely developed. On the other hand, it must be borne in mind that numerous individual fibers in the lip or tongue might be already atrophied before the eye or the touch could appreciate any change in bulk.

Even before the atrophy of the tongue becomes extreme, analogous disturbances usually appear in neighboring groups of muscles. As a rule, the muscles of the lips are next affected after the tongue. The first thing the patient notices is a peculiar feeling of stiffness or tension in the lips. Movement becomes gradually more and more difficult; and the patient becomes unable to pucker up his lips so as to whistle. Speech is also noticeably interfered with, for now all those letters the pronunciation of which demands labial movements are very imperfectly articulated, and at last can not be uttered at all. These are O, A (long), P, F, B, M, and V; and also the sound of double O, as in tool. It also becomes gradually evident that the lips atrophy. They grow thin, with sharp edges and wrinkled skin. Fibrillary contractions are not infrequently visible.

This atrophy of the orbicularis oris is followed by atrophy and paresis of some of the other muscles of expression supplied by the lower division of the facial nerve. The general facial expression of a patient with bulbar paralysis thus comes to bear a very characteristic stamp: the mouth remains half open, and seems to be broadened out, the lower lip hangs down, the naso-labial folds are deepened, and indeed the whole aspect is persistently lachrymose. Even in laughing, the lower half of the face relaxes comparatively little; while the region supplied by the upper division of the facial nerve, and the movements of the eyeball, remain as a rule perfectly normal.

The third group of muscles affected are those of the pharynx and larynx. The soft palate becomes paretic, and produces further trouble in swallowing. Quite often the liquid ingesta are regurgitated through the nose. The voice becomes nasal. The production of many sounds, and in particular of B and P, is now impossible, since, in addition to the labial paresis, a portion of the essential current of air escapes through the nostrils. This explains why the letters mentioned can sometimes be pronounced better if the nose be compressed. The paralysis of the constrictors of the pharynx impedes deglutition more and more, till the impairment of nutrition becomes extreme.

The enfeebled action of the laryngeal muscles is betrayed, in the earlier stages of the disease, by a certain weakness and monotony in speaking. Modulations of the voice, and the production of the higher notes, as in singing, are no longer possible. If the innervation of the larynx becomes still more impaired, it becomes a very serious matter. If the arytænoid cartilages do not press together firmly on swallowing, the entrance to the larynx is inadequately closed, and food is often swallowed the wrong way. Liquid and even solid ingesta get into the larynx, and excite a violent cough; or, being inhaled into the air-passages, they cause bronchitis or lobular pneumonia. The paralysis may reach such a degree that the voice is at best a hoarse whisper. With the laryngoscope we can see that the vocal cords are paralyzed. The inability to close the glottis tightly is extremely unfavorable, for it renders the patient unable to cough vigorously. Mucous accumulations may therefore come to be the source of extreme dyspnœa.

The catalogue of symptoms is not yet ended. As we have seen, the muscular atrophy of the tongue and lips can invariably be detected. That of the pharyngeal and laryngeal muscles can not be demonstrated during life, although it is to be found post mortem. Inasmuch as the process is one of genuine degeneration with consequent atrophy, the affected fibers ought to give the reaction of degeneration to electricity; but this is difficult of actual proof, just as it is in progressive muscular atrophy, because numerous healthy fibers lie side by side with the degenerated ones. Still, in an advanced case, careful examination will usually bring out an evident degenerative reaction here and there in the tongue and lips.

The disturbance of reflex action is often striking. Usually the reflexes are greatly diminished or even absent, so that one can tickle the root of the tongue and the epiglottis without causing the patient to gag. In a few instances the facial muscles exhibit an increase of tendon reflex, as can be shown by tapping upon the tendons, the periosteum of the jaws, or the bridge of the nose. This behavior reminds one of the condition of the muscles in amyotrophic lateral sclerosis (*q. v.*).

Exceptionally, still other muscular groups are involved. Of such disturbances, the most frequent is in the region supplied by the motor branch of the trigeminus, impairing mastication. The impairment of these muscles now combines with the labial and lingual atrophy to render chewing almost impossible. In very rare cases the ocular muscles are also involved, with resulting ptosis and strabismus.

All the symptoms thus far enumerated are exclusively motor. Sensation is perfect to the end. The sensibility of the skin of the face and of the mucous membrane of the tongue and mouth, as well as the sense of taste, are unimpaired. Disturbances of sensation in the distribution of the trigeminus, and more or less deafness, have been reported in one or two cases ; but there is some doubt about the observations. It does, however, seem certain that secretory and vaso-motor derangements are frequent. Salivation deserves especial mention. In many cases of bulbar paralysis it is a constant symptom, so that the patient is obliged to keep a pocket-handkerchief to his mouth, to catch the fluid as it dribbles away. This is due, to a certain extent, to the fact that the secreted saliva can not be swallowed, and, as the lips do not shut tightly, it naturally escapes from the mouth ; but volumetric examinations have rendered it pretty certain that the amount of saliva is abnormally large. The explanation of this has not been determined. Nor as yet do we know much about the vaso-motor disturbances. Many patients complain of a feeling of heat and "boiling" in the head. We may also mention in this connection that occasionally, toward the close of the disease, the pulse becomes very rapid (140–160). This is probably due to paralysis of the vagus.

The course of the disease is invariably protracted. The order in which the symptoms appear is, as a rule, that in which they have just been described. The atrophy and paresis appear first in the tongue, then in the lips and the neighboring muscles of the face, and lastly in the muscles of the soft palate, pharynx, and larynx. Still, there may be some deviation from this. Usually the progress of the disease is very gradual. There may be an apparent arrest of the trouble; or less often there are quite sudden exacerbations. When all the different symptoms are well developed, the clinical picture is unusually characteristic. The peculiar immobility of expression; the broad, slightly gaping mouth, with the atrophied lips; the almost unintelligible speech, low, monotonous, and labored; and the inability to swallow—these often betray the disease at once. The last stage of the illness is the more distressing, in that the intelligence remains to the end entirely unclouded.

The entire duration of the disease is usually several years—say two to five. If death is not caused by some intercurrent trouble, it is brought about in one of three ways : either through inanition, due to the increasing difficulty of deglutition ; or through pulmonary complications—namely, bronchitis, lobular pneumonia, or gangrene, as a result of food passing down the trachea ; or through sudden asphyxia or cardiac failure.

Pathology. Nature of the Disease, and its Appearance as a Symptom of Progressive Muscular Atrophy or of Amyotrophic Lateral Sclerosis.—If we seek the anatomical lesion corresponding to the group of symptoms above depicted, we shall find, on microscopic examination of the nervous system, in all cases of this description, a typical disease of the medulla oblongata. The ganglionic nuclei and the nerves, corresponding to those muscles which we have found to undergo atrophy in bulbar paralysis, present distinct evidences of degeneration. This is most readily demonstrated in the nucleus of the hypoglossus. The ganglionic cells have some of them entirely disappeared, while others are greatly atrophied. The connective tissue is increased in amount, and the walls of the blood-vessels traversing the nucleus are thickened. In the earlier stages there are often many cells which contain granules of fat. The same changes, though perhaps less pronounced, are exhibited by the common nucleus of the vagus and accessorius, that of the facial, and sometimes also that of the glosso-pharyngeal nerve. The other nuclei are perfectly normal. We never find a diffuse "inflammation," but in every case a primary degeneration of the nuclei, which spreads no farther.

Starting from these nuclei, the degeneration and atrophy may be seen to extend into the nerve-fibers which issue from them. The roots of the hypoglossus, vagus, accessory, and facial nerves can often be seen by the naked eye to be diminished in size and of a gray color. The microscope always shows a partial atrophy of their fibers. Finally, there is a corresponding atrophy of muscles of the tongue, lips, and other parts. We need not enter into detail, for the histological conditions are precisely those seen in the muscles of the trunk and extremities in the spinal form of progressive muscular atrophy.

Thus we find progressive bulbar paralysis perfectly analogous with progressive muscular atrophy. The nuclei in the medulla oblongata are the motor and trophic centers of the bulbar nerves and of the muscles which these nerves supply. The relation is precisely that which exists between the anterior cornua of the spinal cord on the one hand, and the spinal nerves and the muscles which they innervate on the other. In both diseases there is a degeneration and atrophy of the trophic and motor center and the corresponding nerves and muscles. In both diseases the atrophy and the functional disability of the muscles keep pace with each other, and in both the affection is strictly limited to the motor tract, sensibility suffering no impairment whatever. Certain questions about bulbar paralysis are as unsettled as similar ones about progressive muscular atrophy. It is uncertain whether the primary degenerative process is limited to the bulbar nuclei, and the degeneration of the nerves and muscles is to be regarded as secondary; or whether the entire motor apparatus, from the ganglionic cell to the muscular fiber, is simultaneously attacked ; or, finally, whether, as Friedreich maintains, the atrophy begins in the muscles, and thence ascends along the nerve-fibers to the medulla. We think it improbable that these points will be cleared up very speedily. Their solution would seem to be only of theoretical interest.

We certainly must recognize, however, the essential identity of progressive bulbar paralysis and progressive muscular atrophy. The resemblance becomes even more striking if we consider that very frequently both diseases are present simultaneously. Often, after a case of progressive muscular atrophy has lasted for some time, the symptoms of bulbar paralysis also appear. And, on the other hand, an illness may begin with bulbar symptoms, and later on be complicated by atrophy of the muscles of the extremities—almost always first seen in the arms. If cases of this sort come to autopsy, we find a combination of the anatomical lesions of both diseases; in addition to the degeneration of the nuclei in the medulla oblongata, there is marked atrophy of the ganglionic cells in corresponding places in the anterior gray cornua of the spinal cord.

We must here refer again to the occurrence of the symptoms of bulbar paralysis in amyotrophic lateral sclerosis (see page 650). In this disease, too, there is the same combination of a degeneration of the nuclei in the medulla oblongata and of the anterior cornua of the gray matter of the cord; but in addition there is a derangement of the motor tract in the lateral columns. This addition modifies the picture, but otherwise the symptoms are almost precisely the same as in progressive muscular atrophy. Even the derangement of the crossed pyramidal tract is in perfect harmony with the other lesions, for it represents merely a further invasion of the tract by which motor impulses are conducted. It seems justifiable, therefore, to say that these three diseases—progressive bulbar paralysis, progressive muscular atrophy, and amyotrophic lateral sclerosis—differing as they do in the localization of their lesions, are yet closely allied. They are essentially—that is, pathogenetically, and perhaps also ætiologically—different results of one disease; or, at least, the pathological process in each case must be nearly the same. In each there is a primary chronic degeneration of portions of the chief motor tract, varying only in region or in extent. If we accustom ourselves to regard these three groups of symptoms as really identical, we shall be less puzzled by the slight variations which different cases may present than if we attempt to differentiate the disorders too nicely on account of unessential variations.

Diagnosis.—The diagnosis of a typical case of progressive bulbar paralysis has no difficulties, if we only hold firmly to the definition of the disease and its symptoms as above depicted. Upon careful examination of the other muscles, and consideration of the course of the disease as a whole, we shall be able in each case to determine whether the bulbar trouble is the sole disease, or merely a part of a more extended degeneration of the motor tract. If there are no symptoms but those referable to the medulla oblongata, we must bear in mind that the phenomena of genuine progressive bulbar paralysis may be closely simulated by other bulbar diseases. The acute troubles, like thrombosis or hæmorrhage, although they produce similar symptoms, can easily be differentiated by the manner of their appearance, contrasting with the invariably slow development of genuine bulbar paralysis. It is, however, much more difficult to eliminate gradually forming tumors situated in the medulla oblongata or its vicinity. Here prolonged observation is frequently needed, until finally such phenomena appear as are foreign to typical bulbar paralysis. Such symptoms are disturbances of sensation and invasion of the upper division of the facial, the nerves of special sense, and the ocular muscles. The same is true of that rare trouble, diffuse sclerosis of the medulla oblongata.

It should also be mentioned that bilateral cerebral trouble may occasion so complete a paralysis of the tongue and lips, according to Lépine and others, as to simulate bulbar paralysis. Such cases have been termed "glosso-labio-pharyngeal paralysis of cerebral origin," or pseudo-bulbar paralysis. Indeed, in rare instances, a similar group of symptoms seems to be referable to unilateral cerebral disturbances. This is explained by assuming that the muscles involved upon both sides receive at least a portion of their motor nervous fibers from the same hemisphere. And yet in most of these cases of pseudo-bulbar paralysis the exclusion of the genuine disease is possible, because certain variations from the typical course of the disease are pronounced enough to set us right. Thus there is an abrupt onset, the paralysis is not perfectly symmetrical, or the lips and tongue react normally to electricity.

Prognosis and Treatment.—Despite the unfavorable prognosis of progressive bulbar paralysis, we must at least try to check the progress of the disease. Electricity might perhaps be regarded as the most promising means to employ. To influence the seat of the trouble, galvanization is chiefly used. The poles are applied to the two mastoid processes, if possible, every day for two or three minutes, and the current is repeatedly reversed. We may also galvanize the sympathetic nerve and the affected muscles of the lips and tongue. Upon the muscles the faradic current might also be tried. When deglutition begins to be impaired, it is an excellent thing to excite the action of swallowing, by galvanism. For this the anode is placed upon the nape of the neck, and the kathode upon one side of the larynx. At every kathodic closure (KaS), or every time that the kathode is passed across the side of the larynx, there is a reflex act of deglutition. The current should be of medium strength.

It might be well to prescribe further a resort to treatment by baths, as at Rehme, or the "cold-water cure" might be cautiously tried. The same internal remedies are recommended as in the chronic diseases of the cord, especially argentic nitrate, ergotine, and potassic iodide. For salivation, atropine may prove beneficial, in pills of $\frac{1}{120}$ of a grain (grm. 0·0005), three or four to be taken daily.

The way of giving nourishment is important if deglutition is impaired. We should try carefully to avoid having the food go down the wrong way, lest pulmonary complications ensue. It is therefore wise not to defer the use of the stomach-tube too long, through which we may introduce milk, eggs, wine, and the various infant foods.

In the distressing close of the disease, narcotics must be exhibited to lessen the patient's suffering, at least as far as we can.

APPENDIX.

THE RARER FORMS OF CHRONIC BULBAR PARALYSIS, AND PROGRESSIVE OPHTHALMOPLEGIA.

As we have seen, the typical form of chronic bulbar paralysis is practically limited in its effects to the distribution of the hypoglossus, the labial division of the facial, and the pharyngeal muscles. Possibly the reason it extends no farther is merely that death is so speedy. But there are a few rare cases where the chronic degenerative process comes to involve other motor nuclei, together with the corresponding nerve-fibers and muscles. Of course the clinical phenomena of these cases vary from the ordinary; and yet there is no real reason to distinguish these from common bulbar paralysis, particularly as all sorts of transitional forms are to be observed. Thus, we have ourselves noticed that there is sometimes a symmetrical and slowly progressive paresis of the upper division of the facial, and in particular of that portion which supplies the cheek, complicating the glosso-pharyngeal paralysis. In other cases we have seen the degeneration attack from the start the entire distribution of the facial, gradually producing a complete "*diplegia facialis.*" Sometimes, also, the ordinary symptoms of bulbar paralysis are accompanied by disturbances in the area of distribution of the ocular nerves, the result of degeneration of the corresponding nerve-nuclei. Erb relates a few cases where there were not only ptosis and impairment in the movements of the tongue and in deglutition, but also paresis of the muscles supplied by the accessorius and the motor branch of the trigeminus.

What seems very remarkable is that the process may be confined entirely to the ocular muscles. A. von Graefe named this condition progressive ophthalmoplegia. Another name is "anterior bulbar paralysis." The disease progresses with extreme slowness and is perfectly symmetrical. The movements of the eye are impaired in all directions. Diplopia is never present. The pupil reacts to light, and usually the power of accommodation is preserved. Finally both eyeballs become absolutely motionless, and there is a well-marked though incomplete ptosis. There is, beyond a doubt, a progressive degeneration of the nuclei and fibers of the corresponding nerves—i. e., the abducens and motor oculi ; but with these the process may stop, spreading no farther. We have ourselves lately met with a patient who presented total bilateral ophthalmoplegia, and in whom this condition had existed without the slightest change for fifteen years.*

We must add, in conclusion, that our anatomical knowledge concerning these rarer forms of chronic bulbar paralysis is very incomplete. The results of a few autopsies, however, join with the clinical phenomena in strongly confirming the surmise above expressed as to the pathological lesions.

CHAPTER II.

ACUTE AND APOPLECTIFORM BULBAR PARALYSIS.

1. Hæmorrhage into the Medulla Oblongata and the Pons.

Hæmorrhage into the medulla oblongata and the pons is much more frequent than into the spinal cord, but it is much rarer than cerebral hæmorrhage. As to its production, the same views are held as will be considered in detail under cerebral hæmorrhage, in the next section. In the first place, there is probably always some disease of the blood-vessels—that is, atheroma or miliary aneurism—and then some factor productive of increased arterial tension. There may be cardiac hypertrophy, nephritis, excessive bodily exertion, or alcoholism. Now and then injuries of the occiput are followed by an effusion into the medulla. It is not rare to have secondary and usually small ecchymoses in acute inflammation of the spinal cord (*vide infra*), and in purulent meningitis or in connection with new growths which are richly vascular.

The anatomical conditions produced by bulbar hæmorrhage are so completely analogous with those of cerebral hæmorrhage that the reader may safely be referred to the succeeding section about these also. The size of the lesion varies greatly. Bleeding extensive enough to affect the greater part of a transverse section is more frequent in the pons than in the medulla oblongata. If the blood is poured out close under the floor of the fourth ventricle, as has been repeatedly observed, it may break into the ventricle. If death be not speedy, the blood is mostly absorbed, and in its place develops either an "apoplectic scar" or an apoplectic cyst.

There may be slight prodromata, but the real symptoms of bulbar hæmorrhage are very sudden. There is almost always a pronounced apoplectic seizure. The patient has a shock, falls down, and becomes dizzy or even unconscious. In other cases there may be headache, vomiting, tinnitus aurium, and clonic spasms, or even a typical epileptiform attack.

In most cases death is speedy, if not immediate. This is probably due in every instance to grave lesions of the respiratory and circulatory centers, rendering continued existence impossible. Sometimes the initial symptoms abate, whereupon the local results of the lesion become appreciable.

One of the characteristics of bulbar paralysis now seen is, that disturbances are particularly great in the distribution of the bulbar nerves. In cerebral apoplexy they never appear in the same way. Another point is, that these paralytic symptoms are combined with paralysis of the extremities in a peculiar way, as a result of the anatomical relations. For the same reason, the order of the paralysis in the extremities may also be peculiar. Of the bulbar paralyses we may mention more or less complete paralysis of the tongue, and a consequent difficulty in articulation (anarthria); frequent inability to swallow; and paralysis in the distribution of the accessorius, facial, and trigeminus. If there is a lesion of the pyramidal tracts in the pons or medulla, we have paralysis of the extremities in addition to the specific bulbar symptoms. If the hæmorrhage be extensive, all four extremities may be more or less completely paralyzed ; but in most instances the paralysis is unilateral. In the larger number of hæmorrhages into the pons there is crossed paralysis. The paralysis of the extremities is upon one side, and that of the facial is on the other side. This is a great aid to diagnosis. It is easy to see how this happens if we bear in mind that the cerebral fibers of the facial cross at a point certainly much higher than the decussation of the pyramids, in which latter place the motor fibers of the extremities cross. Now a hæmorrhage may be situated in one side of the pons, above the decussation of the pyramids but below that of the facial. This would occasion (*vide* Fig. 100, *y*) a paralysis of the facial on the same side with the lesion, and of the extremities upon the opposite side; but if the lesion be higher, above the place where the facial crosses over, all the paralytic symptoms would be on the opposite side of the body (*vide* Fig. 100, *x*).

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* It seems that total ophthalmoplegia may also appear as one of the symptoms of locomotor ataxia or of general paralysis. It has been observed by Mendel as a sequel to diphtheria. In these cases, however, the symptom is probably in part due to a degeneration of the fibers of the peripheral nerves.

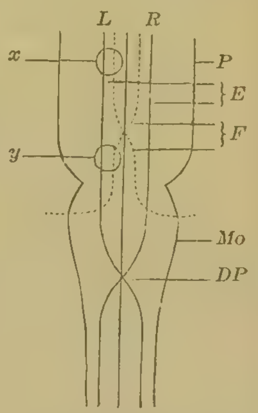

FIG. 100.—Diagram of focal diseases in the pons. *L.* Left. *R.* Right. *P.* Pons. *Mo.* Medulla oblongata. *DP.* Decussation of the pyramids. *E.* Fibers to the extremities. *F.* Facial fibers. *x.* Lesion in the upper half of the pons. *y.* Lesion in the lower half of the pons.

In other, rarer instances we observe similar combinations; only some other bulbar nerve replaces the facial, such as the hypoglossus or abducens. In a few cases the lesion is at the very decussation of the pyramids. This is extremely rare in hæmorrhage, though somewhat more frequent in troubles of a different nature. The result may be that the motor fibers for one extremity are cut off before they cross, and those of the other extremity after they have crossed. Thus is produced the rare phenomenon of a crossed hemiplegia—i. e., paralysis of the arm on one side and of the leg on the other.

Disturbances of sensation in the skin of the paralyzed extremities sometimes result from trouble in the pons, but they are very seldom extreme, and can not be made available for making out the exact locality of the hæmorrhage, since the course of the sensory fibers through the upper extremity of the spinal cord is still almost unknown. The anæsthesia sometimes observed in the distribution of the trigeminus is of more value, as this may be due to a lesion of the nucleus or root of the nerve.

There are other symptoms, which are indeed rare, but which bear an important relation to certain nervous centers of the medulla. Thus, there may be marked respiratory disturbance; the pulse may become rapid or irregular; there may be vaso-motor derangement, as shown by a rise of the cutaneous temperature and by a subjective sensation of warmth; and occasionally there are temporary albuminuria and glycosuria. The temperature of the body is generally normal at first, or nearly so; but in case of a fatal termination it often rises greatly, even to 107·5° (42° C.) and higher.

As to the prognosis of bulbar hæmorrhage, speedy death has been repeatedly observed, as we have said. If the immediate effects be successfully withstood, the prospect becomes more favorable. The effusion is gradually absorbed, the symptoms of compression abate, and there is a steady progress toward comparatively good or even perfect health. More often, however, some of the paralytic symptoms remain stationary, either in the distribution of the bulbar nerves (like the lingual or pharyngeal), or in the extremities, as shown by persistent hemiplegia. If this latter be the case, the subsequent contractions and other symptoms are the same as in ordinary cerebral hemiplegia.

The diagnosis of bulbar hæmorrhage is based upon the apoplectic onset, and upon the presence of specific bulbar symptoms, such as disturbance of speech and of deglutition, and, most characteristic of all, if it occur, a crossed hemiplegia. The differential diagnosis between embolism and hæmorrhage can hardly ever be made with certainty (*vide infra*).

The treatment, not only of the seizure but of the persistent paralysis, should conform to the principles which will hereafter be set forth in describing the treatment of cerebral hæmorrhage. If the bulbar nerves present obstinate symptoms, we must employ the same means as in chronic bulbar paralysis, the most effective being electricity.

2. Embolism and Thrombosis of the Basilar Artery.

The medulla and pons receive their blood chiefly from branches of the anterior spinal, vertebral, and basilar arteries. These branches penetrate the anterior median fissure and then proceed to the nerve-nuclei. A far smaller portion of the circulation flows through the "arteries of the roots." These are minute offshoots of the lateral branches of the basilar and vertebral arteries, which enter the cord at the roots of the nerves and penetrate to the corresponding nuclei. According to Duret, the nuclei of the hypoglossal and accessory nerves are supplied from the anterior spinal and vertebral arteries ; those of the vagus, glossopharyngeal, and auditory nerves by branches of the upper end of the vertebral arteries ; and the nuclei of the facial, trigeminus, and the three nerves to the ocular muscles by branches of the basilar. There may be individual exceptions to these rules. Occlusion by embolism or thrombosis of the arteries just named must occasion a secondary softening in corresponding portions of the medulla, and is, therefore, a not very infrequent cause of apoplectic, or at least very rapidly developed, bulbar paralysis.

The causes of thrombosis or embolism in the arteries just mentioned are the same as we shall consider minutely when treating of cerebral softening. Emboli are most frequent in cardiac disease. They occur only in the vertebral arteries, oftenest in the left one, and are never primary in the basilar artery ; but an embolus may be enlarged by thrombosis after lodging in one of the vertebral arteries, and then block up the basilar. Thrombosis is of more frequent occurrence, and results from chronic changes in the arteries, mainly atheroma or syphilitic endarteritis. The latter disease, one favorite locality for which is the basilar artery, is the commonest cause of acute softening of the pons.

The anatomical condition is likewise similar to that in cerebral softening (*q. v.*). In the region which is deprived of arterial blood by the occlusion of the affluent vessel, the acute anæmia entails necrosis and disintegration of tissue. A spot of "softening" results, made up mainly of vestiges of nervous tissue and numerous cells filled with granules of fat.

When the basilar artery is blocked up, the symptoms appear very suddenly. There is either an apoplectic seizure, or at least a very rapid development of paralysis (occupying only a few days). The symptoms of the first onset are, in all essential points, those of bulbar or even of cerebral apoplexy. Although there is usually no marked loss of consciousness in apoplectic bulbar paralysis, yet no great diagnostic significance can be assigned to its absence. The sudden obstruction of the basilar artery produces such a disturbance of the circulation even in anterior portions of the brain as may suspend consciousness. In some few instances this circulatory derangement may even give rise to choked disk, as seen by the ophthalmoscope. Often there are noticeable respiratory and cardiac symptoms, such as Cheyne-Stokes' respiration, and rapid pulse.

If death be not immediate, and we are therefore enabled to make out the symptoms due to the local disturbance, we usually observe the same phenomena as have just been described under bulbar hæmorrhage. There is sometimes paralysis of all the extremities, but usually there is trouble only upon one side. Then we have the characteristic crossed hemiplegia. The facial nerve or the nerves of the ocular muscles may be paralyzed. It has repeatedly happened that the paralysis seemed at first much greater upon one side, but after a few days changed over to the opposite one. This must be due to changes in the circulation; the thrombus grows larger, or a collateral circulation is developed. The specific bulbar symptoms are the familiar ones of all bulbar derangements—namely, lingual paralysis with resulting difficulty in articulation, pharyngeal paralysis, and rarely deafness, as a result of lesion of the acoustic center. Of course, the severity and extent of all these symptoms must vary according to the location and size of the spot of softening.

The prognosis of cases of this sort is almost always unfavorable. Death results at latest after a few days. It is often ushered in by a high temperature. Exceptionally, there is a transition into a chronic form.

We need say nothing about treatment, except that the same remedies are employed as in other acute bulbar diseases.

3. Acute, or Inflammatory, Bulbar Paralysis.

(*Acute Bulbar Myelitis.*)

"Acute bulbar paralysis," in the stricter sense of the term, means a form of disease where marked symptoms of bulbar derangement appear acutely—that is, within a few days or weeks. The anatomical lesion is probably an acute inflammation of the medulla oblongata. It is a rare disorder, and its ætiology is doubtful. There are usually mild prodromata: vertigo, headache, and, in one case of our own, painful sensations in the back of the neck. Evident bulbar symptoms very quickly follow. Usually the first of these is dysphagia. Not only is deglutition impaired, but the paresis of the soft palate and of the laryngeal muscles allows liquids to enter the nostrils or the larynx. The tongue also becomes gradually paralyzed, speech becomes indistinct, and, if the soft palate be involved, nasal.

Sometimes the extremities also become paretic, as a result of the extension of the disease to the region of the pyramids; but in many instances the extremities remain unimpaired to the end. Paralyses of the facial nerve and of the ocular muscles are somewhat more frequent. The temperature is sometimes a little elevated (100°–102°, 38°–39° C.), but not always. The pulse is almost invariably rapid ; in our patient it was 148.

The prognosis is apparently always bad. Often death takes place in four to eight days, or it may be not till the end of two or three weeks. It is invariably preceded by all the tokens of paralysis of respiration. In our case there was well-marked paralysis of the diaphragm at the end.

As yet, few autopsies have been reported. Generally the medulla presents no macroscopic changes. Exceptionally, it can be seen to be softened and mottled with minute hæmorrhages. The microscope detects abundant evidence of inflammation : granule-cells, infiltration with nuclei around the blood-vessels, thickening of the walls of some of the blood-vessels, small extravasations, swollen axis-cylinders, etc. It should also be borne in mind that precisely similar clinical phenomena seem often to be referable to peripheral changes, such as multiple neuritis affecting the bulbar nerves.

The treatment of acute bulbar paralysis is, of course, almost hopeless. In early stages we should apply counter-irritation to the nape of the neck; and we might prescribe mercurial inunction. It might also be well to employ the constant current, applied at the back of the neck, and also used to excite the movements of deglutition. We found injections of strychnine useless. Toward the end, narcotics are indispensable.

CHAPTER III.

COMPRESSION OF THE MEDULLA.

Acute compression and other injuries of the medulla are most frequently due to fracture or dislocation of the atlas and axis. As is well known, dislocation of the axis, or backward dislocation of the atlas, usually causes instant death.

Gradual compression is seen in chronic disease of the bones around the medulla, in caries and tumors of the occiput and of the first two vertebræ. Enchondroma of the base of the skull ; new growths of the sphenoid, at its junction with the occipital ; tumors of the dura; and sometimes even tumors of the cerebellum—may all excite by their pressure the gravest bulbar symptoms. We should also mention aneurism of the vertebral artery at its upper end, and of the basilar, as capable of doing similar harm. In all these cases the main cause of disturbance is undoubtedly the mechanical pressure, either directly destroying the nervous tracts or interrupting the transmission of nervous influences ; but the circumstances may be further complicated by hæmorrhages, and sometimes perhaps by inflammation of the medulla itself.

The clinical phenomena of gradual bulbar compression resemble those of spinal compression, in that they usually begin with symptoms of irritation in the distribution of those nerves the roots of which are first affected. There are neuralgia of the trigeminus, twitching of the facial muscles, tinnitus aurium, etc. If the compression becomes greater, there are more serious bulbar symptoms: disturbances of speech and deglutition, paralysis of the tongue, soft palate, face, and very likely motor and sensory symptoms in the extremities. Usually we also see general cerebral symptoms, such as vertigo, headache, vomiting, and sometimes epileptiform convulsions.

We can not, of course, draw up a definite and rigid list of symptoms, since both the individual symptoms and the general course of the disease exhibit great variations according to the way in which the compression is brought about. The diagnosis can be made in those cases only where some ætiological factor like trauma or caries of the vertebræ is known to exist. Aneurism of the vertebral artery is said by Möser sometimes to give rise to a loud systolic murmur heard between the mastoid process and the spine. In all other cases we can seldom do more than surmise the truth. Slow compression is distinguished from genuine progressive bulbar paralysis chiefly by the course it pursues—that is, there are initial symptoms of irritation—by the greater complexity of the clinical phenomena, like sensory lesions and hemiplegia, and sometimes by the asymmetry of certain symptoms. If the anterior part of the medulla is compressed, in the region of the pyramids, there may for a time be no bulbar symptoms, but merely motor symptoms in the extremities. These are chiefly paretic or spastic.

The prognosis is almost always bad, as can be inferred from the nature of the causative disease. Death is brought on either by inhalation-pneumonia, or by paralysis of respiration. Treatment must be purely symptomatic, and should follow the same rules as in progressive bulbar paralysis.

V.—The Diseases of the Brain.

SECTION I.

DISEASES OF THE CEREBRAL MENINGES.

CHAPTER I.

HÆMATOMA OF THE DURA MATER.

(*Internal Hæmorrhagic Pachymeningitis.*)

Ætiology and Pathology.—Hæmatoma of the dura mater is the name given to effusions of blood found on the inner surface of the dura mater. They are of considerable area, but of moderate thickness, and are usually encapsulated. There has been much discussion as to their mode of origin, and views still differ. One is that the hæmorrhage is the primary lesion, and that the connective-tissue membranes are developed only by the organization of the clot. This conception was originally the prevailing one, but was opposed by Virchow, who was led by the results of his own investigations to maintain that the hæmorrhage was always secondary. The primary process he believed to be a peculiar sort of inflammation —"hæmorrhagic pachymeningitis." This gave rise to a new growth of richly vascular connective tissue, into which the hæmorrhage took place. Of late, however, the tendency is again to regard the hæmorrhage as the initial change, at least in certain cases, and to refer it to an affection of the walls of the blood-vessels which diminishes their power of resistance.

The mildest forms of internal pachymeningitis present a delicate membrane upon the inner surface of the dura mater, quite easily separable, of a reddish color, and dotted with numerous red and brownish spots. These spots are due to minute hæmorrhages and collections of hæmatoidin. The membrane itself is a delicate interstitial tissue, traversed by numerous wide capillaries.

In more advanced cases the thickening is much greater. There are usually several layers, the newest and most superficial being nearest the brain. The oldest, which is in apposition with the dura mater, is composed of connective tissue that has already become rather firm and fibrous. It is evident from this lamellar structure that the whole process goes on by fits and starts. The clinical course of the disease will be seen below to agree well with such a view. The effusions are sometimes very extensive, even larger than a hen's egg, and exercise no slight pressure upon the underlying cerebral parenchyma. The hæmorrhage always takes place inside the mass, or between its layers. The effusion may, however, break through the innermost layer, so that the blood flows into the arachnoid spaces ; this is known as "intermeningeal apoplexy."

The favorite location of the hæmatoma is the parietal region. It is sometimes found at the base of the brain, in the posterior or middle fossa. Occasionally the hæmatoma is bilateral.

Hæmorrhagic pachymeningitis is not a rare disease. It is sometimes found to exist in a moderate degree in chronic cardiac, renal, or pulmonary cases, which come to autopsy. Usually there have been no special symptoms, the lesion being discovered incidentally. It has been found in like manner in connection with a great many acute infectious diseases, like typhoid fever and small-pox. It is a more important and more frequent complication in other chronic cerebral diseases, in particular such as induce marked atrophy of the brain as a whole. It is especially common in general paralysis of the insane and in other forms of dementia. Chronic alcoholism is also regarded as a potent ætiological factor. In topers it is not very unusual for the hæmatoma to be so extensive, if it occurs at all, as to cause grave cerebral derangement. Very likely changes in the vascular walls, like atheroma and fatty degeneration, contribute an important part to the result in such patients. Hæmatoma may also occur in all diseases where there is a general hæmorrhagic diathesis. Thus it is seen in pernicious anæmia, leukæmia, and scurvy. Here certainly the hæmorrhage is the primary event, as it also is in the traumatic cases, of which a number have been observed.

As might be inferred from the ætiological factors enumerated, the disease is found chiefly in advanced life, and much oftener in men than in women.

Symptoms.—Not infrequently a hæmatoma of the dura is found post mortem, which had during life been entirely unsuspected. Either the hæmorrhage was not extensive enough to cause any symptoms, or the brain exercised that remarkable tolerance which it sometimes shows even when there are wide-reaching lesions; or such symptoms as there may have been escaped particular notice, in the severity of the more general symptoms (of typhoid fever or some similar disease). But in other cases hæmorrhagic pachymeningitis excites grave symptoms, although they are seldom so characteristic as to reveal the diagnosis ; for individual cases vary greatly, according to the size of the hæmorrhages, their location, and the frequency of their recurrence.

Almost always the beginning of the disease is rather sudden. It may even be like an apoplectic seizure. The symptoms are referable partly to the general effect of the hæmorrhage upon the brain, and partly to the exact locality of the hæmorrhage. The more general symptoms comprise headache; impairment of intelligence (that is, stupor or even complete coma); slow or irregular pulse; vomiting; and contracted pupils—all being symptoms of cerebral compression. Now and then we even find choked disk. Other phenomena are added to the above when the hæmatoma occupies its usual position, upon one side and in the neighborhood of the motor cortical region, or central convolutions. The hemiplegic symptoms are not infrequent, such as hemiparesis, and, from the irritation which the effusion produces in the motor centers, twitchings and convulsions in one half of the body. Sometimes these symptoms are limited to a single extremity or to the distribution of the facial nerve. Aphasia has been repeatedly observed when the hæmorrhage was near the island of Reil. If the effusion increases, the motor disturbance becomes correspondingly aggravated, and may become bilateral. Sensation is usually little impaired.

The further course of the disease varies greatly in different cases. In the worst cases there is speedy death, usually ushered in by deep coma. In others, the first symptoms are followed by improvement, although mild indications of cerebral pressure persist, such as headache or vertigo, or else local symptoms, like hemiparesis. It is possible for the effused blood to be absorbed, and complete recovery to ensue; but usually new hæmorrhages and corresponding symptoms arise. It is precisely this appearance of the symptoms in separate attacks, this frequent recurrence of severe cerebral disturbances, that is characteristic of hæmatoma of the dura mater. As already intimated, the way in which the anatomical lesions develop explains this perfectly. Thus the disease may drag on for months and years, sometimes improving and sometimes aggravated. Then some attack at last proves fatal. Arrest and actual improvement are still possible even in the later stages, although often the features of the case have meanwhile undergone essential alteration because of the progress of some causative disease. In general, the clinical phenomena of hæmatoma of the dura are often complicated and obscured by the co-existence of the primary disease.

The **diagnosis** is therefore difficult. The main points may be recapitulated as follows: First, the existence of ætiological factors, like alcoholism, or chronic cerebral disease; second, the sudden onset, and also the abrupt appearance of further symptoms, the alternation of rapid aggravation and improvement; and third, the existence of symptoms which experience has taught us to refer mainly to the cortex of the brain, namely, unilateral convulsions, monoplegic paresis and contractures, and contracted pupils. Nevertheless, frequent errors in diagnosis can not be avoided.

Treatment.—The possibility of therapeutic interference being successful is very small. In apoplectic shocks, ice to the head is useful; and if the patient be robust, it may also be advisable to use local depletion, by leeches on the temples or behind the ears. It is also customary to prescribe some such thing as senna or calomel for "intestinal depletion."

If the first onset is successfully withstood, the main things as to further treatment are general hygienic and dietetic directions, so as to guard as far as possible against fresh hæmorrhages. Alcohol and excessive bodily or mental exertion should be forbidden. Of course, paralysis or other persistent disturbances may call for special treatment.

CHAPTER II.

PURULENT MENINGITIS.

(*Purulent Cerebral Leptomeningitis. Meningitis of the Convexity.*)

Ætiology.—Purulent inflammation of the dura mater has no clinical importance, for it is very rare, and occurs only as the result of the extension of disease from neighboring parts. We shall accordingly consider below purulent inflammation of the pia mater only. One important variety of this disease has already been discussed (see page 103 *et seq.*) as one of the infectious diseases, under the name of epidemic cerebro-spinal meningitis. There we saw also that the occasional sporadic cases of primary or "idiopathic" meningitis are probably identical ætiologically with those of epidemic meningitis. In all other instances, purulent meningitis is a secondary disease—that is, the specific agent, which excites the purulent inflammation, originates in some other organ primarily, and affects the meninges only secondarily. We should, therefore, seek most carefully in every case of purulent meningitis, at the bedside and more particularly at the autopsy, to discover the way by which the pathogenetic virus reached the meninges. We have no right to say that the case is one of primary meningitis, strictly so called, until we have made a most careful examination with a negative result. From a clinical standpoint, it is true that many cases of secondary meningitis do seem as if they were primary, because not infrequently the really primary disease excites insignificant symptoms, or perhaps no symptoms at all.

Secondary purulent meningitis is most often due to disease of the cranial bones, and in particular to disease of the petrous portion of the temporal bone, with the auditory apparatus therein contained. If we consider the anatomical relations of the middle and internal parts of the ear, we can easily understand why inflammation in them is not infrequently followed by meningitis. Usually it is a caries of the petrous portion, itself due to an otitis media, which leads to an irruption into the cranial cavity. This is especially apt to take place through the thin vault of the tympanic cavity. It may also extend from the mastoid cells, or by direct propagation along the sheaths of the acoustic or facial nerves, or along the vessels which lie in the petroso-squamous suture. The dura is first attacked, and then the pia. In many instances, the neighboring venous sinuses (transverse, cavernous, and superior petrosal) transmit the inflammation, being first attacked by a suppurative thrombo-phlebitis. Again, exceptionally, a purulent inflammation in the upper part of the nasal cavity may lead to meningitis.

Another and frequent source of meningitis is found in the various traumatic injuries of the cranium. In the great majority of these cases there is an open wound, admitting infectious agents which are suspended in the air. The suppuration often commences in the spongy texture of the diploë, thence extending to the dura and pia, either directly or by way of a purulent thrombosis of some sinus into which the veins of the diploë enter. It is, indeed, generally affirmed that we may have a traumatic purulent meningitis without any open wound ; but this is not easily explained, according to our present views as to the origin of purulent inflammations. It is equally difficult to understand, what many authors affirm, that the heat of the sun's rays, striking upon an uncovered head, may excite purulent meningitis. In most cases of sunstroke we find marked hyperæmia of the meninges, but no inflammation.

Meningitis may have an intra-cranial origin ; it is sometimes the sequel of cerebral abscess. No matter what starts the abscess, if it extends to the surface of the brain, it causes a more or less extensive purulent meningitis at that point. An abscess may burst into one of the lateral ventricles, and the infection be carried from that point to the pia at the base of the brain.

All the cases thus far contemplated allow of the explanation that the inflammation reaches the meninges by direct extension ; but there is another group of cases where the agent that infects the pia mater originates at some distant part of the body, and is probably conveyed by the blood or lymph currents. These cases are often termed metastatic meningitis.

Of this sort is the secondary meningitis seen in connection with genuine lobar pneumonia, a combination already discussed (see page 198). The meningitis is also sometimes a complication of empyema, rarely of pyæmia and septicæmia, ulcerative endocarditis, and very rarely of typhoid and the acute exanthemata (small-pox, scarlet fever), and of acute articular rheumatism. In each case we must, of course, determine whether the meningitis may not have a connecting link between itself and the primary disease, such as otitis in scarlatina, or secondary empyema in typhoid fever.

Pathology.—For the pathological anatomy of purulent meningitis, we may refer mainly to the statements made on page 94, under epidemic meningitis, for the lesions are similar. The only way to determine whether a meningitis is secondary or primary is by finding or failing to find disease in neighboring or remote parts, for example pneumonia. The seat of the meningitis will vary according to that of the primary inflammation, if there be any. If the meningitis is due to caries of the petrous bone or to an injury of the skull, the purulent exudation is usually most abundant in the immediate neighborhood of the primary lesion, between the pia and arachnoid. Thence it gradually extends along the surface of the brain, sometimes chiefly on the convexity and sometimes at the base. But in general it may be said that both the secondary and the metastatic varieties of meningitis as a rule affect the convexity, although this is by no means invariably the case. This rule explains why these cases are sometimes termed meningitis of the convexity, in contrast to tubercular meningitis, which latter, as we shall find, has a preference for the base of the brain, and hence is called basilar meningitis. The spinal pia mater is sometimes simultaneously attacked, but not so constantly as in primary, or epidemic meningitis. The brain is almost always involved—the inflammation extends along the vessels which dip from the pia mater into the cerebral parenchyma. It is not a rare thing to find minute abscesses or ecchymoses in the interior of the brain. The whole parenchyma is usually moist, œdematous, and of a doughy consistence. The meningeal exudation exerts upon the brain a pressure which gives rise to important symptoms; by it the superficial cerebral convolutions are often considerably flattened. The lateral ventricles almost always contain more or less sero-pus.

Clinical History.—So varied are the primary diseases which may entail a meningitis, that it is hardly possible to make a sketch of the disease which would suit all cases. If the meningitis comes on during the course of pyæmia, pneumonia, or some other severe illness, its proper symptoms are often inextricably confused with those of the primary trouble; and when the skull or the brain has been mechanically injured, it is very hard to determine whether a meningitis has been excited, because the trauma may of itself produce such serious effects. The following description, therefore, applies chiefly to cases of apparently primary meningitis, or to cases where the meningitis, although secondary, is well marked.

The beginning in such cases may be sudden, or it may be somewhat insidious. Sometimes the grave symptoms appear almost at once, accompanied by a chill and high fever. Sometimes there are for a while indefinite and more or less ambiguous prodromata, but almost always it is the headache which first attracts attention. This grows worse with more or less rapidity, and almost always becomes very violent. Exceptionally it may be insignificant. Not infrequently it varies considerably, being much worse at some hours or on some days than on others. The location of the pain is sometimes frontal, sometimes occipital, and sometimes over the whole head. Next in prominence to the headache, particularly in the later stages of the disease, is the mental disturbance. The patient complains of vertigo, becomes dull and stupid, or begins to wander. The delirium may be extremely violent, but usually there is depression rather than exaltation, and the stupor merges into coma. That the headache still continues may be inferred from the frequent raising of the hand to the head and the grimace of pain whenever the head is moved, till finally the coma becomes so profound that even these reflex actions cease.

Usually these general cerebral symptoms are attended by others referable to the particular locality affected. The neck is rigid. This is most marked when the posterior fossa and the medulla are affected. Then there are all sorts of paralytic or irritative symptoms in the distribution of the cranial nerves, mainly due to lesions of the nerves where they emerge from the base of the brain ; there is derangement of the motores oculi, as shown by paralysis or nystagmus ; the pupils are unequal, or are contracted or dilated, and do not react to light ; there is paresis of the facial, or trismus, or grinding of the teeth. All these symptoms may appear equally plainly in other forms of meningitis. Sometimes we can detect optic neuritis with the ophthalmoscope. Other symptoms are due to cerebral disturbance, often apparently located chiefly in the cortex. Thus there may be twitchings of individual muscles, or even pronounced convulsions in one or more limbs, or paralysis of one extremity or of half the body. Sometimes the autopsy explains these phenomena, but often we fail to find any marked anatomical lesion to correspond to them, and are obliged to ascribe them to circulatory or functional derangement.

Of the remaining symptoms, the fever is most important. Almost always the temperature is decidedly elevated, not infrequently reaching 104° or 105° (40°–40·5° C.). The fever is, however, very irregular. There may be repeated chills with great elevations of temperature. The pulse is generally rapid, and often somewhat irregular. Exceptionally it is less frequent than normal, because of cerebral compression. Vomiting is not a rare symptom, particularly at first. There is almost invariably constipation, and the abdomen is often tense and concave. The urine is scanty, and often contains a trace of albumen. Secondary diseases are sometimes found post mortem, such as lobular pneumonia, due to inhalation of food during the comatose state.

The entire course of the disease occupies only a few days in very acute cases, and scarcely ever exceeds a week or ten days. The termination is almost sure to be fatal. In the few cases of recovery which have been reported the diagnosis is doubtful. In most instances deep coma precedes death, though sometimes it is ushered in by convulsions. There is often a great rise of temperature (107·5°, 42° C., or higher) before the close of life.

Diagnosis.—The diagnosis of purulent meningitis is sometimes pretty evident; but it may be very obscure, so that we can not always avoid confounding it with other severe acute diseases, such as typhoid, pyæmia, and general tuberculosis. In general the most characteristic symptoms of any variety of meningitis are intense headache, rapid onset of grave cerebral disturbances, delirium and insensibility, stiffness of the neck, and disturbances in the distribution of the cranial nerves (especially impaired motion of the eyeball and optic neuritis). These last, although often slight, are generally present ; and in connection with these separate symptoms we must also always consider the whole course of the disease and any ætiological factors which may exist. Typhoid fever is excluded by its usually slower onset, the greater delay in the appearance of grave cerebral symptoms, the rose-spots, the greater size of the spleen, the characteristic stools, and the peculiar fever-curve. Severe septic and pyæmic diseases, including ulcerative endocarditis, likewise excite cerebral disturbances which might be misleading, but these diseases are to be recognized by their ætiology (external wounds, abortion, etc.), cutaneous ecchymoses, septic retinitis, swelling of the joints, and repeated rigors. Uræmia may also simulate meningitis. Sometimes the character of the urine, and the predominance of convulsions, will set us right, but not always. We may state in conclusion that every one who sees many cases (including ourselves), must repeatedly have met with patients presenting the symptoms of a severe and acute cerebral affection apparently primary, without demonstrable cause, and seeming to justify a diagnosis of meningitis, but yet yielding post mortem no signs of disease beyond "hyperæmia," "œdematous swelling," and similar changes of only secondary importance. We are as yet wholly unable to explain such cases.

Granting that meningitis exists, what variety is present? The ætiology is a great help in answering this question. We should endeavor to learn whether there has been traumatism or some old ear trouble. It is well to employ the aural speculum. We can not say that a patient has epidemic meningitis unless several cases occur simultaneously, although herpes is very characteristic, as it appears only exceptionally in other varieties. Usually tubercular meningitis also can be diagnosticated only by means of the ætiology. Its symptoms, of course, are in almost all particulars identical with those of purulent meningitis. Sometimes, however, tubercles can be detected in the choroid by means of the ophthalmoscope. For further particulars see the next chapter.

Treatment.—The treatment of the different forms of meningitis varies but little. Locally, the favorite remedies are ice applied to the head, which should be shaved if practicable, and local depletion by means of leeches behind the ears or on the temples. Many physicians recommend cutting off the hair and rubbing in antimonial ointment, or applying ethereal tincture of iodine. We have never tried this. Cool baths with douching can not be employed unless the patient can be moved without too much pain. For violent pain or great restlessness we must use narcotics. The best is morphine subcutaneously. We can not hope for much benefit from other internal remedies, such as iodide of potassium or calomel.

Prophylaxis demands, above all, prompt recourse to the otologist for aural trouble of any kind, and strictly antiseptic treatment of all injuries of the skull.

CHAPTER III.

TUBERCULAR MENINGITIS.

(*Basilar Meningitis.*)

Ætiology.—Tuberculosis of the leptomeninges is always a secondary affection—a sequel to previously existing tubercular disease of some other organ. Why the pia mater should be so often singled out for secondary infection with the tubercular virus, or what path that virus traverses to reach the pia—about these questions we know very little. We can merely state what the other tubercular diseases are, which, as experience shows, entail tubercular meningitis most frequently. These primary affections may be of themselves productive of grave clinical phenomena, the meningitis merely adding to the complexity of the picture. Again, the primary trouble may not have betrayed itself at all, or its symptoms may have been long ago arrested, so that the meningitis seems to be a primary disease. In some cases even the most careful examination will fail to detect the origin of the trouble.

Tubercular meningitis is oftenest a sequel to pulmonary tuberculosis. It may appear as a terminal complication in cases of advanced phthisis, or it may come on while the signs of pulmonary disease are as yet very slight. Next in order as a causative affection comes tubercular pleurisy. This origin is not infrequent. As we have already seen, most cases of apparently primary pleurisy are due to tubercle. This statement is supported by the fact that it is not very exceptional for the symptoms of tubercular meningitis to supervene suddenly upon what had seemed to be genuine convalescence from pleurisy. In children, and sometimes in adults, the virus may be carried to the meninges from cheesy, tubercular, bronchial or mesenteric glands, or from tubercular or "fungous" disease of the bones or joints. Another danger to adults is tubercular disease of the genito-urinary apparatus. It should also be noticed that a single large tubercle in the brain may lead to miliary tuberculosis of the meninges. In short, we see that it is not impossible for any tubercular infiltration, wherever situated, to communicate infection either to the meninges alone (in some remarkable way), or simultaneously to them and many other organs. In this last case, where in all probability the blood carries the virus through the system, the meningitis is merely a part of a general miliary tuberculosis (see page 236). When the meninges are alone or predominantly affected, there must be some peculiar manner of infection, about which, however, as we have already confessed, we have no information.

We sometimes hear the attack ascribed to such causes as over-exertion, mental excitement, or traumatism; but we need hardly say that these can not be properly regarded as ætiological factors, and that usually they are merely coincidences. Age, however, does have an influence; children are much oftener attacked than adults, although the latter also are liable to it.

Pathology.—As in tuberculosis of serous membranes, so in tuberculosis of the pia, there are two effects of infection to be distinguished from each other: (1) the development of the specific new growth—that is, of miliary tubercles; and (2) the inflammation. The relative degree of these two varies. Sometimes the tubercles are very abundant and the inflammatory exudation comparatively scanty; and in other cases the inflammation is considerable, although relatively few tubercles are discoverable. The tubercles are usually found in greatest number along the course of the larger blood-vessels, and therefore chiefly in the furrows and clefts of the surface of the brain, in the fissure of Sylvius, at the chiasma, the pons, the medulla, and the cerebellum. And, in general, the base of the brain is usually more affected than the convexity—hence, as we have said, the name of "basilar meningitis." There are, however, exceptions to this rule. We very often find that the region supplied by one or more arteries suffers above other parts; this must be due to the manner of infection. The inflammatory lesions consist of hyperæmia, usually well marked, and a sero-gelatinous exudation of variable amount. That this exudation is partly cellular can always be proved by the microscope, and often even macroscopically from the great cloudiness of the pia; but still we seldom find enough to justify us in calling the process one of genuine purulent inflammation. Small hæmorrhages into the pia are quite often found. The brain itself is usually flattened from the pressure of the meningeal exudation. Often the inflammation involves the brain-substance itself, as shown microscopically by tubercles, inflammatory changes, and capillary hæmorrhages. The ventricles usually contain, although not invariably, a hydrocephalic effusion. This led earlier observers to term the disease "acute hydrocephalus." The effusion is serous, but generally turbid from cellular constituents, and not infrequently tinged with blood. The choroid plexus is engorged, and may present tubercles. The spinal cord, in the majority of cases, shares in the tubercular disease. Here, too, we find inflammation of the pia and miliary tubercles. This fact has a clinical bearing, being explanatory of many of the symptoms.

Clinical History.—Tubercular meningitis almost always begins with a prodromal stage, which is often brief, but may last one or two weeks, or even longer. The patient may be apparently well (*vide supra*) until this comes on, or he may have already shown signs of some other tubercular affection. He now feels badly, at any rate, and begins to complain of headache, worse at some times than at others. There is anorexia and very often constipation. Another frequent prodrome is an attack of vomiting, which may or may not recur. Sleep is disturbed, either by the headache or by a certain general restlessness. We have occasionally met with cases where the illness began with pronounced mental disturbance. The patient became irrational and said and did queer things, and then a few days later there appeared distinctive meningeal symptoms. In two patients, who were topers, the disease began just like delirium tremens.

After an initial period of variable duration, the general health becomes more and more impaired. The headache increases. The patient takes to his bed, begins to be delirious, and soon presents well-marked symptoms of grave brain trouble. Intelligence becomes more and more impaired. The patient is sleepy, and can be roused imperfectly by the voice, if at all. At the same time he is usually quite restless at first, grasping at invisible objects in the air, picking the bed-clothes, and continually moving his legs. The delirium may be low or noisy; the patient may keep up a constant singing or screaming or whistling. The persistence of the headache even in this stage is shown by the facial contortions and complaints of the sufferer, whenever there is a temporary approach to consciousness. There is also, as a rule, decided tenderness in the nape of the neck on pressure, frequently accompanied by great stiffness of the neck. Sometimes there is stiffness of the entire spinal column, and pain in the same. This is certainly due to the coincident spinal meningitis.

Another group of symptoms in the distribution of the cranial nerves are identical with those seen in the other forms of meningitis. Ptosis is not infrequent, on one or both sides, due to paresis of the motor oculi. There is strabismus, either internal or external. Symptoms of irritation of the nerves governing the movements of the eye are very frequent, especially in the early stages of the disease. Thus we see slow involuntary lateral movements of the eyeballs, and sometimes nystagmus. The pupils are often unequal; they may be enlarged or contracted, and often they undergo marked and repeated variations in size. The reaction of the pupils to light is usually sluggish, and may be absent. With the ophthalmoscope we find not infrequently neuritis, or choked disk. In some instances, but not in all, we find also tubercles in the choroid, which, of course, greatly assists diagnosis. Sometimes there is occasional twitching in the distribution of the facial nerve, or a slight tonic contraction, or again paresis on one side. The natural explanation of all these phenomena is that the nerve-trunks are interfered with at the base of the brain, whether by the pressure of the exudation or by participation in the inflammatory process, or by the minute hæmorrhages which sometimes take place into the sheath of the nerves.

Disturbances in the extremities may be caused by various lesions. Motor symptoms of irritation are apparently referable for the most part to changes in the cortex of the brain. We see occasional twitching of larger or smaller groups of muscles, or rarely convulsions. These latter may be unilateral, or in a single extremity. Sometimes there is well-marked paresis of one half the body or paralysis of one limb, or there may be aphasia, although it is only in a part of the cases that we find post mortem any lesion which explains these symptoms. In most cases there is a particularly large collection of tubercles in certain places upon the cortex cerebri, occasioning a local compression or an inflammatory œdema, which in its turn excites the phenomena mentioned. Sometimes the brain-substance itself is found in a state of red softening underneath these spots. Another and not very rare symptom is a peculiar stiffness of the limbs, due either to direct irritation or to reflex action. The reflexes in the lower extremities are generally exaggerated at first, but later on become diminished, and finally abolished. The reflexes upon one side may be more vigorous than upon the other. As to sensation, it is hard to reach definite conclusions, because of the patient's stupor. Sometimes there is well-marked cutaneous hyperæsthesia, probably referable to an implication of the spinal cord in the process.

The behavior of the pulse and temperature is interesting. The temperature is usually elevated, but often only to a slight extent—that is, varying between 100·5° and 102° (38° and 39° C.). Often the temperature falls quite low, only to rise again, the alternations being at irregular intervals. Exceptionally the temperature may remain high (104° F., 40° C.) most of the time. Toward the end there is usually a decided change in temperature, either upward or downward. In many instances there is a very low temperature before death; in two cases we have seen a temperature of 87·8° (31° C.). Or the temperature may rise to 106° (41° C.) or higher just before death. The pulse is often abnormally slow in the early stages of the disease, even numbering only 40 to 50 beats per minute. This is referable without doubt to the increased intra-cranial pressure. Later on the pulse becomes small and rapid. The transition may be very sudden. The vagus is at first irritated, and then paralyzed. The pulse is often irregular.

Respiration is generally moderately accelerated. If the breathing is very deep and rapid, we should always think of simultaneous miliary tuberculosis of the lungs. Toward the close of the disease the respiration often assumes the Cheyne-Stokes type: there is a long pause, followed by very superficial and gentle respiration, which gradually grows deeper and deeper, then diminishes again, and is succeeded by another complete pause. This symptom is always most ominous, for it indicates that the excitability of the respiratory center is already greatly impaired.

Symptoms referable to still other organs are few. Vomiting is rare in the later stages of the disease. The abdomen often presents a "boat-shaped" concavity, as the result of tonic muscular contraction, and is hard and tense. There is almost always constipation. The spleen may be somewhat enlarged. The urine sometimes contains a trace of albumen. On account of the drowsiness, it is usually voided in the bed or retained in the bladder. Almost invariably there is rapid marasmus.

The entire duration of tubercular meningitis varies somewhat, chiefly because of the varied length of the first stage. When the disease is once fully developed, the illness seldom lasts more than three to ten days longer. Frequently the illness is divided into three stages: 1. The stage of cerebral irritation, with headache, stiff neck, vomiting, and delirium; 2. The stage of cerebral compression, chiefly due to the hydrocephalus, and causing drowsiness, slowness of the pulse, paralysis of the motores oculi, hemiplegia, etc.; and 3. The paralytic stage, presenting deep coma, relaxation of the previously contracted muscles, accelerated pulse, and marked variations of temperature. Such a division is too diagrammatic to correspond accurately to the real phenomena, but will, nevertheless, often aid us in getting a general idea of the course of the disease.

The termination of tubercular meningitis seems to be inevitably fatal. Sooner or later the patient loses consciousness completely, his pulse grows very small and rapid, his respirations irregular and intermittent (Cheyne-Stokes), his temperature, as we have said, either rises high or falls far below normal, and, finally, death is ushered in by a paralysis of all the vital functions. A few physicians have reported cases of recovery; but was the diagnosis correct? While we would by no means absolutely deny that recovery from tubercular meningitis might occur, it would certainly be very difficult to prove, in any particular instance, that such a thing had happened.

Tubercular Meningitis in Children.—The disease is so prone to attack children that it seems desirable to subjoin a few remarks about the peculiarities of the affection as observed in them.

Often the little patient is pale and weakly, with tubercular antecedents; but sometimes apparently healthy and vigorous children are attacked. Tubercular meningitis may be the sequel of measles, whooping-cough, or some other disease, which has occasioned the development of the tubercular process. Usually the severer symptoms are preceded by a rather long prodromal stage, during which the child is fretful, eats little, and grows thin and pale. In children as well as adults, the second stage is generally ushered in by headache and vomiting. The headache is not very often violent; but children complain with remarkable frequency of pain in the abdomen and chest. The cause of this symptom is unknown. The pulse is almost invariably slow, often somewhat irregular, and it frequently undergoes surprisingly rapid changes in rate—for example, varying twenty or more beats inside of a few hours. Very early the child becomes dull and drowsy. Frequently it emits a peculiar deep sigh, or that sudden loud scream or "cephalic cry" which physicians long ago learned to recognize and fear. The symptoms referable to the cranial nerves and the nervous disturbances in the limbs are similar to those seen in adults. Strabismus is almost constant. Very often there is trismus, and a distinctly audible grinding of the teeth, most distressing to the by-standers. Trousseau laid weight upon the appearance of red spots (*taches cérébrales*) upon the skin after it has been mechanically irritated; but these have no diagnostic value. They are due to increased reflex action, and are seen in all sorts of acute diseases. The fever is generally, as in adults, not very high, 100° to 102° (38°–39° C.). Respiration is usually rapid, and often is irregular.

The change from bad to worse is almost always announced by a rapid increase in the pulse-rate, to 160 or 200. The child becomes completely comatose. Very often there are repeated epileptiform convulsions, either universal or affecting single extremities. Death is usually preceded by a decided rise in temperature.

Diagnosis.—When the symptoms are pronounced, the diagnosis of meningitis is easy, and we have merely to determine just what variety is before us. That the disease is due to tuberculosis is never to be ascertained by means of the meningeal symptoms themselves, but is rendered possible only by the ætiology, if that be discoverable. Here, as in all tubercular diseases, we have chiefly to consider (1) heredity and (2) the evidence of previous or existing tubercular affections in other parts of the body. Under this second head are to be considered scrofula, diseases of the bones and joints, pulmonary tuberculosis, pleurisy, and tuberculosis of the genitals or of the choroid. If our search be unsuccessful, we may sometimes get a hint of the truth from the general appearance of the patient: for example, he may be pale, or narrow-chested. And, again, the absence of trauma, aural disease, or epidemic meningitis will make tuberculosis more probable.

In its early stages, or when it varies from the usual course, tubercular meningitis may be very difficult to diagnosticate. This is particularly true when the patient is a child. The early malaise and vomiting are treated as "ordinary gastric catarrh" until the grave cerebral symptoms disclose the mistake in diagnosis. In such cases we should be careful not to disregard the initial slowness and irregularity of the pulse. This alone should make our prognosis guarded. The fever may be prominent at the commencement, and tempt us to call the case one of incipient typhoid fever; and, indeed, the correct diagnosis is often impossible until the disease develops further. In regard to this, see the preceding chapter on purulent meningitis, where the exclusion of severe septic diseases and of uræmia is also considered.

Before the autopsy we must remain in great uncertainty as to the number and distribution of the tubercles, the existence of a large effusion into the lateral ventricles, etc. We are often amazed at the post-mortem examination by the apparent insignificance of the lesions. Paralysis of the cranial nerves (eyes, face) implies that the base of the brain is gravely affected. If such symptoms are absent, and there are mental disturbances, and motor symptoms of irritation displayed in the extremities, we are led to infer meningitis of the convexity. If the nervous disorder be mainly unilateral, we may assume that one hemisphere is more affected than the other.

Treatment.—However hopeless the prospect, we are nevertheless bound to employ all the remedies at our command, as in other forms of meningitis. Above all, we should be thorough in applying ice to the head, and may also try local depletion and lukewarm baths, with douching. The inunction of mercurial ointment has also been recommended. The most common internal remedies are calomel—half a grain to a grain (grm. 0·03–0·05) for a child every two hours—and infusion of senna. Iodide of potassium may also be freely given, fifteen grains daily to a child, and two or three times as much to an adult. Whether it does any good is extremely doubtful. If the patient is very restless, narcotics are indispensable. Stimulants are often given in the last stage of the disease, but generally without effect.

About prophylaxis, we need merely refer to the general statements on page 231, in regard to prophylaxis from the various tubercular diseases.

CHAPTER IV.

THROMBOSIS OF THE CEREBRAL SINUSES.

Ætiology and Pathology.—The sinuses of the dura mater sometimes present a thrombosis, under circumstances similar to those which induce the same process in other veins. The most frequent occasion for such thrombosis is marasmus, however brought about, with the accompanying feebleness of circulation. This is the explanation of those not very rare cases found among wretched and ill-nourished children in the first year of life, and also among adults in a similar physical condition, as in phthisis. In many of these instances passive congestion also seems to contribute to the formation of the thrombus.

Half way between marantic thrombosis and the inflammatory variety immediately to be described, come those cases which are seen in typhoid fever and other severe acute infectious diseases. Here the specific virus apparently plays an important part (just as in thrombosis of the femoral vein), although very likely the cardiac weakness also promotes the thrombosis.

Genuine inflammatory thrombosis—that is, thrombosis in connection with real phlebitis of the sinus—is almost always due to the extension of inflammation from some neighboring part. The most fruitful cause is suppuration in the petrous bone, the result of otitis or caries. This spreads to the walls of the transverse or petrosal sinuses, which are close by. Also wounds, necrosis, or other affections of other cranial bones may excite thrombosis ; likewise, although seldom, deep-seated inflammation of the soft parts of the head and face, like large furuncles or erysipelatous abscesses, may produce the same result.

Thrombosis due to marasmus is most frequently found in the superior longitudinal sinus, while the inflammatory variety usually occupies either the transverse, petrosal, or cavernous sinuses. Of course the thrombus may grow out from its original sinus into neighboring ones. Important clinical symptoms are caused by secondary venous stasis in the veins which empty into the occluded sinus. These symptoms are most pronounced when the longitudinal sinus is affected ; objectively, we find the meningeal veins which lie on the surface of the brain distended and tortuous; and often there are extensive meningeal ecchymoses. Even the cerebral parenchyma beneath shows distinct evidence of passive congestion, and minute capillary hæmorrhages have been repeatedly found in it.

Symptoms.—In some instances moderate thrombosis of the cerebral sinuses has been found post mortem, although there had been no symptom suggesting it before death. In other cases the thrombosis does excite undeniable cerebral disorder, but the symptoms are so general and ambiguous that the most we can do is to suspect the existence of the clot without being at all certain about it.

Sinus thrombosis in marantic children usually causes coma, stiffness of the neck and back, strabismus, nystagmus, and sometimes clonic spasms in the face and limbs. The symptoms in adults are similar, comprising headache, drowsiness, occasionally delirium, sometimes coma, and varying symptoms of irritation or of paralysis in the distribution of the cranial nerves (nystagmus, strabismus, trismus) and in the extremities. But even all these symptoms are insufficient to make the diagnosis certain. They must be re-enforced by certain other phenomena more distinctly referable to the peculiar circulatory disturbances occasioned by the thrombosis. Occlusion of the cavernous sinus sometimes excites well-marked symptoms of stasis in the ophthalmic veins. Thus the retina may be seen through the ophthalmoscope to be passively congested, there is œdema of eyelids and the conjunctiva, the eyeball is unusually prominent, and the frontal vein is distended. In case of an inflammatory thrombosis, the periphlebitic swelling may cause symptoms in the distribution of the neighboring nerves, especially paresis of the oculo-motor or abducens, or trigeminal neuralgia. In thrombosis of the transverse sinus an œdematous swelling is occasionally seen behind the ear, near the mastoid process. If the clot projects into the petrosal sinus or even actually into the internal jugular, the lower part of this vein collapses. And inasmuch as the external jugular can empty itself with unusual ease into the unfilled internal jugular vein, the external jugular is also affected and becomes less prominent upon the affected than upon the normal side. Sometimes it is even possible to feel the thrombus in the internal jugular. Such thrombosis causes pain and swelling in the neck on the abnormal side. When the superior longitudinal sinus is blocked up, there are symptoms of nasal engorgement (epistaxis), and distention of the veins about the temples, which veins are connected with the longitudinal sinus by emissary veins. We must confess, however, that all these special symptoms are comparatively rare, and often difficult of demonstration even when present.

The symptoms become more complex where there is a suppurative phlebitis, because there are usually pyæmic symptoms as the disease progresses. Thus, there may be rigors and high fever, pulmonary abscess, suppurative arthritis, etc. We have already mentioned the combination of thrombosis of a sinus with purulent meningitis.

The prognosis is almost always bad, both because of the nature of the causative disease and because of the grave cerebral derangement or the secondary pyæmia. Treatment can be only symptomatic.

SECTION II.

DISEASES OF THE BRAIN-SUBSTANCE.

CHAPTER I.

DISTURBANCES OF CIRCULATION IN THE BRAIN.

(*Cerebral Hyperæmia. Cerebral Anæmia.*)

It is presumable that so sensitive an organ as the brain is much influenced even by slight disturbances of circulation ; but as yet we have comparatively little knowledge of the production and character of such disturbances, their very existence being very difficult to demonstrate. There are many instances where marked cerebral symptoms justify the assumption that the brain is in some abnormal condition, and yet where there are many arguments against any marked anatomical lesion. Here we surmise that there is some circulatory derangement, although we have no actual and direct arguments to rely upon. For example, we refer to this cause certain cases of headache, sensations of pressure in the head, vertigo, general hyperæsthesia, and of that protean and nevertheless easily distinguishable disease known as cerebral neurasthenia (*q. v.*). At present, however, we can not determine how far circulatory disturbances are actually concerned in these cases, or of what kind they are, or whether there may not be purely functional disease of the brain entirely independent of changes in the blood-vessels.

Certain groups of cerebral symptoms, which come on in paroxysms, seem the most clearly referable of all to circulatory disturbance. There can hardly be a doubt that the phenomenon known as fainting or syncope is due to sudden cerebral anæmia. As is well known, fainting is usually the result of a clearly demonstrable cause. Frequent and familiar causes are emotional excitement, terror, unusual psychical impressions (like the sight of blood), the influence of great heat, or great physical strain, as by long standing. The condition of the stomach has certainly a great influence in many cases. There are many persons who, if they go long beyond the usual time without eating (particularly without breakfast), are very liable to syncope. Some individuals are especially subject to fainting fits. Such persons are often slight and anæmic (for example, convalescents), but some are in appearance robust and vigorous. Many children are subject to fainting.

Just what causes the cerebral anæmia in all these cases is doubtful. Mental excitement is usually supposed to lead, in fainting, to a contraction of the minute cerebral arteries. It is not, however, impossible that in these cases also, as in others, sudden cardiac weakness is one factor, although, if so, it is strange that we never see a trace of cyanosis. Where the attack is apparently connected with unusual conditions of the abdominal organs we are reminded of the relations of the splanchnic nerve to the heart (Goltz's experiment of beating the belly of a frog), and of the possibility that the brain might be left anæmic if the abdominal vessels suddenly dilated and absorbed a large proportion of the whole blood-supply.

The symptoms of an ordinary fainting fit are known to every one. There are usually certain prodromata. The person begins to "feel badly." Dizziness comes on, the senses are confused, the ears ring, there are spots before the eyes or total darkness, the floor seems to move, and surrounding objects begin to spin around. All this is almost always accompanied by nausea, and sometimes there is actual vomiting. If the person can lie down promptly the attack is sometimes averted without complete loss of consciousness. Otherwise there is unconsciousness for a time varying from some minutes to even a half-hour or longer. What the bystander notices most, even at the first, is the pallor which overspreads the face and often becomes extreme, and which is the visible expression of the coincident cerebral anæmia. Very often the face and body are bathed in cold perspiration. The pulse is usually small and rapid.

There is no real danger in an ordinary attack. The most important therapeutic measure is to lay the patient horizontally as soon as possible, to favor the return of blood to the brain. Mild stimulants should also be employed; the face should be sprinkled with cold water, the temples rubbed with vinegar or cologne-water; brandy or wine should be administered. We can overcome a tendency to fainting fits only by strengthening the constitution.

The results of chronic cerebral anæmia are observable when the cerebral condition is part of excessive general anæmia. Almost all cases of chlorosis, pernicious anæmia, and acute anæmia from loss of blood (as in ulcer of the stomach) display most plainly the symptoms of cerebral anæmia. The phenomena are essentially the same as in syncope, only less in degree. Consciousness is maintained, except in the worst cases. A sort of persistent drowsiness, however, often attended by frequent gaping, is one of the most constant symptoms. The patient is most distressed, as a rule, by loud tinnitus aurium, persistent nausea, and sometimes by obstinate headache. All these symptoms are aggravated if the patient sits up in bed, and are least marked when he lies as quietly as possible in a horizontal position. The treatment of this condition is of course identical with that of the causative disease and the general anæmia.

Cerebral hyperæmia, like cerebral anæmia, may be either chronic or paroxysmal. Of the chronic variety we know almost nothing. It seems doubtful, to say the least, whether there is really a "general plethora," or whether the headaches and vertigo that "full-blooded" persons complain of are due to hyperæmia of the brain. Nor have we any direct proof that the cerebral symptoms resulting from chronic poisoning (alcohol, tobacco), or from persistent mental over-exertion, are brought about by hyperæmia, as some assume, and not rather by functional disorder of the nervous elements themselves.

We have the best reason to claim cerebral hyperæmia as the cause of cerebral symptoms in instances of "cerebral congestion" or "rush of blood to the head." There is a more or less sudden appearance of general excitement, with a sensation of warmth in the head and neck, strong pulsation of the carotids, a red face, general hyperæsthesia and irritability, headache, vertigo, tinnitus, spots before the eyes, and nausea. An attack lasts half an hour to an hour. Apparently there is vaso-motor disturbance, causing a sudden enlargement of the cerebral blood-vessels, and due either to a paralysis of the vaso-constrictors or to a stimulation of the dilators. In severe cases there may be maniacal excitement, or there may be stupor and drowsiness and other symptoms of lowered intellectual activity, resembling a slight apoplectic attack (see a later chapter). In such a case we can not determine whether there is hyperæmia alone, or whether there is not some further lesion, like a small hæmorrhage.

In treating congestion we should keep the patient as quiet as possible, with head and shoulders raised; and, secondly, we should endeavor to draw the blood away from the brain. This purpose will be served by hot foot-baths, sinapisms applied to the chest and the calves, and purgatives, like senna or colocynth. It is also beneficial to apply cold to the head. In a severe case it is proper to put leeches to the temples or the mastoid processes.

To prevent, as far as possible, the recurrence of the attacks, we must have regard to the general constitution of the patient. We may mention, as of chief importance, diet (no alcohol) and a course at some watering-place, or "cold-water treatment."

CHAPTER II.

GENERAL PRELIMINARY REMARKS UPON THE LOCALIZATION OF CEREBRAL DISEASES.

(Topical Diagnosis of Cerebral Lesions.)

THE physiological relations of the brain are such that the symptoms of cerebral disease are determined to a greater extent by the locality than by the nature of the lesion. If, for example, there arises at any place a break in the continuity of the cerebral motor tract, the result, as we already know (see page 536), is hemiplegia upon the opposite side of the body. The result is precisely the same, whether the interruption is due to a hæmorrhage, an abscess, a new growth, or an embolic softening. If in any way the function of the motor fibers is suspended, then the necessary sequence in every case is a paralysis of definite extent and definite characteristics. Much the same may be said of many other symptoms which appear when there is a lesion of one or more definite places, but which are never referable to a special abnormal process, regardless of the portion of brain thereby affected.

However self-evident these simple statements may appear, it required a long time for them to gain universal acceptance among physicians. The chief cause of this was the conception entertained by the older physiologists in regard to the functions of the brain. Flourens, in 1842, taught that functionally all parts of the cerebrum were alike, and therefore any one part could act vicariously for any other; and this view had numerous adherents among physicians, as well as physiologists. It was nevertheless experience at the bedside and the autopsy-table which first led to observations and discoveries irreconcilable with this view. Above all, it was the lesions found in aphasia which forced men to localize one cerebral symptom as due to an affection of one particular spot in the brain. In 1861 Broca announced that the appearance of aphasia is always due to a lesion of the third left frontal convolution; and this was the starting-point of the doctrine of localizations in general. Nine years later (1870) appeared the famous treatise of Fritsch and Hitzig detailing successful attempts at irritation of the surface of the brain in animals, and thus overthrowing the old idea that the gray cortical substance could not be irritated. It was shown that irritation of certain places in the cortex is followed by muscular contractions in well-defined portions of the opposite side of the body, so that we are justified in assuming the existence of a number of cortical centers, the boundaries of which are comparatively narrow. These results were soon confirmed by numerous observations in cerebral pathology in man; and to-day our information about the motor functions of the cerebral cortex forms the best-known portion of the doctrine of cerebral localization. Of late years successful work has been done in this exceedingly difficult field by Meynert and Flechsig among anatomists; Ferrier, Munk, Goltz, and other physiologists; and such pathologists as Charcot and his pupils, Nothnagel and Hughlings Jackson. It is true that we are only just beginning to know something about the subject. There are numerous contradictory views asserted, and numerous questions unanswered. The following summary, therefore, is to be regarded merely as expressing the prevailing opinions now existing. Much in it will surely be altered in the course of time ; but still this doctrine of special localization of the various cerebral functions marks out in general outlines the only foundation upon which we can hope to erect a system of cerebral pathology and diagnosis. In the following sketch we shall, for practical reasons, put the results of clinical observations in the foreground, and merely speak incidentally of the corresponding experimental achievements. This will be the quickest way to gain acquaintance with the practical points in the diagnosis of what Griesinger called the "focal diseases"; and then, when we take up the separate varieties of cerebral disease, we shall have these general remarks to refer to.

1. The Motor Region of the Cortex Cerebri.

Clinical observation and the results of experiment both teach that a part of the cerebral cortex is distinct from the rest, inasmuch as it is the exclusive seat of motor functions. This "motor region" (*vide* Figs. 102 and 103, page 713) is made up of the two central convolutions (*gyri centrales anterior et posterior*, in Fig. 101), and the paracentral lobule (*vide* Fig. 103), which lies on the median surface of the cerebrum. It is also anatomically different from the other regions of the cortex, as Betz was the first to point out, for it alone possesses certain large pyramidal ganglion-cells, which are in all likelihood motor. However extensive the destructive processes which attack other parts of the surface of the brain, provided they do not involve these particular convolutions, they cause no paralytic symptoms ; while all diseases which destroy any considerable portion of the "motor" region inevitably result in a paralysis on the opposite side of the body.

We can differentiate still further. There are separate regions which act as special centers for the various groups of muscles. The center for the movements of the facial muscles (lower division of the facial nerve) lies, as it would seem, at the lower end of the central convolutions, and particularly of the anterior central convolution. Near by, apparently still lower, is found the center for the movements of the tongue. The center for the movements of the arm lies somewhat higher than the center for the facial, occupying roughly the middle portion of the anterior central convolution. The center for the lower extremity is found partly in the uppermost portions of the central convolutions, but apparently lies for the most part in the paracentral lobule. Any minuter division is not yet possible.

There are already quite a large number of cases of hemiplegia known which were caused by some disease in the motor region, like a tumor or spot of softening. We should add, in regard to the pathological anatomy of these cases, that they all, without exception, presented a secondary descending degeneration of the pyramidal tract (compare page 680), extending through the internal capsule, crus cerebri, and medulla into the corresponding lateral and anterior columns [that is, on the same side in the anterior columns and on the opposite side in the lateral]. The hemiplegia due to cortical lesion does not differ from that due to focal disease lower down in the motor tract (compare page 536) in its clinical aspects. We shall consider the symptoms more particularly in the chapter on cerebral hæmorrhage.

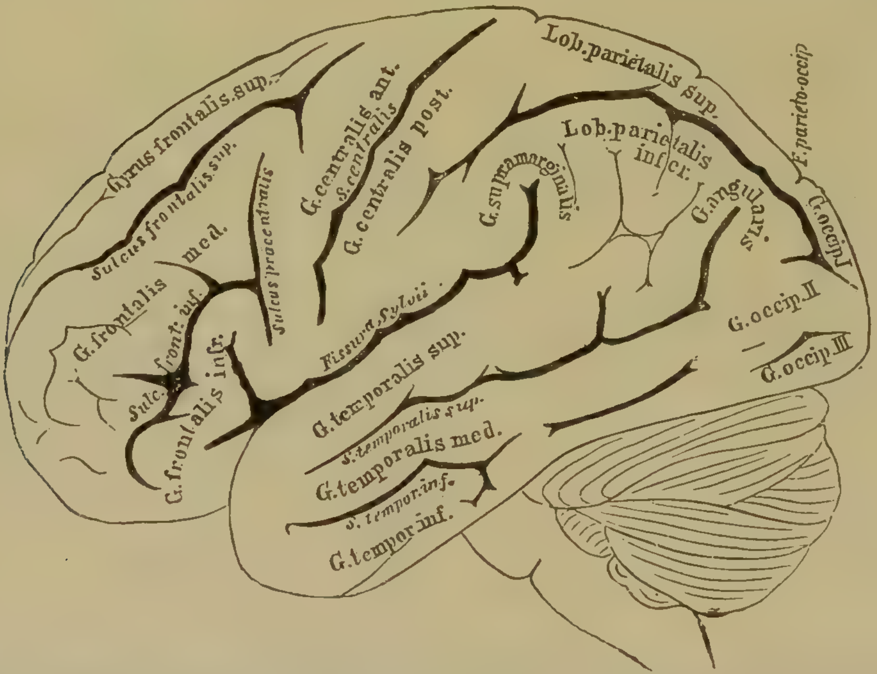

FIG. 101.—Lateral aspect of the brain (from ECKER). The gyri and lobules are in Roman type, the sulci and fissures in italics.

It is nevertheless possible in many instances to decide that the disease involves the motor portion of the cortex of the brain. This is due to the following peculiarities:

In the first place, we have already remarked (page 536) that the relative positions of the motor centers for the face, arm, and leg are such as to allow readily of isolated paralysis of any one of these portions of the body—that is, "monoplegia." In fact, we already possess a long series of observations where circumscribed lesions in the motor area of the cortex produced paralysis of one side of the face, or of one arm or leg, and of no other part. Such paralysis is termed monoplegia of the face, or the arm, or the lower extremity. And it follows, from what has been said, that even during life we can state, with considerable accuracy, the spot on the surface of the brain where the disease must be situated. Still more frequently, a combined paralysis of two portions of the body is to be observed as a result of cortical lesion; the commonest is a simultaneous paralysis of the arm and face; more rarely we see the arm and leg paralyzed together. On the other hand, we may feel certain, from the position of the motor centers, that no single center of disease could paralyze simultaneously the leg and the face, while the arm escaped injury. As a matter of fact, no such combination has ever been observed.

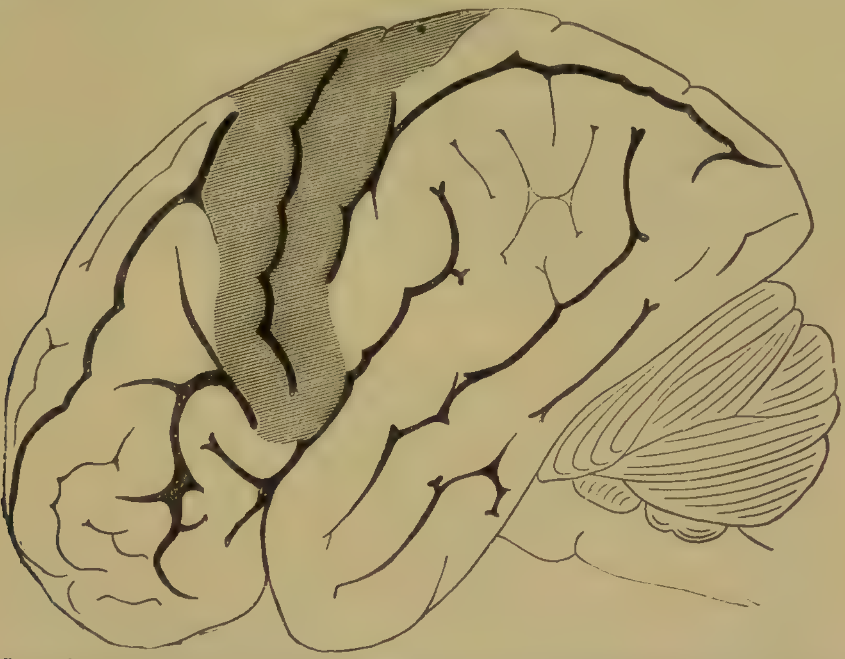

FIG. 102.—Lateral aspect of the brain (after ECKER). The motor region of the cortex, consisting of the anterior and posterior central convolutions, as well as of the paracentral lobule shown in Fig. 103, is shaded.

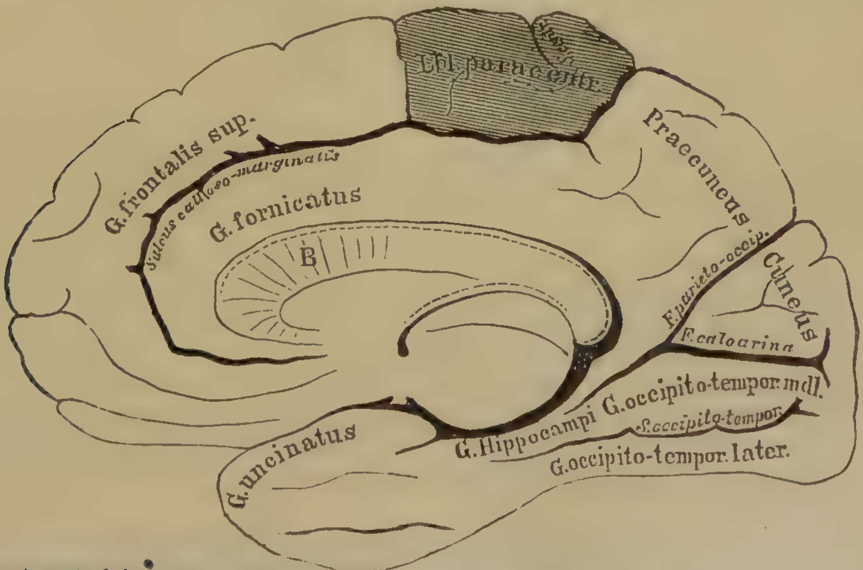

FIG. 103.—Aspect of the median surface of the cerebrum, which is shown when the two hemispheres are separated from each other by a sagittal section. B. Corpus callosum. The differences in the type have the same meaning as in Fig. 101. The paracentral lobule, as a part of the motor region of the cortex, is shaded. (Copied from ECKER, only the paracentral lobule is made more sharply prominent than in the original).

Beside this limitation of the paralysis just discussed, localized disease of the cortex has another characteristic. In it the symptoms of irritation of the motor centers are noticeably frequent. There are tonic and clonic spasms, which, like the paralysis, not infrequently affect a single arm, or an arm combined with half the face. Sometimes, however, they involve the entire half of the body. These paroxysmal spasms are termed "cortical epilepsy," or partial epilepsy, or Jacksonian epilepsy; for the movements are just the same as in genuine epilepsy. Numerous cases of disease have taught us that these circumscribed epileptiform attacks occur almost exclusively in affections of the motor cortex. They furnish information as to the precise locality of the lesion; for spasms in the distribution of the facial nerve imply that mainly the lower third of the central convolutions is affected; of the arm, the middle third; and of the lower extremity, the upper portions of the same. At the same time, the spasms and the paralysis vary greatly in their relations to each other. Often, for example, when there is hæmorrhage into the central convolutions, violent unilateral convulsions come on simultaneously with the paralysis. In the case of tumors and other lesions which develop slowly, partial epileptiform spasms will often appear quite a long while before there are symptoms of paralysis. Finally, it is not rare for epileptiform attacks to occur repeatedly in regions that are already paralytic. Either of the occurrences described in the two preceding sentences are particularly strong evidence that the cortex cerebri is diseased. Beside the pronounced epileptic attacks, disease of the motor region of the cortex may give rise to less violent symptoms of motor irritation, like occasional twitching, rhythmical twitching, and tonic contraction.

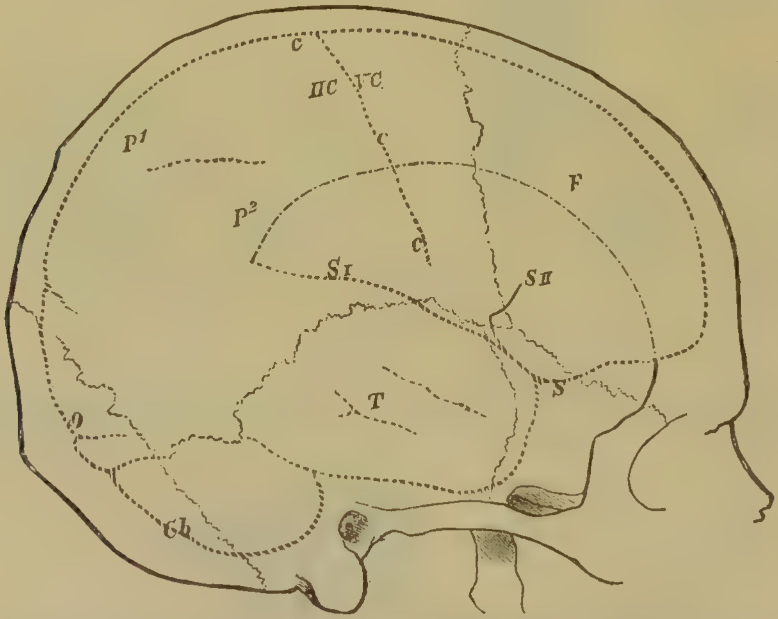

FIG. 104.—(Drawn according to ECKER.) Explanation of the topographical relations between the surface of the brain and the skull. *c.* Fissure of Rolando. *HC.* and *VC.* Posterior and anterior central convolutions. *S. S*I. and *S*II. Fissure of Sylvius. P^1. P^2. Upper and lower parietal lobes. *O.* Occipital lobe. *Cb.* Cerebellum. *T.* Temporal lobe. *F.* Frontal lobe.

About the condition of sensation when there is cortical paralysis we know as yet too little. The late experimental researches of Munk have led to the conclusion that, in animals, the so-called "sphere of sensation" lies in the same region as the motor centers of the cortex. We might, therefore, be somewhat inclined to presuppose that a disturbance of sensation would invariably accompany cortical paralysis in man also, but about this point clinical observations do not yet give perfectly harmonious results. In many cases sensation is undoubtedly normal, while in others simultaneous disturbances of sensation have been clearly demonstrated. Of especial interest is the well-attested fact that the muscular sense may be diminished in the extremities involved—that is, the patient can not tell, with eyes closed, the position of the affected limbs.

2. THE OTHER PARTS OF THE CORTEX CEREBRI, EXCEPT THE CENTER FOR SPEECH.

1. *Frontal Convolutions.*—Unilateral disease of the anterior portion of the brain may be quite extensive without causing notable disturbance of any kind. Certainly the upper two frontal convolutions have no motor functions. It is, however, maintained that the portion contiguous to the anterior central convolution, called the foot of the frontal convolutions, does contain motor centers; but even about this doubt has lately arisen. The third (lowest) frontal convolution on the left side has, as we shall soon see, an undoubted connection with the motor processes of speech.

There is a quite generally accepted view that the cortex of the frontal portion of the brain is to be regarded as the "seat of the higher psychical functions." Some few cases are on record where extensive bilateral lesions of these parts had for their only symptoms mental disturbances. In general paralysis also, and in other forms of dementia, it is very probable that the atrophy is greatest in the anterior part of the cerebrum. Nevertheless, we can not emphasize too much the fact that, at present, we have no certain knowledge about the minute relations of the psychical functions to the different sections of the brain.

2. *Parietal Convolutions.*—We know practically nothing about the functions of the cortex of the parietal lobe, and the symptoms which might imply disease of that portion of the cerebrum. The results of clinical observations thus far made with these points in view are quite contradictory. In regard to the motor functions of the parietal region, apart from the posterior central gyrus, we know only that in the supra-marginal and angular gyri seem to lie the centers for associated conjugate movements of the eyes. In the angular gyrus is perhaps also a center for the ocular portion of the facial nerve, orbicularis oculi, and levator palpebræ superioris. The parietal lobe seems to have important relations to sensibility. According to Flechsig, most of the sensory fibers of the tegmentum seem to have their central termination here. Disturbances of the muscular sense seem to be especially common with lesions in the parietal region.

3. *Occipital Convolutions.*—The clinical and experimental investigations of the last few years have all shown that the occipital portion of the cerebrum contains the cortical center for visual sensations. It is here, in all probability, that the fibers of the optic nerve terminate in the cortex cerebri. A glance at the following diagram (Fig. 105) will make it easy to understand the disturbances of vision which result from lesions of the occipital lobe. *L* represents the left eye and *R* the right, *Ch* the optic chiasma, where, as is now certain, some of the fibers of the optic nerves cross to the opposite side. The fibers (distinguished by a broken line) from the outer or temporal half of each retina extend, without crossing, into the corresponding optic tract (*Tract. opt.*), while those from the inner or nasal half of the retina cross over in the chiasma. The right occipital lobe, for example, comes in this way to receive the fibers from the outer half of the right retina and from the inner half of the left. If the right occipital lobe becomes disorganized, then the images formed upon the parts of the retinæ just named, belonging to the left half of the field of vision, are unperceived. With each eye the patient sees only such objects as lie in the right half of his field of vision, and is blind to all that lies upon his left. This sort of visual disturbance, where each

eye becomes blind to the same "homonymous" portions of the field of vision, is termed hemianopsia, or hemiopia. A lesion of the right occipital lobe causes, therefore, left-sided hemiopia, and, *vice versa*, destruction of the left occipital lobe entails right-sided hemiopia.

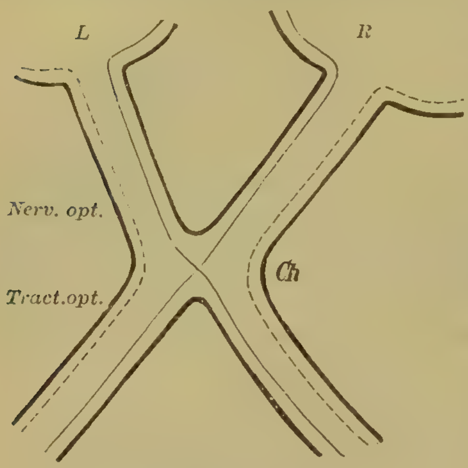

FIG. 105.—Diagram of the course of the optic fibers in the chiasma.

It may also be well to mention briefly another peculiar disturbance of vision which is perhaps due to a lesion of the occipital cortex. Fürstner noticed certain phenomena in the insane, which implied that the patient, although he could see, and could not therefore properly be called blind, yet did not recognize the objects—that is, was no longer able to interpret the meaning of the visual image. Munk has given to this condition the name of "soul blindness," conceiving it to be a "loss of visual memory." Soul blindness seems to occur chiefly in superficial lesions in the occipital region, while the special visual center itself seems to lie chiefly in the cuneus and the first occipital convolution.

4. *Temporal Convolutions.*—The relation of the temporal lobe to hearing is apparently analogous with that of the occipital to vision. Whether extensive disorganization of the temporal lobe, or of the fibers that enter it, can produce actual deafness of the opposite ear, has not yet been proven, inasmuch as very few cases have yet been studied. It may, however, be regarded as extremely probable that a lesion of the first, or uppermost, temporal convolution occasions that peculiar phenomenon known as "word deafness" or "soul deafness," with which we are at once to become more intimately acquainted.

3. THE CENTERS OF SPEECH AND THE DISTURBANCES OF SPEECH.

(*Aphasia and Allied Conditions.*)

The Various Forms of Aphasia, and their Anatomical Localization.—As we remarked at the very beginning of this chapter, the peculiar derangements of speech observed in many cerebral diseases were the first symptoms which were found to be caused by a distinctly localized cerebral lesion. For the better understanding of this extremely interesting subject, it is necessary that we should enter somewhat minutely into the processes connected with normal speech.

Incitement to speech—that is, to the oral expression of our thoughts to others—comes either from an internal impulse or from external causes which excite this impulse. Speech always requires internal mental activities—the presence of ideas and their transformation into that which we wish to communicate by speech. Where there are no conceptions there can be no words. The idiot is silent, because, like the newborn infant or the brute, he has nothing to say; but, on the other hand, the impulse to speak must also be present. In melancholic insanity we sometimes observe persistent loss of speech, not from any lack of something to say, but because there is no incitement to the act, or because inhibitory influences immediately repress any tendency to utter words. If we take for granted that the mental material for speech exists, then the transformation of this material into actual speech is a result of the following complicated processes, the disturbance of which, individually, produces the various forms of aphasia.

In the first place, the speaker must be acquainted with the word which expresses the mental conception. If, for instance, he wishes to tell another the name of some animal, he must know the appropriate word—"dog," "sparrow," "frog." This knowledge, which, as far as our mother tongue is concerned, we all acquired in childhood, may, as experience shows, be lost again in case of cerebral disease. Just as we may ourselves forget a word momentarily, or as any healthy person may, at the sight of an animal that is perhaps rather rare, be "unable at the minute to think of its name," so in disease one may forget all or a greater or less number of words. Such a patient sees a dog, and knows well enough that it is an animal possessed of such and such qualities, but he has forgotten its name. The association between the conception "dog," and likewise between the perception of a dog by the eye, and the appropriate vocal representation "dog," is lost. This condition is termed amnesic aphasia, because it is due to complete or partial loss of the memory for words. The patient knows perfectly well what he wishes to say, but the words escape him. At the same time, in cases of pure amnesic aphasia, the power of repetition is unimpaired. As soon as we say "dog" to the patient, he repeats the word perfectly well; and sometimes he also perceives that this is really the correct word; but in other instances, although the word is correctly repeated, the patient does not become conscious of its meaning (*vide infra* "word deafness").

Of great interest are certain cases where there is only partial amnesia. These have been repeatedly observed. Thus a patient forgot nothing but his own name, remembering all other words perfectly; or the loss of words may be confined to but one language, the patient being still able to express himself tolerably well in another tongue. In a case observed by Graves, the patient still knew the initial letter of all words; if, for example, he saw a cow, he would know that the corresponding word began with C, and would look under C in a dictionary till he found the word.

If the memory for words be retained, the next requisite for speaking is the transfer of the word image into such action of the muscles of our organs of speech as is calculated to produce the word in question as an actual sound. This motor process is so complicated that an extremely accurate co-ordination of movements is demanded for the correct pronunciation of the word. Man therefore possesses a separate center, in which this transfer of the word image into the motor processes of speech takes place. If this center be diseased, there again results a loss, or at least a greater or less impairment, of speech. The patient is in this case well aware of the word he wishes to say, but he can not pronounce it. He has, if we may use the expression, forgotten the movements that are essential to speaking. His tongue and lips are not really paralyzed, but the patient no longer knows how to make use of them for talking. He has reverted to the condition of childhood, before he had learned to talk. The patient often makes the greatest effort to speak. The word he wishes to utter "keeps hovering before him"; he moves his mouth in the most striking manner, but brings out only an occasional sound, and that incorrect. This form is known as ataxic aphasia. Of course it is equally impossible for the patient to repeat a word after some one else. He keeps his eyes fixed on the mouth of the speaker, and endeavors to imitate the motions of his mouth, but he is either partially or totally unable to reproduce the sound.

Ataxic aphasia exhibits many degrees of intensity. On the one hand there are cases of complete aphasia, where the patient can utter only such separate sounds as "a," "e," etc. And, on the other hand, there are also very mild cases where there are merely slight errors in pronunciation. The patient pronounces many words correctly, but with others there are such mistakes as the transposition of individual letters, the misplacement or omission of letters, or, finally, the adding on of letters. For example, he will say thens instead of then, widow instead of window, dipter instead of dipper, hefd instead of held, wrelster instead of wrestler, and belnow instead of below. This mildest form of ataxic disturbance is termed "stumbling over syllables" (*Silbernstolpern*), or "literal ataxia." In most instances the patient can pronounce some words tolerably well, others only imperfectly and with difficulty, and still others not at all. Usually the patient gradually learns a few common words and expressions (e. g., "good morning") by means of persistence in repeating them as they are uttered by another, so that he pronounces them better and better. What is very remarkable, and not so very rare, is that a patient will be able when in a passion to pronounce a word, such as an oath or an exclamation, perfectly well, because it is done to a certain extent involuntarily, while he can not utter the same words if he wishes to say them. Association also often exerts an appreciable influence; for example, a patient who finds it absolutely impossible to pronounce "six," utters it with perfect distinctness if he begins to count from one, in the ordinary way, up to six. There are many facts connected with this subject which can not here be discussed. Each case demands separate and earnest study, and will usually be found to present an abundance of interesting peculiarities.

Allied to ataxic aphasia are two other disturbances of speech, known as monophasia and paraphasia. Monophasia is very rare; in it the patient has command of but one single syllable, or a single short phrase, and this is pronounced whenever he attempts to talk. A patient of our own could for a long while utter nothing but meaningless words, "*selber sag ich nämlich selber*" (self say I namely self). The entire verbal thesaurus of another (female) patient whom we saw consisted of the meaningless sounds "bibi" and "eibibi." Still a third could say only "tinne, tinne." The patient is quite well aware that what he says is wrong, but, despite all his efforts, every attempt to speak excites these same sounds. It produces a comical impression to see the patient use the same invariable word, with all sorts of facial expression. Thus the woman mentioned above begged for things, with "bibi," in a coaxing tone, while sometimes she would give vent to violent anger with a loud "bibibibi."

Paraphasia is a confounding together of words. The association between the idea and the corresponding word is broken up, and instead other words come to the tongue. Some of these are proper words enough, but others are quite meaningless. Such a patient may talk a long while without conveying any idea to the listener, inasmuch as he says "brush" instead of "bed," or "gove" instead of "give," etc. It is a very interesting fact that in paraphasia the influence of certain associations are often manifest. The patient, for instance, utters a wrong word which has a certain resemblance in sound to the right one, begins with the same syllable, etc. Purely ideal associations sometimes also play a part; thus one of our patients called a white handkerchief "snow," etc.

As has been already shown, in amnesic aphasia the connection between the word and the conception it represents is so imperfect that the rising up to consciousness of the idea fails to call up the corresponding word. Now, on the other hand, the opposite may occur; the word, when it is heard, may fail to call up the appropriate mental image. Kussmaul has given this condition the name of word deafness (Wernicke's sensory aphasia). The patient is not really deaf, for he hears everything, but he no longer understands what he hears, and he has forgotten what the words signify. The vernacular sounds to him as a foreign tongue would to a healthy but unlearned man. A moderate amount of word deafness is very frequent in aphasia, particularly in the amnesic variety; but this last, and word dumbness, are not identical. A person may forget the word corresponding to some idea and yet understand the meaning of that word perfectly when he hears it. It is an easy matter to prove whether word dumbness exists or not by asking the patient to do something—for example, to touch certain parts of his body, or to perform certain motions (without, however, making any explanatory gestures ourselves), and seeing whether he understands and complies. Of course, the demonstration of word deafness can usually be made with regard to the names of concrete things only, and certain verbs and adjectives, but is hardly practicable in other cases (for example, with abstract words and adverbs), particularly if there be aphasia at the same time.

These various forms of aphasia, just described, seldom occur singly and unmixed. They are usually found in combinations, which vary greatly, so that a complete idea of the derangement existing in any case can be obtained only by careful examination and continued observation of the patient. Granting, however, that the variety of aphasia has been determined, what light will this throw upon the localization of the cerebral disease?

As early as 1825 Bouillaud affirmed that disease of the anterior lobes of the brain is alone capable of producing disturbances of speech. In 1836 another French physician, Marc Dax, pointed out for the first time that only lesions of the left half of the brain cause aphasia; and in 1861 Broca was able, as already stated, at last to declare that the "center for speech" lies in the third left frontal convolution. This statement has since been confirmed innumerable times, although it must be added that disease here is a cause of ataxic or motor aphasia alone. Still more accurately, it is the posterior portion of the third left frontal convolution, the pars opercularis, so called, which gives rise to this symptom. It is in this region, therefore, that those complicated processes of motor co-ordination which are essential to the utterance of a word take place. Word deafness, on the other hand, according to all the newer observations of Wernicke, Kahler, and Pick, seems invariably to be referable to disease of the first or uppermost left temporal convolution. Probably the same is true of amnesic aphasia. It is apparently, then, this region which is essential to the normal association between the auditory image of the words we hear and the appropriate mental conceptions. Any minuter localization of these and the remaining forms of disturbance of speech is at present impossible. It is probable, though not certain, that the left island of Reil also has some connection with aphasic disorders. However this may be, the corresponding portions of the right hemisphere are not usually connected with this sort of disturbance—a fact perhaps analogous with the predominant use of the right hand; that is, of the left cerebral hemisphere. It is only in a few exceptional cases—for example, of left-handed persons, or of such as present congenital defects in the left half of the brain—that observers have noticed aphasia in connection with disease of the corresponding frontal and temporal convolutions of the right side.

The diagnosis of aphasia in general is an easy matter if we stick closely to the true conception of aphasia. The examination would have to be a superficial one to lead to confounding it with bulbar symptoms such as dysarthria (*vide supra*, page 685), or with the disturbances of speech which result from other lesions which entail paresis or paralysis of the hypoglossus or certain fibers of the facial.

No general rules can be laid down as to the prognosis and treatment of aphasia, inasmuch as everything must of course depend upon the nature of the disease which excites this symptom. We will merely emphasize here the therapeutic fact that methodical exercises in speaking and in language may be decidedly beneficial. In ataxic aphasia such instruction may be imparted in about the same way as to the deaf and dumb, sight and touch being invoked to aid in a fresh training of the appropriate muscles. In amnesic aphasia the memory must be trained, that the forgotten words may once more be "stamped" upon it. Of course, all such efforts demand great tact and patience, and accomplish little unless they are methodical and persistent.

Disturbances Allied to Aphasia: Agraphia, Alexia, Amimia, and Apraxia.—Aphasia is very often accompanied by a group of other symptoms, which are likewise due to disturbance of the processes of association. Besides the language of words we possess two other means of expression—writing and gesticulation. Our mental concepts are associated not only with certain sounds but also with certain visible forms, so that, on the one hand, we use them to impart our thoughts to others, and, on the other, by their aid we learn the thoughts of other people. In aphasia this capability also is often more or less lost. If it be impossible for the patient to make himself understood by means of words, and we give him a pen, that he may write down his wishes, it is often found that this, too, is out of the question. The patient tries to write, and may indeed set down one or two words, or one or two letters, but he is no longer capable of writing a single sentence, or perhaps even a single word, correctly. This is termed "agraphia." Probably, in most instances, the agraphia is amnesic. The patient has forgotten the written characters. Usually, although not in every case, he can copy correctly what another has written. When the patient writes a wrong word in place of the right one, we call it paragraphia. Here, too, processes of mental association become manifest as in paraphasia (*vide supra*). With the disturbance in writing is often associated alexia; the patient can not read any better than he can write—that is, the characters which he sees fail now to call up the associated mental conception. Alexia is not inseparably connected with word deafness. It is possible for a patient not to understand a spoken word, and yet to recognize it at once if written.

The aphasic are also quite often unable to express themselves by pantomime or "dumb show." Frequently the patient makes no effort whatever of this kind, or what signs he does make are evidently wrong and misleading. We have repeatedly seen an aphasic patient nod his head, when it was plain that he meant to say No, and *vice versa*.

Finally, there is apraxia, a disturbance which, to be sure, is often associated with aphasia, but implies more extensive lesions. The essential point in apraxia is that the patient has more or less completely forgotten what the different objects about him really are. The condition must be closely allied to that in so-called "soul blindness." The patient sees the objects, but fails to recognize them for what they are. He takes a knife for a spoon, the washbowl for the chamber-pot, the soap for a piece of bread, and acts accordingly!

A definite anatomical localization of all these disorders which have just been briefly alluded to can not at present be given. Alexia, and still more apraxia, suggest lesion of those tracts which are connected with, or situated in, the posterior part of the cerebrum, the region of memory for visible objects.

4. THE CENTRUM OVALE, INTERNAL CAPSULE, CENTRAL GANGLIA, AND REGION OF THE CORPORA QUADRIGEMINA.

Centrum Ovale.—The white substance of the hemispheres is made up, so far as we know at present, both of commissural fibers, which connect the various cortical centers together, and of fibers which proceed downward from the centers of the cortex and connect these centers with peripheral parts of the body (corona radiata). As to the symptoms caused by diseases which destroy the commissural fibers, there is hardly anything known. We can only surmise that in case of disturbances of association, such as we have studied under aphasia and kindred disorders, there may sometimes be a lesion of commissural fibers, as of those connecting together the temporal and frontal lobes. A break in the continuity of the fibers of the corona radiata must of course result in the same symptoms as if the corresponding center were itself destroyed. This explains why circumscribed lesions of the centrum ovale, if they involve the motor fibers of the corona radiata, which proceed from the central convolutions (and only if they do this), cause hemiplegia, or, if very limited, monoplegia. In an analogous manner, disease of the white substance of the occipital lobe may entail hemiopia; of the temporal lobe, auditory disturbances, such as word deafness. More than once, quite extensive disease of the white substance of the frontal lobe on one side has been discovered post mortem, although no symptoms whatever had been caused by it. Only when the coronal fibers which proceed from the third left frontal convolution are involved in disease is motor or ataxic aphasia inevitable.

Internal Capsule.—The most important facts relating to the functions of the internal capsule, as far as at present known, have been already stated. In particular, it was pointed out that through the posterior limb of the internal capsule, in a comparatively narrow space, passes the pyramidal tract on its way from the central convolutions to the crura cerebri (see Fig. 64, page 534). Here, then, even a very limited focal disease must lead to complete hemiplegia on the opposite side of the body. Clinical experience also shows that the largest number of cases of persistent hemiplegia are occasioned by disease in this spot. In these cases the facial nerve is usually also involved, although its fibers apparently lie somewhat further forward than the tracts which are destined for the extremities.

The sensory tract (compare page 511, and Fig. 64, page 534) lies at the posterior extremity of the internal capsule, and apparently includes not only the fibers for cutaneous sensation, but also for the organs of special sense. Complete disorganization of this spot ought therefore to cause, in the opposite half of the body, not only anæsthesia of the skin, but also simultaneously a corresponding impairment of smell, taste, and hearing, and hemiopia—in short, a so-called complete cerebral hemianæsthesia. Still, in regard to this very point we are much in need of further and definite observations. Charcot's statement that in these cases the disturbance of vision is not hemiopia, but total amblyopia of the eye opposite to the focal lesion, has caused great confusion, for such a fact would of course be hard to reconcile with the appearance of hemiopia in diseases of the occipital lobe, although this latter occurrence is well established. However, this statement of Charcot's is by no means absolutely proven, so that meanwhile we are at liberty to maintain the view that the visual disturbance in cerebral hemianæsthesia may be hemiopia.

Certain practical conclusions in regard to diagnosis can be deduced from the preceding facts. A purely motor hemiplegia, unattended by impairment of sensation, implies a lesion that does not involve the posterior portion of the internal capsule; but probably this portion is also affected, when there is not only paralysis, but considerable sensory disturbance. The sensory disturbance does not invariably extend to all the senses ; quite often there is nothing but cutaneous anæsthesia.

About the importance of the other parts of the internal capsule, not mentioned here, nothing is known. A statement made by Charcot should be added in conclusion—namely, that when there are also post-hemiplegic symptoms of irritation, such as post-hemiplegic chorea, there is very probably a lesion of the posterior extremity of the internal capsule.

Central Ganglia; Caudate Nucleus (Corpus Striatum proper), Lenticular Nucleus, and Optic Thalamus.—Before the course of the pyramidal tract had been accurately determined, ordinary cases of cerebral hemiplegia were almost universally ascribed to lesions of the central ganglia, and in particular of the caudate and lenticular nuclei. At present, however, observations seem to force one to the conclusion that a complete hemiplegia can be produced only by a cutting off of the pyramidal tract. There are, indeed, numerous cases of hemiplegia presenting circumscribed disease of the central ganglia; but they can probably all be explained by supposing either that the pyramidal tract, as it lies in the contiguous internal capsule, is directly involved in the disease, or that its functions are suspended by the indirect effects of the neighboring lesion—for instance, by the pressure it exercises. Accordingly, we find that circumscribed lesions of the central ganglia in the neighborhood of the internal capsule generally produce a temporary hemiplegia—that is, the paralysis gradually improves as the indirect influence of the focal disease upon the internal capsule ceases. Chronic and incurable hemiplegia, however, if due to a lesion anywhere in this region, always implies an actual lesion of the pyramidal tract in the internal capsule. In regard to hemianæsthesia, also, the facts seem to be quite analogous. It was formerly held that this phenomenon was especially connected with a lesion of the optic thalamus, the reason being that the sensitive fibers lie so close to the thalamus in the posterior extremity of the internal capsule that they become themselves involved.

About the symptoms which lesions of the central ganglia directly produce little is definitely known. The results of both clinical observation and experiment are quite contradictory, and in repeated instances quite extensive disorganization of these parts has existed without producing any symptoms to speak of during life. In particular, it should be borne in mind that softening may occur in the lenticular and caudate nuclei, and yet not a trace of hemiplegia be observable. It seems probable also that the optic thalamus has nothing to do with voluntary motion; but it seems to have some importance for movements of mimetic expression (Bechterew, Nothnagel). In hemiplegics we sometimes see that one half of the face can not be moved voluntarily, but it is moved very actively in emotional expression, such as laughing or crying. In such cases we can decide that the thalamus is intact, while in lesion of the thalamus the contrary is observed—the affected half of the face can be moved voluntarily, but it remains completely rigid in emotional expression. As to perceptive functions, there is only one which the optic thalamus is certainly known to possess: the central termination of some fibers of the optic nerve lies in its posterior portion (known as the posterior tubercle or pulvinar), while other fibers go to the corpus geniculatum externum. Destruction of the hinder part of the thalamus accordingly produces complete hemiopia (*vide* page 716) of the opposite side. It has been affirmed that the optic thalamus has connections with other sensory tracts, but definite proof is lacking. Focal disease of the thalamus has repeatedly occasioned "post-hemiplegic chorea" and other post-hemiplegic symptoms of irritation. According to recent experiments, the lenticular nucleus contains centers for regulating heat.

Corpora Quadrigemina and Crura Cerebri.—Diseases of the corpora quadrigemina are infrequent, and usually, when they do occur, they are merely a part of more extensive lesions of the brain. They are therefore very rarely considered from a diagnostic point of view.

The anterior tubercles are certainly connected with the fibers of the optic nerve. If both of the anterior tubercles be destroyed, total blindness is inevitable, while if only one be disorganized, hemiopia is to be anticipated. Still, these symptoms are, of course, too ambiguous ever to be regarded as pathognomonic of localized disease of the anterior corpora quadrigemina. Another point to be considered, whenever the corpora quadrigemina are diseased, is the position of the nuclei of the nerves which preside over the motions of the eyeball, and in particular of the oculomotor nerve. This explains why unilateral, or even bilateral, paralysis of the oculomotorius has been repeatedly observed in connection with lesions of the corpora quadrigemina, as have also nystagmus and immobility of the pupil.

[Nothnagel considers that a staggering gait, resembling that of a drunken man, associated with oculomotor paralysis, if associated with general symptoms of tumor, is strongly indicative of tumor of the corpora quadrigemina. The eye paralysis must, however, be an early symptom in order to be of value. The paralysis in such cases is apt to be of irregular distribution, often involving upward and downward movements of the eye.—K.]

Should the crura cerebri become involved in the disease, the resulting symptoms are often very characteristic of the locality affected; there is paralysis of one side of the body (arm, leg, facial nerve), and at the same time a crossed paralysis (that is, one situated upon the opposite side) of the motor oculi. A glance at Fig. 65 (page 535) will explain this phenomenon. Thus, a circumscribed lesion on the right side would destroy the fibers of the third nerve on that side, and therefore produce a right-sided paralysis of the oculomotor nerves, and at the same time, if extensive enough, the lesion would involve the pyramidal fibers of the right crus, and thus occasion left hemiplegia. That disease of the tegmentum would necessarily entail sensory disturbances may be taken for granted, although there is as yet very little clinical proof of it.

5. The Cerebellum.

Quite extensive destruction of the cerebellum may take place without any symptoms to indicate it. In such cases, however, the disease is almost invariably confined to the hemispheres; but, if the central portion or vermiform appendix be attacked to any great extent, peculiar symptoms almost always result, pointing in many instances with considerable certainty to disease of the cerebellum.

There are two especially characteristic symptoms—a peculiar uncertainty of gait (cerebellar ataxia), and troublesome vertigo.

Cerebellar ataxia affects only the trunk and lower extremities. Both standing and locomotion are interfered with. When the patient is lying in bed he can move his legs almost as well as ever, and with normal vigor; but as soon as he gets up, the characteristic motor disturbances become very evident. Even while standing still the whole body can usually be plainly seen to sway back and forth. This becomes more marked if the patient bring his heels together. If he stand with his legs widely apart, the trouble is less noticeable. Closing the eyes, as a rule, does not aggravate the swaying, inasmuch as the cutaneous and muscular sensibility of the lower limbs remains normal in uncomplicated cerebellar disease. When the patient tries to walk, he sways and totters, precisely as if he were deeply intoxicated, but usually in a very different way from that seen in tabes dorsalis. Instead of the uniform stamping and pitching gait of the latter, cerebellar ataxia causes a real staggering of the whole body, so that in severe cases the patient loses entirely the ability to walk straight, but seems to fall forward, as it were, in a zigzag line, now to the right and now to the left. Sometimes, but by no means invariably, it is noticed that, in walking, the body sways principally in one particular direction, either forward or backward, or to one side. Such peculiarities, however, do not enable us, with our present knowledge, to determine with certainty just what position in the cerebellum the lesion occupies. The most we can do is to surmise, in such a case, that the middle peduncles of the cerebellum (*vide infra*) are involved. It is worthy of note that, with few exceptions, the ataxia does not involve the upper extremities. Many a patient who can scarcely walk unaided is still able to perform the most delicate manipulations with his hands. This shows that it is only in maintaining the bodily equilibrium—essential to standing and locomotion—that the function of the cerebellum is important.

As already stated, this cerebellar ataxia is in most cases attended with pronounced vertigo. There is not, however, a complete correspondence between the locomotor disturbance and the dizziness. Exceptionally one symptom may be present without the other. The vertigo is usually felt only when the patient stands or moves about, being almost always absent when he lies quietly in bed. We are as yet ignorant just how it is produced. Vertigo is quite frequently produced by other cerebral diseases. It can not, therefore, be regarded as indicating disease of the cerebellum unless it is very persistent and decided, and is associated with the characteristic cerebellar gait.

Little is known about other symptoms of disease in the cerebellum. There may be some diagnostic value in persistent occipital headache, particularly if other cerebellar symptoms be present. If they do not exist, the headache is too ambiguous to be of much value; and, besides, an affection of the cerebellum may exceptionally be attended by pain in the side of the head or in the forehead. Vomiting is of still less value. It is, to be sure, frequently occasioned by chronic disorders of the cerebellum, and by tumors in particular, but may be equally well the result of disease elsewhere. Tumors of the cerebellum are very apt to cause disturbances of vision; but it is certain that these are not a direct result of the cerebellar lesion, but are occasioned by the development of a choked disk (*vide* chapter on cerebral tumors).

We must add in conclusion a few words about diseases of the middle peduncles (crura ad pontem). Usually it is an irritation of these which causes those peculiar symptoms known as forced movements and forced positions. Thus, in such a case, the patient always lies upon one particular side in bed. He may be quite conscious, or in a state of complete unconsciousness. If he is put in any other posture, he at once involuntarily reassumes his former position. Not infrequently this forced position of the trunk is accompanied by a corresponding forced position of the head and eyeballs, while the extremities are seldom affected. Genuine forced movements are seen far less often. They produce either often-repeated rotations of the body on its longitudinal axis, or, if the patient be able to walk at all, involuntary circular movements ("circus movements"), etc. It is not possible, even by minute analysis of these symptoms, to determine in which of the two peduncles the source of irritation exists. In a few very rare cases of brain disease these same symptoms have been observed, although no affection of the middle peduncles of the cerebellum could afterward be detected.

For convenient reference we subjoin a summary of the most important facts bearing upon the localization of cerebral diseases.

1. The most frequent cause of ordinary hemiplegia is a lesion of the pyramidal tract in the posterior limb of the internal capsule. If the hemiplegia be persistent, then this tract is actually destroyed; if temporary, the tract has been functionally deranged for a time by focal disease in neighboring parts of the brain.

2. Monoplegic cerebral paralysis is usually due to affections of the cortex of the brain, that is, the central convolutions and the paracentral lobule. Monoplegia of the face and tongue is the result of lesions in the lower extremity of the anterior central convolution. Monoplegia of the arm is referable principally to some lesion of the middle third of the anterior central convolution. Monoplegia of the lower extremity implies some affection of the paracentral lobule.

3. Hemiplegia or monoplegia, if associated with epileptiform convulsions affecting either one half or one particular portion of the body, are almost always caused by cortical lesions. These same symptoms of motor irritation without accompanying paralysis are likewise ascribable to some irritation of the above-mentioned regions of the cortex.

4. Hemiplegia with crossed paralysis of the oculomotor nerve indicates a lesion of the crura cerebri.

5. Hemiplegia with crossed facial paralysis implies, with an approach to certainty, that the lesion is situated in the pons.

6. Post-hemiplegic chorea (*vide infra*) seems to occur especially when there is focal disease in the neighborhood of the optic thalamus or of the posterior part of the internal capsule.

7. Hemianæsthesia (of the skin and of the organs of special sense) seems to result principally from lesions of the most posterior portion of the internal capsule.

8. Hemiopia may be due to a lesion of the occipital lobe. Probably, also, a lesion of the posterior extremity of the internal capsule may cause it, in which case it is usually associated with hemianæsthesia. Finally, it may be produced by affections of the posterior tubercle of the optic thalamus, or of one of the anterior corpora quadrigemina, or of one of the optic tracts.

9. Genuine motor aphasia indicates disease of the third left frontal convolution.

10. Word deafness seems to be due to disease of the first left temporal convolution.

11. Difficulty in articulation implies disease of the medulla, as does also dysphagia.

12. Staggering gait and vertigo are the most constant symptoms of cerebellar disease. Forced positions and forced movements are seen chiefly in connection with lesions of the crura cerebelli ad pontem.

[13. Staggering gait and ocular paralyses are indicative of lesions of the corpora quadrigemina.–K.]

CHAPTER III.

CEREBRAL HÆMORRHAGE.

Ætiology.—The cause of cerebral hæmorrhage should always be sought in some disease of the coats of the minute cerebral arteries. In 1868 it was first shown by Charcot and Bouchard that in almost every case of cerebral hæmorrhage there are miliary aneurisms of the small arteries of the brain substance, some one of which has burst and allowed the blood to escape. All later investigators have confirmed their statements about the occurrence and importance of these miliary aneurisms. The aneurisms may attain a diameter of a millimetre or more. They usually appear like spindle-shaped dilatations of the entire circumference of the vessel, although sometimes the bulging is confined to one side of it. So far as has yet been learned, the process of development starts with disease of the intima. This layer presents at first proliferations and also a fatty degeneration of the endothelium. Later on, however, the intima atrophies, just as the muscular coat does. Inasmuch as the intra-cerebral arteries possess almost no true adventitia, it is easy to see that these vessels are especially predisposed to aneurismal dilatation. It has been affirmed that the disease of the vascular wall, which leads to the formation of these aneurisms, is identical with ordinary arterio-sclerosis (*vide* page 331) or atheroma. Charcot denies it, but the later investigations of Eichler make it seem very probable. Indeed, we very often, although not invariably, find that cerebral hæmorrhage attacks persons who present either a general arterio-sclerosis or a more limited atheromatous disease of the cerebral arteries; and most of the factors which are said to promote cerebral hæmorrhage are the same as favor the development of arterio-sclerosis.

It has long been recognized that age has an important bearing on these cases. Although exceptionally a younger individual may be attacked, the majority of sufferers are over fifty years old—that is, at the time of life when arterio-sclerosis usually becomes most fully developed. Again, cerebral hæmorrhage is decidedly more frequent in men than in women, which is also true of atheroma. Alcoholism, syphilis, and gout are also reckoned among the ætiological factors of both disorders, and in both a hereditary predisposition is not very rarely demonstrable. What is called the "apoplectic habit" also deserves brief mention. Although there is no variety of constitution which exempts its possessor from the possibility of cerebral hæmorrhage, yet it can not be denied that often the victims of apoplexy do exhibit a certain "habit." Such persons are not very tall, but are corpulent, broad-chested, with a short, thick neck and round face. They have not been disinclined to the pleasures of the table and the bottle, and sometimes they suffer from emphysema, moderate hypertrophy of the heart, and general arterio-sclerosis, as the condition of the radial and temporal arteries may disclose even during life.

Granting, therefore, that disease of the arteries, and more particularly miliary aneurisms resulting from chronic endarteritis of the smaller cerebral arteries, must be regarded as the chief cause of cerebral hæmorrhage, then, on the other hand, the question suggests itself whether an abnormal elevation of the blood-pressure may not have some part in determining the hæmorrhage. If the coats of the arteries be normal, surely they would not be torn, no matter how great the arterial tension became; but if aneurisms have already been developed, then there can be no doubt that a persistent or even a temporary elevation of the blood-pressure must favor the bursting of the vessels.* In this sense a cerebral hæmorrhage, occurring in patients with certain forms of cardiac hypertrophy (contracted kidney, idiopathic hypertrophy, etc.), combined with disease of the vessels, may be referred in part to the increased arterial tension; but it is most of all with regard to many exciting causes, which are immediately followed by a cerebral hæmorrhage, that increased blood-pressure assumes great importance. Here it is temporary. Cerebral hæmorrhage may, for example, follow excessive muscular exertion, the ingestion of a large amount of food, indulgence in alcohol, taking a cold bath, or violent mental excitement. In all such cases, however, the change in the arteries is a necessary prerequisite.

It should be mentioned, in conclusion, that quite considerable hæmorrhage may also take place in the course of such general diseases as are associated with impaired nutrition and a consequent diminution in the resisting power of the vascular walls. Cerebral hæmorrhage under these circumstances is merely symptomatic of a general hæmorrhagic diathesis, as we find it in leukæmia, pernicious anæmia, and those affections which are called, in a stricter sense, hæmorrhagic diseases, such as scurvy and purpura hæmorrhagica. The grave infectious diseases, including septicæmia, typhus or typhoid fever, and variola, may occasion hæmorrhage into the brain as well as into other organs. The hæmorrhages are generally, however, from capillary vessels, and are very rarely extensive.

Pathology.—The miliary aneurisms do not develop in all the cerebral arteries with equal frequency, and accordingly we find certain regions particularly liable to cerebral hæmorrhage, being very much oftener affected by it than others. It is the large central ganglia in the neighborhood of the lateral ventricles that suffer most frequently—namely, the optic thalamus, caudate and lenticular nuclei, and also the adjacent white matter of the internal capsule and centrum ovale. Hæmorrhages in other portions of the brain are much less frequent—such as hæmorrhages into the convolutions, the pons, the cerebellum, the crura cerebri, or the medulla. If the blood escapes into the neighborhood of a ventricle, it may burst into the latter. Likewise, in rare instances, an effusion of blood near the cortex may make its way out upon the surface of the brain.

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* [Mendel has found that the normal blood-pressure in the lenticulo-striate arteries, which are most prone to rupture, is much higher than it is in the cortical and many other small arteries of the brain.—K.]

An extensive collection of blood in one of the hemispheres may exercise so decided a pressure upon surrounding parts that the results of increased tension upon the affected side are at once recognized when the skull is opened. The dura on that side is more tightly stretched, the falx is crowded over to the opposite side, the convolutions on the convexity seem flattened, and the furrows are shallow. Exceptionally, when there is a very large effusion reaching nearly to the surface, we may even detect fluctuation.

On cutting through the brain-substance we find the seat of hæmorrhage, and are enabled to determine its position and extent. Its size, of course, varies considerably in different cases; it may be small, or it may occupy a large part of an entire hemisphere. The wall of the effused mass is made up of ragged and torn cerebral tissue, and the mass itself contains *débris* of the nervous elements entangled in the coagulated blood. The blood-clots are almost always very dark-colored when fresh. Later on, the mass changes to a chocolate-colored or more brownish-yellow pulp, composed of the disintegrated remnants of the nervous substance and the clotted blood. The microscope reveals, particularly in the immediately surrounding tissues, numerous fatty granular cells. These are white blood-corpuscles which have absorbed the fat resulting from the decomposition of the myeline substance. There is also always an abundance of hæmatoidine crystals, due to the disintegration of the red blood-globules. At a greater distance from the effusion the tissues present a yellowish tinge, from the imbibition of such blood-pigment as has reached them in a state of solution; and there is also usually an œdematous softening of the parts not too far removed from the hæmorrhagic focus.

If the patient survive, the mass is gradually absorbed. It slowly diminishes in size, and the surrounding parts tend to reassume their normal relations. The final result in many cases is a cavity filled with serous fluid and bounded by smooth walls. This "apoplectic cyst" remains stationary. In some instances, however, and particularly if the effusion be rather small, the walls approach each other as more and more of the fluid is absorbed; there is a great hyperplasia of connective tissue; and so, finally, there is nothing left but a so-called apoplectic scar, usually of a yellow color, due to vestiges of the blood-pigment. The position and dimensions of the permanent lesion determine, of course, the question of secondary descending degeneration (*vide* page 680), as well as the nature and extent of the persistent clinical symptoms.

Clinical History.—The symptoms of cerebral hæmorrhage agree closely with the anatomical lesions just described. The miliary aneurisms themselves, even when numerous, seldom excite any symptoms. Sometimes, however, it is possible that the slight circulatory disturbances they occasion produce the mild headache and similar annoyances which often precede, for a longer or shorter time, the occurrence of cerebral hæmorrhage.

As soon as an aneurism bursts, however, and blood escapes into any part of the brain-substance, there is immediately seen a group of grave cerebral symptoms, collectively termed an apoplectic attack, or "shock." As the blood escapes under a pressure nearly equal to the general arterial pressure, and doubtless much greater than that to which the soft substance of the brain is normally exposed, the affected portion of the brain is at once subjected to a considerable increase of tension, which is transmitted for various distances in all directions. It need not be said that the destructive influence of the hæmorrhage may vary exceedingly, and that therefore the symptoms are by no means equally severe in all cases. The larger the rent in the blood-vessel, and the more rapid and abundant the consequent hæmorrhage, the worse is the apoplectic attack. Bleeding from larger vessels is therefore usually attended by graver symptoms than from the minute arterial twigs. An extensive cerebral hæmorrhage sometimes causes the patient to fall down suddenly in complete unconsciousness, while smaller hæmorrhages may occasion only a temporary attack of vertigo and slight cloudiness of intellect. If the tear in the wall of the artery be very small and narrow, permitting the blood to escape but slowly, then there may be no sudden attack at all, the phenomena requiring a certain length of time for their development.

There is also an important relation between the location of the hæmorrhage and the severity of the apoplectic attack. The chief symptom of these cases is loss of consciousness (about which we shall soon speak at length); and as this certainly is due to an interruption of the functional activity of the cerebral cortex, it is plain (1) that the nearer the cortex is to the hæmorrhagic focus, the more apt are the symptoms to be serious. It is confirmatory of this that hæmorrhage into the more deeply situated portions of the brain, like the crura cerebri or the pons, quite often occasions comparatively slight symptoms. But (2) there is a fact about the circulation in the brain that often causes the shock from hæmorrhage into the brain-stem to be greater than the shock following hæmorrhage into the cortex or the white substance of the hemispheres. The explanation is that the brain-stem has comparatively much larger arteries than the other parts just mentioned, which contain only minute blood-vessels. Furthermore, as Duret and Heubner have shown, the blood-vessels are so distributed that the arterial tension in the brain-stem is not a little higher than in the other portions. This renders intelligible the clinical phenomenon that hæmorrhages in the territory of the main arteries, besides being, as we have said, the most frequent of any, produce apoplectic symptoms even when the effusion is comparatively small; while sometimes hæmorrhages of about the same size in the cortex or white substance may not be noticed.

The clinical phenomena of the apoplectic attack will now be considered in detail. The onset is sometimes absolutely without warning, but in other cases it is preceded for a greater or less length of time by certain prodromata. These are either the result of the disturbance of circulation caused by the disease of the blood-vessels in the brain, and then, as already stated, they comprise occasional headache, vertigo, tinnitus aurium, spots before the eyes, languor, and muscular weakness; or they are caused by minute hæmorrhages, which seem not infrequently to precede a greater one. In such a case, the friends relate that of late the patient has had one or more slight and brief attacks, characterized by faintness, temporary trouble in speaking, sudden but temporary weakness of an arm or leg, and similar symptoms. The prodromata may extend over several days or even weeks and months preceding the severe attack.

In other cases there are no such premonitory symptoms. The apoplexy occurs unexpectedly and suddenly. In the midst of apparently vigorous health the patient sinks down "as if he had been struck." In still other cases there are indeed no prodromata, but the symptoms do not at first appear in all their severity, and occupy some hours or even a whole day in their gradual development. This is due to a slow and gradually increasing hæmorrhage, and is termed a slow or delayed apoplectic attack. The patient grows confused, anxious, and delirious (a case of our own had pronounced hallucinations of sight); the arm and leg on one side become paretic, and gradually more and more completely paralyzed; and after a few hours complete unconsciousness comes on.

The attack may be rapidly fatal. In such cases the abnormal pressure probably involves the medulla oblongata and paralyzes the cardiac and respiratory centers there situated. Usually, however, there is merely a complete loss of consciousness, more or less rapidly developed. Sometimes the patient has time to lie down. He usually sinks back in his chair or falls to the floor, and becomes deeply comatose. The face is often noticeably flushed, and the pulse full and tense, but not infrequently somewhat slow, because of the increased cerebral pressure. The respirations are deep, noisy, stertorous, and likewise often slow. The relaxed cheeks and lips are often drawn deeply in at every inspiration, and puffed out at every expiration. The temperature is usually subnormal at first, later regaining the normal, or even a higher point. In a rapidly fatal case, however, the temperature remains depressed till death. It is not very rare in severe cases to observe a peculiar position of the head and eyeballs, they being all turned in one direction. This phenomenon is termed by Prévost conjugate deviation (*déviation conjuguée*) of the eyes and head. It is generally temporary, and is said by Landouzy to be connected principally with a lesion of the lower temporal lobe. There is no perfectly constant relation between the lateral deviation and the half of the brain affected. Apparently in most cases the eyes are directed toward the injured side, and so to a certain extent "look toward the lesion" and away from the paralyzed side of the body (*vide infra*). The pupils present no constant peculiarities. Often they are of normal size. In other cases they are contracted, dilated, or unequal. No definite diagnostic conclusions can be drawn from them. In the worst cases the pupils will not react to light; in other cases they react, but often sluggishly.

During the deep apoplectic coma the extremities generally lie completely motionless and limp. In the worst cases reflex action is wholly suspended; but sometimes the vigorous thrust of a pin or the pinching of the skin will excite an occasional slow reflex twitch, or a motion as if to ward off the tormentor. Whether the apoplexy has caused hemiplegia at all, and if so on what side, can not always be easily determined during the initial coma. Still, it is often to be observed, even now, that one angle of the mouth hangs down lower than the other, and that the corresponding cheek is more puffed out during expiration than is the other; that the extremities of one side are even more completely relaxed and limp than those on the opposite side of the body, and that the reflex action and defensive movements are almost absent upon one side (the paralyzed side), while they can be clearly demonstrated on the other.

In contrast to the usual laxness of the arms and legs during the apoplectic coma is the tonic rigidity sometimes seen in the extremities, particularly on the side opposite to the hæmorrhage. This symptom seems to be especially, although not exclusively, connected with a bursting of the escaping blood into a lateral ventricle. It is rather exceptional for cerebral hæmorrhage to be attended with general or unilateral epileptiform convulsions—a symptom which, as we have seen, is referable to irritation of the motor regions of the cortex.

It should be mentioned that in many cases of cerebral hæmorrhage the urine passed after the attack has been found to contain small amounts of albumen or sugar. This symptom is usually ascribed to compression of the medulla from the effusion. There is usually retention of urine; in other cases there is involuntary micturition.

A certain number of patients never awake from the initial coma. Death may not be immediate, but they remain completely unconscious; the respirations become more rapid and irregular (sometimes of the Cheyne-Stokes character), and there is a rattling in the throat, because mucus and saliva run down into it; the pulse, which was at first retarded, now becomes accelerated; the face grows paler and more and more sunken; the eyes are deep in their sockets; the cornea becomes opaque; and at last, after the coma has lasted some hours, or even one or two days, death occurs, often attended by a considerable rise in temperature.

This termination is, however, by no means the usual one. More frequently the patient survives the attack. The bleeding ceases, the clot contracts, and begins to be disintegrated and absorbed. At the same time the pressure exerted upon surrounding parts grows less and less, the more distant parts of the brain gradually recover from the shock, and consciousness slowly returns. The patient begins to open his eyes when he is spoken to in a loud tone; he raises his hand to his head, sighs, and yawns; gradually the intellect clears up, he tries to talk, or to express himself by signs; memory returns, and he recognizes those about him once more. Exceptionally, recovery is interrupted by a fresh and perhaps a fatal relapse. This may result from a renewal of the hæmorrhage. Generally, however, improvement persists, the patient fully regains his consciousness at the end of a few days, and it now becomes possible for the first time to "estimate the damages."

The symptoms thus far described belong to severe apoplectic attacks. There are also, as we have said, cases of all degrees of mildness, as regards the first onset. In these there is no deep and persistent coma. The patient loses consciousness only temporarily, if at all. He is seized with vertigo, or with sudden headache, and is for a time stupefied. Nausea and vomiting are of quite frequent occurrence, just as in ordinary syncope. Yet cases presenting these comparatively slight early symptoms, and with few even of these, may exhibit the genuine focal symptoms referable to the hæmorrhage, such as hemiplegia, in all their severity. These latter phenomena must now be considered.

Among the symptoms of impairment of function resulting from cerebral hæmorrhage, only those are to be regarded as direct focal symptoms which are caused by the actual destruction of a region of the brain. Where the hæmorrhage takes place we have seen that a larger or smaller extent of the brain-substance is completely destroyed by the sudden and forcible escape of the blood. The dimensions of this lesion are represented later by the apoplectic scar or cyst, and its position by the nature and extent of the persistent, and for the most part irreparable, loss of function (*Ausfallserscheinungen*). But there are, in addition to these direct symptoms, other indirect focal symptoms, which outlast the apoplectic shock, and vary with the locality of the hæmorrhage. These do not, however, correspond to the territory actually destroyed. They are due to the influence exerted for a certain length of time by the hæmorrhagic focus upon the immediately surrounding structures. The pressure of the effusion, the disturbance of circulation resulting from it, the collateral œdema, and perhaps also the imbibition of the soluble products of disintegration, are the chief factors in exciting these indirect symptoms. They do, indeed, outlast the initial shock, but are nevertheless temporary, vanishing sooner or later, at the end of several days or weeks, or even months.

Even if the character of the initial attack and the symptoms still exhibited have been minutely determined, yet we are unable to say, at first, whether the existing focal symptoms are direct or indirect. We can decide about this only after further observation. If the early symptoms disappear within the next few days or weeks, or after the first two or three months, we can then affirm, retrospectively, that they were indirect. Such as outlast the first six months are to be regarded as direct, and as not destined to improve much. From a practical point of view this distinction is of extreme importance. We shall revert to the subject when considering the course of cerebral apoplexy.

A minute description of all the focal symptoms which might occur after cerebral hæmorrhage, and of the light thus thrown upon the location of the hæmorrhage, need not be attempted here, for it would necessitate a repetition of all the facts enumerated in the preceding chapter. It is only requisite to describe, in detail, the chief and by far the most frequent result of a cerebral hæmorrhage—ordinary cerebral hemiplegia.

It has been mentioned that most of these hæmorrhages occur near the lateral ventricles. Hence, in a majority of instances, the motor pyramidal tract, as it traverses the internal capsule, is either directly destroyed or at least indirectly affected. Consequently, most patients who survive the apoplectic shock present a paralysis of that half of the body which is opposite the seat of hæmorrhage. On minute examination we usually find, in the first place, that even in the distribution of the facial nerves there is a distinct difference between the two sides, the lower division of the facial (which supplies the muscles of the cheek, nose, and mouth) being evidently paralyzed on one side, while its upper division (going to the eyes and forehead) is entirely, or almost entirely, intact. The forehead can be wrinkled on one side as well as on the other; but if the patient tries to turn up his nose, or alter the shape of his mouth, the paralysis becomes evident. Often indeed, while the face is quiet, it can be noticed that one naso-labial fold is obliterated, or that one corner of the mouth hangs down. It is an interesting fact that the paresis of the lower division of the facial is much more noticeable during voluntary efforts, as in showing the teeth, than when the patient smiles involuntarily. A patient will sometimes try in vain to draw back the corner of his mouth, then begin to laugh at his own awkwardness, and thereupon open his mouth in an almost perfectly normal manner. We have seen above, in the chapter on cerebral localization, that this condition can probably be explained by the relation of the optic thalamus to the movements of mimetic expression. Why there is such a difference between the upper and lower divisions of the facial nerve in cerebral hemiplegia is not certainly known. Possibly it has some connection with the fact that the muscles supplied by the upper division (frontalis, corrugator supercilii, and to a certain extent the orbicularis) are very seldom exercised upon one side alone, but always bilaterally. Perhaps both sides receive nervous fibers from each cerebral hemisphere, so that, if a single center be intact, it alone answers for the muscles on both sides.* Sometimes, however, careful examination shows a slight paresis of the frontalis muscle on the paralyzed side. In the distribution of the lower division of the facial, also, ordinary cases of cerebral hemiplegia present almost always a more or less marked paresis, and only exceptionally a complete paralysis.

There is quite often a slight impairment of the hypoglossus in addition to the paresis of the facial nerve. If the patient puts out his tongue, its tip deviates toward the paralyzed side. This is a result of paresis of one of the genio-hyoglossi. When both these muscles contract, they may be said to push the tongue forward. If this thrust be more vigorous on one (the healthy) side, the tongue is deflected toward the other (paralyzed) side. In ordinary cerebral hemiplegia this is almost always the sole way in which the movements of the tongue can be seen to be impaired. Sometimes, however, the slight paresis of half of the tongue, combined with the facial paresis, entails a noticeable difficulty in articulation. This, to be sure, is not great, and is often appreciated by the patient alone, who is conscious that an effort is required in order to speak.

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* There is a remarkable general rule which should be mentioned here, and which is perhaps to be explained in the same way, namely, that those muscles which are usually called into play in pairs are never completely paralyzed in cerebral hemiplegia. Furthermore, it is impossible for us to contract most of these singly, on one side alone, or at least not without special practice; this applies, for instance, to the corrugator supercilii, the frontalis, the motores oculi, and the muscles of mastication and respiration.

The soft palate is rarely much affected. It may, however, hang rather lower down on the paralyzed than on the unaffected side, and move less. The uvula is inclined sometimes toward the healthy and sometimes toward the paralyzed side. There are no special disturbances of function as a result of these changes.

The trapezius is the only muscle of the trunk which is ordinarily much affected in cerebral hemiplegia. As a result of the paresis of this muscle, the shoulder sags and can not be raised as high as on the normal side. If the patient tries to take a very deep breath, it is in some cases possible to see that the paralyzed side lags behind a little in respiration, which is undoubtedly due to a paresis of the respiratory muscles on that side. It is perhaps due to this that the pulmonary diseases which attack hemiplegic patients frequently develop in the lung on the affected side, where respiration is deficient.

The most important element in the hemiplegia is the paralysis of the extremities. Immediately after the hæmorrhage it is often so complete that even the slightest voluntary motion in the affected arm and leg is impossible. Other cases, however, exhibit only a more or less severe paresis (hemiparesis) from the first; or the complete paralysis at any rate is confined to certain groups of muscles, the others still retaining vestiges of their normal contractility. Even when there is total hemiplegia at first some of the muscles usually regain a certain amount of their old power later on (*vide infra*).

The behavior of the reflexes is comparatively constant in nearly all cases. Almost invariably there is increased tendon reflex on the paralyzed side. If, however, the initial shock be very severe, there may at first be no tendon reflex whatever. In all cases of any duration it is always exaggerated, and often very much so. A tap upon the tendons and bones (periosteal reflex) of the arm, as well as of the leg, excites the most vigorous and manifold contractions. Very often a persistent ankle clonus can be obtained. It is noteworthy that even on the normal side, particularly in the lower extremity, it is almost always possible to demonstrate an exaggeration of the tendon reflex, although less pronounced than on the paralyzed side. Many have expressed the opinion that the increased tendon reflex upon the paralyzed side is a result of the secondary degeneration of the pyramidal tracts in the spinal cord. In our judgment this view is entirely unfounded, inasmuch as the exaggeration of the tendon reflex often appears within a few days, or even hours, after the apoplectic attack—that is, before a secondary degeneration in the spinal cord is to be thought of. We would rather seek to explain the phenomenon as being due to the suspension of certain reflex inhibitory influences because of the cerebral lesion.

There is very often found, particularly in cases of long standing affected with well-marked contractions, an increase of "direct mechanical excitability" in the paralyzed muscles, such that a tap upon them causes them to contract vigorously. We are, however, of opinion that, in part at least, this is also a reflex symptom, to be referred to the mechanical irritation of the fascia of the muscles (fascial reflex).

The skin reflexes in hemiplegia behave in precisely the opposite way from the tendon reflexes, being almost invariably decidedly diminished on the paralyzed side. In the paralyzed arm it is usually impossible to excite any skin reflex whatever; and in the corresponding leg there is either no, or at any rate a greatly diminished reflex. The difference is seen especially well in the abdominal and cremaster reflexes (*vide* page 543); on the paralyzed side they are almost or quite absent, while they remain normal upon the opposite side. This contrast often is of service in determining the location of the hemiplegia when the patient is in a stupor or coma.

Sensation is but little impaired in most cases of cerebral hemiplegia. It is usually possible, in a hemiplegia occasioned by this particular lesion (cerebral hæmorrhage), to find at first, by careful testing, a slight blunting of cutaneous sensibility; but this is seldom very great, and later on frequently becomes even slighter than at first. Slight paræsthesia is not infrequently complained of on the affected side, especially at first. Any marked disturbance of sensation indicates, as has been seen (compare page 721), that the posterior extremity of the internal capsule is involved. Such cases are rare. In them we may observe a complete cerebral hemianæsthesia combined with the hemiplegia. According to Gowers, a temporary hemiopia is often present directly after the occurrence of a cerebral hæmorrhage. Nor is it very exceptional to find a persistent hemiopia associated with hemiplegia; but as yet little is known about the pathological anatomy of such cases. The most likely thing would seem to be some lesion of the fibers of the optic nerve in the internal capsule or in the posterior tubercle of the optic thalamus. The muscular sense is not usually affected in hemiplegia. Lately it has been affirmed that paralysis of cortical origin invariably causes anomalies in the muscular sensibility of the parts which are paralyzed, but the statement lacks confirmation. Our own observations would certainly lead us to doubt whether it is universally true.

Turning now to the further course of hemiplegia, we find a new group of important symptoms. First of all should be considered the changes in the paralyzed muscles. If the hemiplegia be incomplete, even from the start, almost the normal degree of motility may in a comparatively brief period be regained by the affected side. At most there will persist a certain slight amount of weakness and stiffness; and even these will gradually diminish. From what has been already said, it is evident that in these cases the initial paresis is an indirect focal symptom, and accordingly vanishes as soon as the effusion ceases to affect the tissues not immediately involved.

But even where there is a complete hemiplegia it is exceptional for this condition to remain unabated throughout the entire region affected. After a few weeks, or even a few days, one part and another of the paralyzed half of the body begin to recover their former motility. The improvement goes on slowly, and in the most favorable cases the paralysis may for the most part have vanished at the end of some months. Usually, however, the improvement never becomes more than partial. It is noteworthy that the lower extremity almost always improves more than does the upper. Many patients gradually become able to walk alone once more, perhaps with the aid of a cane, although the arm remains almost useless. It must be confessed that the gait is seldom perfectly natural. The patient takes short steps, drags the affected limb more or less, and in many cases does not move it straight forward, but with an outward sweep. Very often the functions of the ileo-psoas and quadriceps return. The patient can then walk alone even if the other muscles remain paralyzed, because by these muscles the leg can be held firm like a wooden leg. In the arm, the chief improvement is usually in the fingers and the elbow-joint, the movements of the shoulder-joint recovering least.

The explanation of this improvement during the first few months is not fully ascertained. Probably it is largely true that only the persistent paralytic symptoms are direct focal symptoms, while the temporary motor disturbances are only an indirect result of the hæmorrhagic focus, and vanish when once the pressure, œdema, etc., have ceased. It is, however, not absolutely impossible that gradually new tracts (perhaps originating in the sound hemisphere *) assume vicariously some of the functions which are at first arrested. That fibers which have been destroyed are ever actually regenerated is very improbable; and, as has been mentioned already, there is not much improvement after the first six months.

The permanently paralyzed muscles become very often contracted later on. The appearances produced exhibit considerable uniformity. The arm, which suffers more from paralysis than the leg, is also usually more contracted. The fingers are almost invariably flexed; the forearm contracted in a position of pronation, and usually flexed rather than extended; and the upper arm adducted (the pectoralis major being chiefly affected). These contractures correspond to the natural positions which the paralyzed arm almost always assumes if left to itself. This, taken alone, is a reason for ascribing the contractures chiefly to the fact that the arm moves less than normal, and that, as a necessary sequel, certain muscles become permanently shortened. This view would make the changes "passive contractures." Another argument in its favor is that the deformity can to a certain extent be prevented by the persistent use of passive motion, which renders any permanent shortening of the muscle impossible. Nevertheless, Charcot and his pupils, including Bouchard, hold an entirely different opinion—namely, that the contractures are due to the secondary degeneration of the pyramidal tract. In defense of their position no fact can be adduced, save that fatal cases exhibiting hemiplegic contractures do invariably present the secondary degeneration mentioned; but of course this is no proof that the two things have any causative relation to each other. A contracture occurs only in connection with persistent paralysis; a persistent paralysis never is seen unless the pyramidal tract be destroyed; and if it be destroyed, a secondary degeneration must result. The contractures and the secondary degeneration are therefore two sequelæ, independent of each other. And that the degeneration should "irritate" the fibers, and thus excite muscular contractions, is extremely improbable; for, according to all analogy, fibers which are undergoing degeneration can not be stimulated, and can not therefore transmit any sort of stimulus to the paralyzed muscles.

If the contractures become marked in the lower limb, they may involve either the extensors or flexors. In the leg a moderate contracture of the calf is the most frequent condition. Hitzig has called attention to a remarkable fact, which is that often the contractures are very slight in the morning, when the patient first wakes up, becoming aggravated after he has moved about a little. Hitzig refers this phenomenon (which has not yet been at all satisfactorily studied) to abnormal "associated movements" of the paralyzed muscles. Such associated movements do occur: movements of the sound side excite associated movements in the opposite members; and efforts to move the paralyzed parts excite in their turn associated contractions of the normal muscles. Sometimes, also, involuntary movements of the lower limb on the paralyzed side are observed when the patient exerts himself to move his corresponding arm all he possibly can. Very often on drawing the affected leg up on the trunk we observe a marked dorsal extension of the foot as an associated movement.

Another peculiar phenomenon must be mentioned here. It is what Weir Mitchell has termed post-hemiplegic chorea. Some time after the paralysis begins

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* As has been already stated (page 731), it seems proper to assume that certain muscles enjoy a double innervation—that is, from each hemisphere. This applies particularly to such muscles as usually are exercised in pairs. We might perhaps in this way explain why the lower extremity is capable of more improvement after hemiplegia than is the arm. The legs have to be used simultaneously or consentaneously, as in walking, much more than the two arms. Another fact deserves brief mention: on careful examination, it is sometimes possible to make out a slight paresis in the sound side, which may perhaps be referable to lesion of the direct motor fibers (Pitres).

the parts affected by it exhibit involuntary movements, reminding one of chorea or athetosis (*vide* page 541). Sometimes these movements are continuous, sometimes they occur only as associated movements in connection with voluntary motions of the paralyzed, or even of the sound, side. Hemiplegia due to cerebral hæmorrhage very seldom exhibits this phenomenon. It is said to occur chiefly in focal disease of the posterior extremity of the internal capsule, and of the optic thalamus. It is much commoner in cerebral infantile paralysis (*vide infra*).

It is interesting to observe the trophic and vasomotor changes in the paralyzed parts. At first the skin may be somewhat redder and warmer on the paralyzed than on the sound side. Nothnagel has shown that, even in the distribution of the cervical sympathetic, symptoms of vasomotor paralysis occur. They are partly temporary and partly persistent, and comprise increase of temperature and color in the paralyzed side of the face, swelling of the eyelids, and contraction of the pupil; but they are usually slight. We very frequently find, especially on the back of the hands, more or less puffiness, which is likewise usually regarded as of vasomotor origin. It should, however, be considered that the natural movements of any part of the body greatly promote the nervous and lymphatic circulation, and that the quietude of paralysis may therefore have much to do with the œdema. In hemiplegia of some duration the extremities upon the paralyzed side are always cooler than normal, and the hand in particular is very often deeply cyanotic. The skin sometimes becomes harsh and fissured, and often is thickened. The internal surface of the hand, in case of contracture, is frequently quite damp with perspiration.

Among the specific trophic symptoms of hemiplegia Charcot includes "acute malignant decubitus." This sometimes develops with extreme rapidity within a few days after the shock, and usually occupies the buttock on the affected side. There appears a circumscribed redness with the formation of vesicles, which is soon succeeded by a deep-reaching necrosis of the soft parts. We have ourselves never met with this in a case which has been properly nursed, and we can not therefore help thinking that its development is not wholly due to trophic disturbance, but to pressure and to the penetration of septic matter below the skin. Of course, patients long confined to bed with hemiplegia are as liable to bedsores as are any others similarly situated.

The permanently paralyzed muscles gradually atrophy somewhat in the course of years; yet, in uncomplicated cases of cerebral hemiplegia, this atrophy is never of the degenerative sort, nor is it usually very great. The paralyzed muscles therefore continue to react perfectly to faradism (see page 547). The joints of the paralytic extremities, and in particular the knee and shoulder, may exceptionally become inflamed. The arthritis is sometimes acute, and sometimes more of a chronic variety. Its cause is not evident. Charcot thinks that it is probably a neurotrophic symptom, and he is of the same mind with regard to the rarely seen swellings of the peripheral nerve-trunks of the paralyzed side ("hypertrophic neuritis").

Mental symptoms are rare, except the initial loss of consciousness. There is sometimes, however, a persistent general uneasiness of mind, accompanied by great excitability and wakefulness. In a large number of cases of persistent hemiplegia there finally come on, in the course of years, constantly increasing indications of mental weakness. The patient grows dull and forgetful. Very often he exhibits a peculiar tendency to weeping, bursting into tears at the slightest provocation. But frequently he is subject to quick alternations of feeling, weeping and laughing in the same minute.

The general nutrition is often good for a long while. Sometimes there is even a decided tendency to corpulence. In other cases, and especially in the bedridden, marasmus gradually comes on and hastens the fatal termination, particularly if there be some intercurrent trouble, such as a bedsore or bronchitis.

We have detailed the peculiarities of hemiplegia somewhat minutely, because the statements will apply to all cases of cerebral hemiplegia, no matter in what place the pyramidal tract is interrupted or by what sort of a lesion. It is needless to enter into the diverse symptoms which are caused by diversity in the exact location of the hæmorrhage. The hemiplegia itself is the same, whether the effusion be in the cortex, internal capsule, crus cerebri, or pons. It it easy to infer from the preceding chapter what the accessory symptoms are which would enable us to localize the trouble. We need here mention only the frequent combination of right hemiplegia and aphasia. This occurs when there is a large effusion in the left hemisphere, extending from the internal capsule to the neighborhood of the third frontal, or possibly the uppermost temporal (compare page 716) convolution.

Diagnosis.—The diagnosis of cerebral hæmorrhage rests on the sudden onset of the apoplectic symptoms, and on the later symptoms (if there be any) of impairment of the cerebral functions. The diagnosis can scarcely ever be made with absolute certainty, for cerebral embolism may exhibit almost identical phenomena: the differential diagnosis between the two will be given in the following chapter. Occasionally there may be danger of mistaking other cerebral affections for a hæmorrhage, such as meningitis and tumors. The same may be said of a suddenly developed uræmia, and of constitutional sepsis. In these cases the rapid onset of grave cerebral symptoms of a general nature, such as unconsciousness, simulates the apoplectic coma.

Prognosis.—The first question is, whether the patient will survive the initial shock. The answer depends upon the severity of the early symptoms. The deeper and more persistent the unconsciousness, the more deficient the respiration and pulse, the less the prospect of recovery; but we can never decide absolutely. If the patient have withstood the first onset and be hemiplegic, then the possibility of improvement hinges on the question whether the paralysis is a direct or an indirect focal symptom. Inasmuch as there are no means of knowing about this at first, we must speak very guardedly. It should never be forgotten that the hæmorrhage may recur. The predisposing disease of the blood-vessels renders individuals who have had one stroke liable to be visited, sooner or later, by another.

Treatment.—The treatment of the apoplectic shock demands, first of all, rest in bed, with the head and shoulders elevated. To avoid bedsores it is very important to maintain cleanliness, and to watch attentively that portion of the skin which is pressed against the bed by the weight of the body. An ice-bag should be put upon the head, and particularly over that side on which the hæmorrhage is supposed to be. Bleeding was formerly universally practiced, but of late its usefulness is doubted. It is not indicated unless the deep congestion of the face, the violent pulsation of the carotids, and the full, slow pulse show increased arterial tension. In such a case, if the patient seem otherwise robust, we may bleed at the commencement of an attack, in the hope of checking the flow of blood by lowering the intra-arterial pressure. In similar conditions experience shows that the local abstraction of blood from the temples is sometimes advantageous. The bowels should be well emptied by enemata, and later on by drastic purgatives. If the respiration and pulse fail, we may try stimulants (ether, camphor), but very likely they will be without success.

[Horsley and Spencer have found, by experiments on animals, that if the internal carotid be tied the bleeding from the divided lenticulo-striate artery is checked. Horsley therefore suggests that in the very earliest stage of cerebral hæmorrhage ligature or compression of the internal carotid may check the bleeding, and prevent the formation of a large clot and extensive destruction of the brain. This procedure has not yet been tried upon the human subject.

Where there is much difficulty in respiration during the period of coma, relief may sometimes be obtained by turning the patient upon the side.—K.]

When the patient is safely over the shock, our resources for aiding him in the further stages of his trouble are very limited. As long as headache and symptoms of fever persist, the application of ice to the head should be kept up, and other disturbances should be treated symptomatically. For uneasiness and wakefulness, small doses of morphine or chloral are given. Treatment of the hemiplegia must be deferred for the first three or four weeks, until all the initiatory symptoms of irritation are over. Then electricity plays the chief *rôle*. Local galvanization should be tried, the current being made to pass transversely through the head, with as much regard as possible to the position of the hæmorrhagic focus; the current should be feeble, and the application should occupy two or three minutes. With this may be combined galvanization of the sympathetic nerve on the side of the hæmorrhage. Nor should galvanization (stroking with the cathode) and faradization of the paralyzed muscles and nerves be neglected. If favorable changes take place, it is, however, uncertain how much should be ascribed to treatment; since, as has already been stated, there is often improvement without treatment.

Passive movements and massage of the paralyzed limbs are very important as a prophylaxis against contractures. They should be commenced promptly, and be methodically continued. Massage, and later on systematic and appropriate gymnastic exercises, may contribute much to the restoration of voluntary motions. The same object is also promoted by rubbing with spirits of camphor, chloroform liniment, etc.

Internally, potassic iodide is frequently given, out of regard for its "absorbent" properties. We may also try the effect of strychnine; it is most adapted for cases of some duration.

As to baths, they should not be too warm—that is, not over 90°–93° (26°–27° R.). Moderately warm baths, medicated with common salt if it seems desirable, and employed three or four times a week, seem to be beneficial. If it be thought best to send the patient to a regular bathing-place, Wildbad, Ragatz, Teplitz, Wiesbaden, or Rehme may be chosen. At the first-mentioned places none but the cooler springs should be used.

Hemiplegic paralysis often lasts so long that the physician must repeatedly change the details of treatment, so as to support the courage and patience of the sufferer. Particular care should be given to regimen, in order that any recurrence of the hæmorrhage may, if possible, be avoided. The diet should be simple; any large amount of alcohol should be forbidden; and there should be no severe bodily exertion or mental excitement.

CHAPTER IV.

CEREBRAL EMBOLISM AND THROMBOSIS.

(*Softening of the Brain from Embolism or Thrombosis.*)

Ætiology and Pathology.—Occlusion of the cerebral arteries is one of the most frequent injuries inflicted by embolism. Usually the emboli originate in the left side of the heart, from thrombi in the left auricle, or from the thrombotic deposits which form in chronic endocarditis (mitral or aortic disease) upon the valves of the left ventricle. Chronic arterio-sclerosis may also lead to thrombosis in the larger arteries, particularly the aorta; and in case the cerebral arteries themselves are extensively atheromatous, the larger vessels at the base of the brain may furnish material for embolism of the smaller cerebral arteries.

Thrombosis of the arteries of the brain is always due to primary disease of the blood-vessels, the most common cause being the chronic arterio-sclerosis just mentioned. Wherever the atheroma has altered the normal structure of the intima, deposits of fibrine may take place. Their development is further promoted by the subnormal elasticity of the arteries, and by the occasional narrowing of their lumen; for thus the flow of blood is rendered slow, if not even completely checked. It is easy to understand that thrombosis and embolism may each give rise to the other. From every thrombus an embolus may be detached; and every firmly lodged embolus may form a nucleus for thrombosis.

Next to arterio-sclerosis, the most frequent cause of a cerebral thrombus is syphilitic endarteritis. In the chapter on cerebral syphilis this subject will be minutely considered. Whether thrombi ever form here independently of disease of the vessels is doubtful. An apparently spontaneous thrombosis is now and then seen in patients who are cachectic or severely ill (cancer, grave typhoid or typhus fever, pneumonia); in such instances the cardiac weakness, and possibly also an abnormal tendency of the blood to coagulate, are regarded as either the causes, or at least the predisposing factors, of the thrombosis.

In whatever part of the arterial system complete occlusion has been produced by an embolus or thrombus, the results depend upon the possibility or impossibility of blood reaching by collateral channels the region thus deprived of its ordinary supply. If the collateral circulation prove efficient, no harm is done; if not, the tissues must perish and undergo "softening." It is thus a matter of the greatest practical import that the perforating arteries of the brain-stem, and particularly the branches of the middle cerebral artery in the fissure of Sylvius, which supply the great central ganglia and internal capsule, are all "terminal," in Cohnheim's sense—that is, they form few anastomoses with neighboring vessels. Now, the middle cerebral artery and its branches are known from experience to be peculiarly liable to embolism, above other cerebral arteries. Hence we see why the region they supply suffers so severely and so very frequently from emboli. It is noteworthy that the left middle cerebral artery is more frequently plugged by emboli than is the right. In the centrum ovale and cortex there is more opportunity for collateral compensation than in the central ganglia; but even here the supply of blood often proves insufficient, as is shown by the not infrequent occurrence of spots of softening in the cortex and the white substance of the cerebrum. On the other hand, embolic foci are much rarer in the crura cerebri, pons, and cerebellum.

The various steps in the process which begins with embolic or thrombotic occlusion and ends in softening of the brain-substance are essentially the same as occur in other organs (compare chapter on pulmonary embolism, page 247). The tissues which are deprived of their arterial blood perish, become disintegrated, and are transformed into a soft homogeneous mass. Into the empty portion of the artery, beyond the embolus, blood flows in the reversed direction from the veins, and, if the anatomical relations permit, from the minute arteries in the neighborhood; but the supply is not sufficient to nourish the tissues. The walls of the blood-vessels become abnormally permeable and delicate, so that some red globules invade the disintegrating region by diapedesis, and others in the way of minute but genuine ecchymoses. Actual infarctions are, however, never formed in the brain, perhaps because, as Weigert suggests, the nervous structures swell so much as to exclude any large amount of blood; but the little punctiform ecchymoses are in many instances so abundant that the whole softened spot seems distinctly reddish or yellowish. This red or yellow softening is also due in part to the tissues being stained by the dissolved pigment of disintegrated blood-globules. If the discoloration be not very striking, then we speak of a white softening.

Fresh foci of softening are seen through the microscope to be composed of drops of myeline, bits of swollen nerve-fibers, numerous fatty granular cells, and free fat globules. The minimum time required for these changes is one or two days. If within that period a compensatory collateral circulation is set up, the nervous structures may be restored and resume their functional activity. If not, the tissues perish and become disintegrated. The white blood-corpuscles and leucocytes (and possibly also the endothelial cells of the blood-vessels, and the neuroglia and ganglion-cells) absorb the fatty detritus thus made, and come to form the fatty granular cells above mentioned. If the patient live, the dead and disintegrated tissue is gradually absorbed, and it may even finally be replaced by a cyst not different in appearance from those which occur after cerebral hæmorrhage. A minute focus of softening may sometimes also be replaced by indurated cicatricial tissue. If portions of the surface of the brain become softened, quite a deep depression often results. This is filled up in part with serous fluid and in part by hyperplasia of the pia mater. Sometimes the convolutions are still recognizable in places; but they are atrophied, of a yellowish color, and of a greatly increased consistence, due to the growth of cicatricial tissue.

Clinical History.—The occurrence of cerebral embolism is attended with almost precisely the same sort of shock as is cerebral hæmorrhage. We do not need to enter into the particulars again here, but will refer to the preceding chapter (*vide* page 727). In embolism, also, the intensity of the shock varies; it ranges between extreme mildness, when it occasions only transitory confusion of intellect or slight vertigo, to the greatest severity, when there is deep and persistent coma. One chief factor in determining the nature of the case is the size of the occluded artery; another is its position, according as the embolism has taken place in the hemispheres or toward the base of the brain. In general, the shock of embolism is not often so severe and long continued as that of hæmorrhage; and embolism does not so frequently give rise to symptoms of cerebral compression, including slowing of the pulse. On the other hand, epileptiform convulsions are seen more often in embolism than in hæmorrhage. Embolism again may have a rather slow onset, where there is at first a small embolus and this becomes the nucleus of a gradually formed thrombus.

It is not easy to explain why there should be a shock at all in case of embolism. Perhaps the main reason is the diminution of pressure which the embolism occasions in the region directly affected and in its neighborhood. The portion of the artery beyond the plug becoming empty, not only causes a draught upon the blood and lymph, but subjects all the soft surrounding structures to a diminution of tension and to a certain amount of strain (Wernicke). It is not impossible, however, that the disturbance of circulation occasioned in the surrounding blood-vessels by sudden embolism of a good-sized artery is of itself enough to account for the symptoms of shock.

With regard, also, to the persistent symptoms of embolism we may be equally brief, inasmuch as they very closely resemble those which follow a cerebral hæmorrhage. As has been said, it is only when a compensatory collateral circulation is developed within the first forty-eight hours that the early symptoms of focal disturbance can entirely vanish. After this period, the tissues which have been deprived of their normal blood-supply must undergo necrosis; although there is still room for hope that some of the symptoms will prove indirect, and therefore capable of improvement, so that embolic hemiplegia, like that from hæmorrhage, may improve decidedly in the course of the first few weeks.

Inasmuch as the middle cerebral artery is by far the most frequent seat of cerebral embolism, and inasmuch as this artery supplies the internal capsule as well as the great central ganglia, the most frequent focal symptom of embolism of the brain is ordinary cerebral hemiplegia, all the features of which have been depicted in the preceding chapter. Aphasia is associated with it with comparative frequency, for, as already mentioned, the left middle cerebral artery is especially apt to suffer. Less frequent is paralysis of a single member, due to cortical embolism, or an occipital lesion with hemiopia.

Where softening is the result of thrombosis, the symptoms are but seldom abrupt in their onset. Usually the focal symptoms and the more general ones (such as unconsciousness) are developed rather gradually. This is most often seen in so-called senile softening of the brain, a disorder almost always caused by sclerosis of the cerebral arteries. The various symptoms generally come on under cover of repeated relapses and fresh aggravations of the disease. A severe initial shock seldom occurs; but almost invariably there is a gradual and progressive dementia.

The further course and the final termination of cerebral softening vary as do those of cerebral hæmorrhage. Embolism of a larger artery may cause speedy death. If, however, the first shock passes away, the impairment of function which may be left behind may last for years without seriously affecting the general health. There is always danger of a recurrence when the source of the embolism continues unchanged, as in cardiac disease or atheroma.

Diagnosis.—Both the onset and the persistent focal symptoms are so much alike in hæmorrhage and embolism that in many cases it is utterly impossible to decide which caused the apoplexy and hemiplegia. The following are factors in making a differential diagnosis, if one can be made: 1. It is very important whether there be any source for an embolus. Thus, if the patient have valvular cardiac disease (particularly mitral), embolism is more probable than hæmorrhage. 2. A young person is, on the whole, more apt to have embolism than hæmorrhage. At a later period of life one is about as probable as the other. 3. When the shock is severe and persistent, with red face, strong pulsation of the carotids, and signs of cerebral compression (slow pulse), we would think of hæmorrhage rather than embolism, in which latter the initial symptoms are sometimes comparatively slight (*vide supra*). 4. Finally, it is sometimes possible to obtain support for a diagnosis of cerebral embolism by demonstrating embolism elsewhere, as in the fundus oculi, by means of the ophthalmoscope.

In rare instances, also, tumors of the brain, into the substance of which hæmorrhage takes place, may induce symptoms closely simulating a primary apoplectic shock, as may also abscesses which have been previously latent, and then suddenly burst into a ventricle. In such cases a correct diagnosis is seldom possible.

Softening due to a thrombus is the most readily diagnosticated in those cases where there is cerebral syphilis (*q. v.*). Senile softening is inferred from the age of the patient, the signs of general arterio-sclerosis, the abrupt advances of the disease from mildness toward severity, and the developing dementia.

For the prognosis and treatment of cerebral embolism we may refer simply to what was said in the preceding chapter.

CHAPTER V.

INFLAMMATION OF THE BRAIN.

(Acute and Chronic Encephalitis.)

1. ABSCESS OF THE BRAIN (SUPPURATIVE ENCEPHALITIS).

Ætiology.—In most cases of cerebral abscess it is possible to demonstrate with certainty that infectious material capable of exciting suppuration has penetrated to the encephalon. This is particularly true of those not very rare abscesses which follow injuries of the scalp, cranium, or brain (traumatic abscess of the brain). Here the wound is almost always an open one, affording free ingress to the virus. It is not essential that the bones should be injured, for experience shows that, even where the soft parts alone are wounded, suppuration may extend to the brain. The manner in which this extension takes place determines the question whether a suppurative meningitis (*q. v.*) or an abscess shall be developed. Not infrequently we find the two combined. Another source of traumatic cerebral abscess is foreign bodies which penetrate into the brain (for instance, through the orbit), and thus carry septic matter into the very substance of the organ. Some exceptional cases have been reported of traumatic cerebral abscess without any open wound; as yet they are beyond our comprehension. Possibly there is even in these some minute wound invariably present but overlooked.

Analogous to the above causes is suppuration in neighboring parts, which may, by direct extension, occasion cerebral abscess. The same processes are prominent in this connection which we have already found to excite purulent meningitis (*vide* page 698)—particularly suppuration (caries) in the middle ear and in the petrous bone. In such a case the most frequent position of the abscess is naturally the temporal lobe or the cerebellum. Abscesses also occur in the anterior part of the brain, from suppurative processes in the nasal cavity and the ethmoid bone; but this mode of origin is much less frequent.

In yet a third class of cases the morbific agents are conveyed from foci of disease situated in distant parts of the body. These are metastatic or embolic abscesses. They occur, for instance, in pyæmia and ulcerative endocarditis. Abscesses of this sort, however, are usually small, and seldom are conspicuous in modifying the grave general disease. Of more importance are cerebral abscesses connected with certain suppurative affections of the lungs and pleura. These are not so very rare. They are oftenest associated with fetid bronchitis, pulmonary gangrene, and empyema. Purulent meningitis (*q. v.*) may occur in the same way. There can be no doubt that virus is in some way conveyed to the brain, but just how is not yet known.

In some few cases of cerebral abscess no certain ætiology can be made out. To these the term idiopathic is applied. We met with several of them just at the time of an epidemic of cerebro-spinal meningitis; and it therefore seems reasonable to suspect that possibly many apparently spontaneous cerebral abscesses are referable to the same poison as is epidemic meningitis.

Pathology.—The pathological anatomy of abscess of the brain is precisely the same as that of abscesses in other organs. The size varies from minute foci hardly as large as a lentil to great cavities occupying nearly the whole of one lobe. Not infrequently several abscesses appear simultanously in different parts of the brain. The pus is usually greenish yellow. It may be odorless or offensive. Sometimes remnants of the destroyed ("melted") nervous tissue and red blood-globules are mixed with it. The walls of the abscess often bulge out irregularly. The cerebral parenchyma around the abscess for a greater or less distance exhibits white softening. This is due partly to the pressure and partly to the spread of the inflammation. An abundance of granular cells is generally to be found in the tissues near the abscess.

If the abscess be very large and reach nearly to the surface of the brain, it may sometimes be recognized during life by a distinct bulging and fluctuation. The convolutions on the surface of the affected hemisphere are almost always flattened. If the abscess extend clear to the surface of the brain, purulent meningitis is excited. Abscesses which are centrally situated not infrequently burst into a lateral ventricle. An abscess of long standing may finally become encapsulated—that is, become incased in a smooth, firm layer of connective tissue, which prevents further extension of the process. The pus inside gradually becomes thick and cheesy. Apparently, however, it is very seldom entirely absorbed.

Clinical History.—Small abscesses, and even large ones, may for a long while have scarcely any symptoms. This is particularly true of idiopathic abscesses, and of those which develop very slowly and insidiously after apparently insignificant injuries of the head or in connection with chronic inflammation of the middle ear.

In cases following more extensive injuries, the symptoms are more violent from the start; which is also true of many abscesses which develop acutely and enlarge rapidly. The symptoms can hardly be distinguished from acute meningitis. The patient is dull, or grows delirious; he has violent headache and fever. Sometimes the temperature repeatedly rises to a high point. The loss of consciousness becomes more and more complete; and in a comparatively brief time (one or two weeks) there may be profound coma and death. Rarely the violent symptoms abate, and the acute passes into a chronic stage.

The symptoms of cerebral abscess, when it pursues a chronic course, may be divided into (1) the general symptoms and (2) the focal symptoms, resulting from the special position of the abscess. There is no other localized cerebral disorder in which the focal symptoms are so often absent, either for a long while or even throughout the illness. This is partly because the abscesses are often situated in parts of the brain, injury to which does not occasion any demonstrable focal symptoms. Such localities are the white matter of the frontal lobe, and the hemispheres of the cerebellum. A second reason is that an abscess seldom excites indirect focal symptoms by affecting the parts around it.

Among the general symptoms, the most important is a persistent, deeply situated, dull headache. For a long while it may be the only symptom, especially when the abscess is gradually developed after an injury to the head or after aural disease of long standing. The pain is referred mainly to the seat of the abscess; but the exceptions to this rule are not infrequent. Another frequent symptom is vertigo; and in many cases there is vomiting, either after meals or entirely independently of food. Another valuable symptom in diagnosis is the irregular fever, sometimes slight, and sometimes exhibiting great elevations with intervals between them; but in many cases also, particularly where the abscess is encapsulated, there may be no fever whatever. An important negative point is that choked disk is much rarer in cerebral abscess than in case of tumors (*q. v.*).

The general health may be but slightly disturbed. Usually, however, there is decided indisposition. The patient is pale, has no appetite, and loses flesh.

As to the focal symptoms, little need here be added to what is contained in Chapter II of this section. Abscesses involving the motor region of the cortex have been repeatedly found to cause limited epileptiform attacks and paralysis of some one limb. It is particularly characteristic that, as the abscess grows, one symptom of paralysis is added to another, and that often the advance of the paralysis is ushered in by epileptiform convulsions. Abscesses in the occipital lobe have repeatedly been observed to cause hemiopia, and abscesses in the temporal lobe word deafness; and these facts have been used in making a diagnosis of the location of abscesses. Abscesses in the cerebellum may remain unsuspected for a long while; or the above-mentioned general symptoms may occur in especial severity in such cases.

The duration of chronic cerebral abscess varies greatly. In most cases it lasts for months; and sometimes it has certainly lasted for years. Particularly where the abscess gives rise to no symptoms, or merely to slight and indefinite ones referable to the head, it may last a very long while. It is quite often the case that there are separate attacks of the severe symptoms, such as headache, vomiting, and fever, and that in the intervals of variable duration between these paroxysms the patient feels pretty well.

The final termination of cerebral abscess is almost always fatal. Recovery is not impossible, but it has been seen with certainty in only a very few instances. Death either comes on gradually, where, as the abscess grows larger, all the symptoms become correspondingly aggravated; or it may happen quite suddenly. Sometimes it is brought about by the abscess bursting into a lateral ventricle, or by the supervention of meningitis. Often a cerebral abscess terminates in a sudden and unexpected death, where no immediate cause for the event can be found.

Diagnosis.—Although it is often possible to make a correct diagnosis of cerebral abscess, yet there is usually a good deal of difficulty in arriving at such a conclusion, and entire certainty is seldom attainable. The most important points in diagnosis are: (1) The demonstration of some cause such as trauma, chronic otitis, fetid pulmonary diseases, or empyema. (2) The presence of general cerebral symptoms, such as headache, vertigo, and vomiting; and the fact that these are sometimes better and sometimes worse. To aid in excluding tumor, we have (3) the febrile symptoms frequently caused by abscess, but rare in case of tumor; and (4) the extreme rarity of choked disk, which is very frequently occasioned by tumors. The focal symptoms, if there be any, are not characteristic. They grow worse by fits and starts in tumor just as in abscess. One fact, however, is of value, namely, that while tumors (*vide infra*) frequently cause disturbances in the area of distribution of the nerves at the base of the brain (such as paralysis of the motor oculi), an abscess does this only exceptionally. It is often quite impossible to make a differential diagnosis between acute abscess and purulent meningitis, unless focal symptoms are developed. These can be caused by a circumscribed lesion alone, and therefore point to abscess.

Treatment.—The only possible way in which to cure an abscess is by operation; the skull is trephined and the abscess laid open. It can be readily seen that such a procedure is feasible in but few instances. We need to be certain that there is an abscess and where it is located, and, finally, that it is so located as to render operation permissible. Under the present antiseptic modes of procedure, the dangers of an operation need not be over-estimated. For particulars we must refer to works on surgery.

If operation be not justifiable, nothing but purely symptomatic treatment is left us. Ice to the head, narcotics, potassic bromide, electricity, and sometimes also the local abstraction of blood, are the main remedies beyond general hygienic measures.

2. Acute and Chronic Non-suppurative Encephalitis.

While the spinal cord is quite frequently affected by idiopathic circumscribed inflammation (transverse myelitis), analogous cerebral disease is much more exceptional. The scanty information which we possess upon the subject is as follows:

1. **Idiopathic (Inflammatory) Softening of the Brain.**—In rare instances there are found in the brain quite extensive foci of softening, the pathological anatomy of which is almost identical with that of embolic softening, and yet the afferent blood-vessels do not furnish any explanation of the condition. They have accordingly been termed "foci of inflammatory softening." The mode of their production is unknown. The symptoms resemble closely those of softening from thrombus.

2. **Curable Form of Encephalitis.**—In certain cases pronounced symptoms of focal disease exist for a time, and suggest a tumor or the like; but after some months, or even a still longer time, the symptoms gradually abate, and finally there is complete recovery. Such cases undoubtedly occur. They are usually explained by assuming that there has been a localized inflammation, followed by a restoration to the normal state. The nature of the symptoms, as we have observed them, would seem to imply that the lesion is generally in the neighborhood of the cortex, for there is usually paresis of some one part of the body, often associated with certain symptoms of irritation and impairment of speech. Possibly the recovery of these cases may be assisted by electricity and potassic iodide; but we should never venture upon an absolutely favorable prognosis.

3. **Diffuse Cerebral Sclerosis.**—Diffuse sclerosis of the brain is a peculiar disease, which is usually classed as a chronic inflammation. The whole brain may be involved, or the disease may be confined chiefly to a large part of one hemisphere. There is a very marked increase in the consistency of the brain-substance, so that it cuts like a tough piece of leather. The microscope reveals in many cases, but not in all, diffuse hyperplasia of the neuroglia. In a case which we recently examined there was a decided atrophy of the nervous fibers lying in the white substance of the brain. The disease is rare, and its characteristic symptoms can not yet be stated absolutely. They are developed slowly. The most constant among them seem to be hemiplegia without much change in sensation; symptoms of motor irritation, such as epileptiform convulsions (sometimes general and sometimes unilateral), or single twitchings, which may be rhythmical or like those of chorea; and, lastly, dementia. Where both hemispheres are involved, there are usually marked spastic symptoms in the lower limbs.

The disease has been observed in children and in elderly people. Possibly chronic alcoholism sometimes acts as an ætiological factor. Treatment can be only symptomatic.

Multiple sclerosis of the brain is almost always associated with the same affection of the spinal cord. We have, therefore, already described the disease in the preceding section (page 627).

4. **The Acute Encephalitis of Children.** (*Cerebral Paralysis of Children. Spastic Infantile Hemiplegia of Benedikt.*)—Children not infrequently suffer from a definite form of hemiplegia, which deserves a brief special description.

The patient is usually between one and four years old. The commencement of the symptoms is almost always acute. Having been previously healthy, the child is suddenly attacked with malaise and fever. Very often there is nausea and vomiting, speedily or at once followed by grave cerebral symptoms. There is stupor, and convulsions are particularly frequent. This condition may last but one or two days; or it may continue, perhaps with unabated severity, for two or three weeks. Then the acute symptoms abate, and the child recovers comparatively fast; but it is noticed by the parents to be paralyzed; and this paralysis seldom entirely disappears, although it may diminish.

If such children come under observation after the paralysis has lasted some time, as is usually the case, their condition is generally as follows: The cranial nerves, as a rule, are but little affected. The main change is in the extremities of one side; and the arm is almost always worse than the leg. The affected parts betray an arrest of development, more or less impairment of motion, in many cases a marked exaggeration of the tendon reflexes, and almost invariably contractures of more or less severity. The muscles, although generally somewhat atrophied, never exhibit the reaction of degeneration. Sensation, as a rule, is unimpaired. Motor symptoms of irritation are found upon the affected side with striking frequency; the commonest are movements like athetosis or chorea (hemi-athetosis, hemichorea); and associated movements are also not infrequent. The constant movements of the fingers resulting from the athetosis sometimes render the articulations so lax that it is possible to make the fingers assume an angle of ninety degrees or even less with the back of the hand, at the metacarpo-phalangeal joint. The child may keep making motions with its paretic arm while walking. It is not very rare for epilepsy to be developed later. There are convulsive attacks, which usually begin on the paralyzed side, but which may later affect the whole body. The intellectual development of many such children is tolerably normal. Others, however, are more or less demented, or betray defective moral sense.

The whole course of the disease suggests very strongly an acute encephalitis. The process is probably in most cases more or less completely limited to the motor region of the cortex, and has hence been called "poliencephalitis."* It reminds one forcibly of the acute poliomyelitis of children, with a difference merely in the localization of the disturbance. It is not impossible that the two diseases have a very similar, if not identical, ætiology. They can hardly be differentiated in their initial stage; but when further developed they can not be confounded, because the cerebral disease causes unilateral paralysis, leaves the electrical reaction unimpaired, and frequently causes exaggeration of the tendon reflex. It deserves mention that precisely similar phenomena may be presented by children during recovery from measles, scarlet fever, and other acute infectious diseases.

The pathological conditions of the early stage have not yet been studied. Long after the process has run its course, the affected portion of the cerebrum presents marked atrophy with cicatricial contraction. If the surface of the brain has been affected, a corresponding depression is to be noticed ("porencephalia"). At these places the pia is thickened. Sometimes limited cystic formations are found. The pyramidal tract exhibits a secondary descending degeneration. It is thus evident that the process is, from an anatomical point of view, precisely like the atrophy of the anterior cornua occasioned by poliomyelitis.

Of course not all the hemiplegias arising in childhood are due to an encephalitis, for in not a few cases infantile hemiplegia may be due to embolic softening, and perhaps sometimes to hæmorrhage; but in most cases acute encephalitis is distinguished from these by the peculiarity of its initial stage. [There is really no pathological evidence of acute polioencephalitis in children. Sachs and Osler, from the study of a large number of cases, find that the cause of the paralysis is almost always hæmorrhage or embolic softening.—K.]

The treatment at first is to be governed by the same rules as in the initial period of acute poliomyelitis (*q. v*). The hemiplegic symptoms which persist after the first few months will never improve much. The most we can do will be by means of electricity, massage, and cold baths with friction ("cold rubbing").

[* This name, however, has been previously applied by Wernicke to that form of external ophthalmoplegia due to changes in the nerve nuclei in the pons (*vide* page 690).—TRANS.]

For the epileptic attacks large doses of bromide of potassium are decidedly beneficial.

Only a very few cases of primary acute non-purulent encephalitis of adults are recorded. A short time ago we saw such a case in a young man. Death occurred after a few days' illness marked by very severe cerebral symptoms and at the autopsy one cerebral hemisphere was found studded with many little hæmorrhagic-encephalitic foci.

CHAPTER VI.

INSOLATION. SUNSTROKE. HEAT PROSTRATION. THERMIC FEVER.

[**Ætiology and Pathology.**—Under exposure to undue heat, either in the direct rays of the sun, or, during hot weather, in engine-rooms, laundries, and the like, marked effects may be produced on the human organism, manifesting themselves in a widely different manner in different cases. The liability to these effects is much greater with us than in most portions of Europe or in Great Britain, and is enhanced by a moist atmosphere which tends to prevent evaporation from the surface of the body. Attacks may come on at night and under cover as well as by day. While excessive heat is the sole and sufficient exciting cause of the changes and symptoms to be described, exhaustion due to muscular exertion or other cause plays a very important secondary *rôle.* A vigorous person, however, of regular and temperate habits, can stand much greater heat and exertion than an individual who is debilitated or addicted to the use of stimulants. The frequency of attacks after a full meal has been noted. Those newly arrived in the country are, other things being equal, more likely to succumb than natives or those who have become accustomed to the climate. That high temperature due to solar or artificial heat, or a combination of the two, is the prime causative condition, has been clearly shown by experiments on animals. There is practically nothing to add to the researches of H. C. Wood.

Pathological Anatomy.—In cases of sudden death from shock there are no constant and peculiar lesions. The blood is dark, imperfectly coagulated, and collected in the veins; ecchymoses are frequently found.

After death, due chiefly to abnormally high temperature—that is to say, in cases of thermic fever—the heart, and especially the left ventricle, is firmly contracted from post-mortem coagulation of its myosin; the lungs are apt to be much engorged with dark fluid blood, and may be the seat of hæmorrhage; extravasation of blood under the skin is more or less marked, and hæmorrhage into and about the cervical sympathetic ganglia has also been observed. The membranes of the brain and cord are often greatly congested, and there may be evidences of commencing meningitis. The blood-corpuscles are crenated and show a diminished tendency to the formation of *rouleaux.* Rigor mortis is marked and early in the voluntary muscles as well as in the heart, and is attributable to the same cause. Parenchymatous degeneration of the organs is sometimes found.

Symptoms and Course.—The onset of the attack is usually sudden, though there may be slight premonitions, such as dizziness, pain, or uncomfortable sensations in the head.

It will, perhaps, add to clearness to distinguish three leading forms of attack, it being understood that Nature does not always classify cases as sharply as is done here. The first of these comprises cases of heat prostration, denoted by faintness, syncope, nausea, and sometimes vomiting, with marked muscular and general weakness. The surface of the body is cool, the pulse rapid and feeble. The great majority of these cases are mild, and the symptoms pass away more or less quickly on placing the patient in the recumbent posture in a relatively cool and quiet place with free ventilation. After a few hours the patient can be removed to his home, and in a day or two has recovered perfectly, except for some sensitiveness to heat or the sun's rays. There may be transient insensibility, or, on the other hand, the case may terminate fatally very soon from general exhaustion and cardiac failure.

The second form includes cases with respiratory as well as circulatory failure, due especially to the exhaustion of the nerve centers presiding over these functions. Under this head come cases of true sunstroke—patients suddenly losing consciousness while exposed to the sun. The skin is cold and the pulse is feeble; death may quickly ensue or recovery may follow, especially if prompt and suitable treatment be instituted. The after-effects of the attack may be very slow to pass away, and may never disappear entirely.

The most striking characteristic of the third form is the great increase in the temperature—a symptom which has given rise to the term thermic fever. In this form premonitions are more common than in the others. The thermometer may register 110° or even more; the skin is burning hot and generally dry; the pulse is slow and full, or quick and jerking; the respiration is quickened, sighing, or even stertorous; the pupils at first are usually contracted; there may be great restlessness; coma and convulsions sometimes occur; vomiting is common; and, toward the close of life, the sphincters are sometimes relaxed. A fatal result is due to apnœa and asthenia combined. What was said with regard to recovery from the second form holds good also for the third.

Diagnosis.—This is seldom difficult. The circumstances under which the attack comes on are generally patent; and the hyperpyrexia, if present, is distinctive. Acute alcoholism and meningitis are the chief affections which might lead to error.

Prognosis.—The mortality rate of the severer forms of the disease is very high, but the prognosis depends much on the possibilities of securing prompt and skillful treatment. Under this many a case recovers from a seemingly desperate condition. The tediousness and imperfection of recovery have been already alluded to. For long periods in some cases the mental and physical powers are much impaired, and great care has to be exercised as regards exertion, high temperature, and the sun's rays. Insanity is sometimes a sequel.

Treatment.—In the first place, much may be done by preventive measures to obviate the necessity for any treatment. A regular and temperate life will do much; and special precautions of an obvious nature should be taken during hot weather by those whose occupations involve a liability to exhaustion and exposure to unusual heat. It would be well if it were generally known that there is comparatively little danger of sunstroke so long as perspiration is free. Many an attack might be averted by noting the activity of the skin and seeking rest and shelter as sweating diminishes. A considerable responsibility rests upon militia surgeons and others in similar positions during hot weather.

Mild cases of heat prostration seldom require other measures than those already indicated in describing the symptoms. If there be nausea, and it seem desirable to give stimulants, they are better given under the skin or by the rectum. The clothing should be loosened; the cold douche and other refrigerating measures are not indicated unless there is fever, and they should then be used with caution.

For true sunstroke, treatment should be active and energetic, the indications being to reduce the temperature of the overheated centers and to stimulate their activity. If a cool or shady spot be near at hand, the patient should be removed to it and largely stripped of clothing; if not, no time should be lost before resorting to the cold douche on the head and body while stimulants are administered by enema or subcutaneously. External stimulation by mustard or flagellation, and purgative enemata, are said to be sometimes useful. The use of cold externally should not be prolonged in these cases unless there is fever. Nervous exhaustion is the prominent symptom, and all depressing measures are out of place.

The case is widely different with the third form of the attack. Here the immediate danger is from the hyperpyrexia, which must be combated by rubbing the patient with ice, placing him in a tub of water with lumps of ice, or similar measures, until the temperature in the rectum is reduced nearly but not quite to the normal point. In the application of the refrigerating measures the head must not be neglected. The sole indication at first is the reduction of the temperature. Antipyrine subcutaneously has been given in a few cases in the Boston City Hospital, and also in Brooklyn, with good results. After a reduction of the temperature, any symptoms of collapse or exhaustion demand stimulants.

With regard to the employment of bloodletting there is considerable difference of opinion. That cases occur in which this procedure is indicated is undoubtedly true, but they are exceptional; they are characterized by the evidences of great cerebral congestion without hyperpyrexia.

The subsequent management of convalescents from any form of sunstroke is often very important. Prolonged rest, and sometimes change of climate, may be demanded. A symptomatic and common-sense treatment is in place. It has seemed to the editor that quinine, especially in solution with a moderate excess of sulphuric acid, is distinctly useful in those suffering from mild or moderate after-effects of undue heat.]

CHAPTER VII.

TUMORS OF THE BRAIN.

Ætiology.—The precise causes which lead to the development of tumors in the brain are no more certainly known than in regard to other organs. In most cases the new growths are formed insidiously and gradually in persons previously healthy, without ascertainable cause. A circumstance which deserves mention is that sometimes the first symptoms come on immediately or a short time after some injury to the head; but whether this is a matter of cause and effect, or of coincidence, can very seldom be determined.

The sufferer from cerebral tumor is usually in middle life. Certain forms of tumor, however, particularly solitary tubercles, are comparatively frequent in children. Sex seems to exert a decided influence: men are much oftener attacked than women.

Varieties of Cerebral Tumor.*—The most important forms of tumor seen in the brain are as follows:

1. Glioma.—Glioma is a kind of tumor peculiar to the central nervous system, but seen much oftener in the brain than in the spinal cord (*vide* page 676). Apparently the new growth always originates in the neuroglia, which is the connective-tissue framework of the true nervous matter. As seen under the microscope, a glioma is made up of fibers and cells, the latter being precisely like the

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* From a clinical standpoint, the term "cerebral tumor" usually is meant to include also such tumors as originate in the neighborhood of the brain (as in the base of the skull), if they finally involve the brain itself.

normal cells of the neuroglia, while the fibers seem to consist mainly of the numerous cell-processes. Klebs maintained that the ganglion-cells also take an active part in the new growth; but this has not yet been proven. It is characteristic of gliomata that they are seldom sharply defined, but shade off gradually into the healthy tissue. The affected portion of the brain is usually enlarged, but yet maintains pretty nearly its original shape. On cross-section, the glioma presents a gray or reddish-gray surface. It is usually rather soft, and almost always is very vascular. This great vascularity is not without clinical importance, for variations in the fullness of the blood-vessels, and, above all, sudden hæmorrhages into the interior of the new growth, which not infrequently occur, may produce marked symptoms.

Gliomata are most frequent in the white substance of the cerebral hemispheres, but they are also found in the central ganglia, cerebellum, and elsewhere. They are usually single, seldom multiple.

2. SARCOMA.—It is very seldom that any form of sarcoma originates in the brain-substance. It usually commences in the connective tissue of neighboring parts, in the dura mater, the periosteum of the cranium, or the cranium itself (osteo-sarcoma). The sarcoma is most often found at the base of the skull, in the form of a circumscribed tumor of varying consistency, and by pressing upon neighboring parts, or by involving them in the diseased process, it may cause the gravest disturbances. Histologically, we have here such varieties as round-celled, spindle-celled, fibro-sarcoma, etc.

3. GUMMA AND SOLITARY TUBERCLE.—Both gumma and solitary tubercle are very prone to attack the brain. We shall revert to gumma in the chapter on cerebral syphilis. Solitary tubercles may grow to the size of a cherry or larger. They may be single or multiple, and may occupy any part of the brain. They are most often found in the cortex, and in the cerebellum and pons.

Solitary tubercles and gummata, upon cross-section, have a yellowish, cheesy appearance, and are usually distinctly defined. Histologically, they are composed of granulation tissue. It was formerly no easy matter to distinguish gumma from tubercle in all cases; but now complete certainty is attainable by determining the presence or absence of tubercle bacilli (and likewise of syphilis bacilli).

4. CARCINOMA.—Carcinoma completes the list of such cerebral tumors as have much clinical importance. They are here almost always secondary. It has been our experience that secondary cancer of the brain is principally associated with primary cancer of the breast, or of the lungs and pleura; which fact bears a remarkable analogy to the occurrence of secondary cerebral abscess after primary empyema, pulmonary gangrene, etc.

5. Among the rarer varieties of tumor are *psammoma*, which usually starts from the meninges, is hard, generally comparatively small, and therefore often harmless, and contains calcareous matter, so that it grates under the knife; *cholesteatoma*, a rare tumor which has the brilliancy of mother of pearl; *lipoma;* and *angioma.*

The General Symptoms of Cerebral Tumors.—As is the case with all focal diseases of the brain, some of the symptoms of cerebral tumors are connected with the special localization of the new growth. There are definite focal symptoms, varying with the part destroyed, or at any rate functionally impaired, and it is from these symptoms alone that we are enabled to determine the position of the tumor. But, in addition to these focal symptoms, there are certain general symptoms common to almost all cerebral tumors of any size. They are in large part referable to the general compression of the brain due to the enlargement of the new growth. In the first place, numerous clinical facts, which will be immediately enumerated, indicate that whenever there is a tumor of any great size, a large part of the entire encephalon is subjected to pressure; and, secondly, the anatomical appearances of almost every brain affected by a large-sized tumor lead to the same conclusion. The convolutions are flattened and obliterated, the dura mater is crowded against the cranium, and perhaps thinned or even perforated because of the persistent pressure, or, on the other hand, thickened as a result of chronic inflammation. Now and then the effects of pressure are visible even in the bones of the skull: they are worn thin, or even perforated, or their sutures are loosened. Another result of the general intracranial tension, through its effect upon the venous trunks of the brain, is serous effusion into the ventricles (internal hydrocephalus), which occurs very frequently. The largest effusions are caused by tumors in the posterior cerebral fossa, which directly compress the venæ Galeni.

The symptoms of cerebral tumors, referable to the effects of general compression, are as follows:

1. Headache is one of the earliest and most constant symptoms. It is usually persistent, although subject to temporary exacerbations and intermissions. The patient describes it as dull, deeply seated, and stupefying. Although it affects the whole head, it is sometimes (not invariably) referred mainly to the neighborhood of the tumor. It is particularly true of persistent occipital headache that it indicates a new growth in the posterior fossa. Sometimes also it is possible, by tapping upon the skull, to find a hyperæsthetic region. Considerable caution, however, should be used in drawing diagnostic conclusions from such observations. The headache usually lasts to the close of the disease, and, even after the patient has become completely comatose, the persistence of the pain is still evident, from his low groans and the way in which his hand seeks his head.

2. Next in frequency to headache are intellectual and mental disturbances. Even the facial expression may be somewhat characteristic, being peculiarly languid, apathetic, and dull. The patient talks slowly, often having to think a long while before knowing what to say. Memory becomes impaired, especially with regard to the most recent events. The interest of the patient in those about him, and the things he used to care for, grows less and less. He has a sleepy, dazed look, and grows careless and untidy. Of course, individual cases present various deviations from this outline; but in general, cases are a good deal alike, although the degree of mental disturbance may vary from a slight dullness to complete dementia.

If unusual fullness of the blood-vessels, a hæmorrhage into the new growth, or some similar cause, induce a sudden temporary increase of tension, there may be such marked symptoms as syncope or an apoplectiform shock.

3. Other general cerebral symptoms are vertigo, slowing of the pulse, and vomiting. If, however, vertigo be a very prominent symptom, it implies that the cerebellum is especially encroached upon by the tumor. The retardation of the pulse is a frequent symptom, and not without diagnostic value. It has already been mentioned as one result of general compression of the brain, in connection with cerebral hæmorrhage. The rate may be put at about 50 to 60, or even lower. The pulse is also sometimes slightly irregular. Cerebral vomiting may be one of the earliest and most troublesome symptoms. It frequently occurs independently of the ingestion of food, especially in the morning, and is not infrequently associated with dizziness.

4. Epileptiform convulsions are sometimes excited by cerebral tumors, although many patients are free from them. Such attacks in all probability originate invariably in the cortex of the cerebrum, and it is therefore natural that they should be seen most frequently (as they are) in connection with tumors in the cerebral hemispheres. This rule, however, is not without exceptions. If the convulsions are not general, but are unilateral or confined to distinct portions of

the body, they are to be regarded rather as focal than as general symptoms, and may be utilized for the approximate localization of the lesion (*vide* page 711). A certain amount of information in the same direction may also be got from those attacks which begin unilaterally or in one particular limb, and then quickly involve the entire body.

5. Choked disk is one of the most important general objective symptoms of cerebral tumor. We should never omit to make an ophthalmoscopic examination of the fundus oculi in a case of chronic cerebral disease. Some differences of opinion yet exist in regard to the special pathology of choked disk; but we may regard it as extremely probable that the main factor in its production is the purely mechanical one of general compression of the brain. The original view of Von Graefe was that the increased intracranial pressure obstructs the venous return through the vena centralis retinæ into the cavernous sinus. The opinion which prevails at present is that advanced by Schmidt and Manz—namely, that the increased tension forces the cerebro-spinal fluid into the lymph-sheaf of the optic nerve (Schwalbe), and that the "*hydrops vaginæ nervi optici*" thus produced compresses the nerve and the blood-vessels which traverse it. [Leber and Deutschmann have shown pretty conclusively that the increased pressure is not the chief cause of optic neuritis. It favors its production, but probably there must be the presence of some irritant as well, in order to produce the inflammation.—K.] At any rate, choked disk is not a focal symptom. The tumor occasioning it may have any position, if only it gives rise to general compression of the brain.

Disturbances of vision may or may not be entailed by choked disk ; the patient may have amblyopia, defects in the field of vision, or even total blindness. In some few instances, amblyopia has been one of the earliest symptoms of cerebral tumor, insomuch that the patient has applied first of all to an oculist. Usually the sight is preserved for quite a long while, in spite of the abundant objective evidences of choked disk. The latter consist of swelling of the disk, marked distention and tortuosity of the veins, possibly hæmorrhages (from passive congestion), and cloudiness of the disk, although the retina still exhibits its normal transparency. It is not until the long-continued congestion impairs nutrition to such an extent as to cause atrophy of the optic nerve that vision is much impaired.

6. The last general symptom to be mentioned is general loss of flesh and strength. This often appears comparatively early. It is in large part due to the small amount of food taken, vomiting, and wakefulness; but it is not impossible that the grave cerebral disorder itself exerts a direct and unfavorable influence upon all the processes of nutrition. There is in most cases, also, a tendency to obstinate constipation.

Tumors in the Different Parts of the Brain—their Focal Symptoms.—The symptoms above discussed indicate the existence of a tumor, but not its particular location. Other phenomena are necessary to enable us to localize the disease, but it is not exceptional to have none but the general symptoms. Tumors in the white matter of the frontal lobe, or such as affect the corpus striatum, as well as others, may run their course without any focal symptoms; but most cases afford evidence which points with more or less certainty to the exact position of the tumor. Almost all of these focal symptoms have already been fully discussed in Chapter II of this section, and their interpretation follows the rules for all focal lesions of the brain. We may therefore be brief here. It is necessary only to emphasize the fact that with regard to cerebral tumors, as well as other lesions, focal symptoms should be divided into the direct and the indirect. Direct focal symptoms are the immediate result of the destruction of nervous tissue by the new growth, while the indirect are excited by the pressure exerted by the tumor upon the parts closely surrounding it. This pressure varies with the amount of blood in the vessels of the new growth, and therefore the indirect symptoms may undergo temporary exacerbations and remissions. An intermediate position is occupied by those focal symptoms which occur in many cases as the result of certain anatomical changes secondary to the new growth. Not infrequently there is white softening of the substance of the brain around the tumor proper. Probably this condition is generally the result of a compression of the minute blood-vessels surrounding the new growth, but sometimes it is the sequel of an obliterative endarteritis (Friedländer). The latter cause is especially operative where the tumor is a gumma or a solitary tubercle.

1. Tumors of the cerebral hemispheres generally lead to the gradual development of hemiplegia—a focal symptom which is to be regarded as partly direct and partly indirect. Since the new growth is often situated near the cortex, symptoms referable to that region are especially frequent with tumors of the cerebrum. It is therefore not infrequently the case that the hemiplegia is the result of the successive paralysis of single portions of the affected side; for instance, first there is only facial paralysis, then, in addition, paralysis of an arm, then of the lower limb. Very often the extension of the paralysis is accompanied by convulsions, which either are confined to one limb or one side of the body, or are universal. There may be still other focal symptoms, varying with the exact location of the tumor. Thus, there is hemianæsthesia, if the parietal region or the posterior part of the internal capsule be affected; hemiopia, if an occipital lobe suffer; aphasia, if the neighborhood of the left island of Reil be involved, and so on.

2. *Tumors at the Base of the Brain.*—The base of the brain is a very favorite place for new growths. The symptoms are in a majority of cases quite characteristic. Some of the tumors spring from the base of the skull; among these are many sarcomata and syphilitic growths ("gummatous periostitis"). Other tumors originate in the meninges, especially the dura; and still others from the brain itself. Of these last, it is remarkable that some spring from the pituitary gland. The exact starting point is very seldom of much clinical importance, for all the parts mentioned are in such close proximity to one another that there is no great difference in the symptoms produced. We can only decide that there is a tumor in this or that place at the base of the brain.

Tumors at the base of the brain owe their characteristic clinical stamp to the frequency with which the cranial nerves at the base are involved. The anatomical relations are such that these nervous trunks are often compressed by the new growth or actually incorporated in it. Of the symptoms thus occasioned, the most frequent is paralysis of the motores oculi (oculomotor and abducens). This is at first usually unilateral, but it may later affect both sides. If one of the optic tracts be involved, hemiopia may result, and pressure upon one optic nerve may produce unilateral choked disk with unilateral disturbance of vision. Tumors of the pituitary gland are especially prone to cause symptoms referable to the optic nerve at an early period. Lesions of the trigeminus not infrequently cause disturbances of sensation in the face, and occasionally also paralysis of the muscles of mastication. The trunk of the facial often suffers. The facial paralysis thus occasioned throws considerable light upon the diagnosis, for there is usually to be found in the paralyzed muscles the reaction of degeneration, showing that the paralysis is peripheral. We have therefore reason to assume that the lesion is situated at the base of the cranium, rather than central. Another factor is almost always present to support the idea of a peripheral lesion—namely, the frontal muscles are involved (*vide* pages 561 and 731). Peripheral paralysis of the hypoglossal nerve may also be produced by tumors at the base of the brain; but this is much rarer than facial paralysis. Whether other nerves of special sense besides the optic are disordered is a question about which we possess little information as yet, but probably careful examination will not infrequently reveal changes in them.

As might naturally be expected, various degrees and forms of paralysis in the extremities are often found in combination with the above disturbances of the cranial nerves. Such conditions are most frequent where the crus cerebri, with its pyramidal tract, is affected. There is no need of enumerating all the possible varieties of symptoms. We must consider them all carefully in each individual case, and then, by bearing in mind the anatomy of the parts, we shall, in a majority of instances, be enabled to determine with some approach to accuracy the place at the base of the brain where the new growth must be. Sometimes, but not often, we may be led into error by tumors which, though situated in the brain-substance and at a comparative distance, yet by their pressure give rise to indirect symptoms referable to the cranial nerves at the base.

3. *Tumors of the Cerebellum.*—We shall refrain from describing the symptoms which may be excited by tumors in other parts of the brain, with a single exception. Tumors of the cerebellum are comparatively rather common, and deserve a brief notice. The direct focal symptoms of cerebellar lesions, such as the peculiar pitching gait and the vertigo, have been discussed at page 723. But cerebellar tumors generally occasion also very strongly pronounced general symptoms—namely, headache, mainly occipital; sometimes a spasmodic and persistent stiffness of the neck; vomiting; and visual disturbances, mainly due to the frequent existence of choked disk. Analogous to this last symptom would seem to be disturbances in other nerves of special sense. Thus, where the general intracranial pressure is elevated, the acoustic or olfactory nerves seem liable to passive congestion. Tumors in the posterior fossa have several times been found to occasion bilateral anosmia and deafness; and they should always be considered where such a condition is found.

[In view of the increasing importance of the focal diagnosis of tumors of the brain, it has seemed advisable to insert the following brief summary of the focal symptoms of tumors in different parts of the brain:

Prefrontal Region.—Marked mental impairment; symptoms of invasion of the central region (Jacksonian epilepsy, aphasia); disturbances of smell.

Central Region.—Jacksonian epilepsy; monoplegia; partial anæsthesia; motor aphasia.

Posterior Parietal Region.—Word-blindness; disturbance of muscular sense (?); homonymous hemianopsia.

Occipital Region.—Homonymous hemianopsia; soul-blindness.

Temporo-Sphenoidal Region.—Latent region. Word-deafness; disturbances of taste, smell, and hearing (?).

Corpus Callosum.—Latent region. Progressive hemiplegia, often bilateral from invasion.

Optico-striate Region.—Hemiplegia; contracture. In posterior part, hemianæsthesia, homonymous hemianopsia, post-paralytic chorea, athetosis.

Crus Cerebri.—Crossed paralyses of oculomotor nerve and limbs.

Corpora Quadrigemina.—Oculomotor paralyses; reeling gait; blindness (?); deafness (?).

Pons and Medulla.—Crossed paralyses of face and limbs, or tongue and limbs. Other cranial nerve lesions.

Cerebellum.—Marked cerebellar ataxia; marked vomiting. Often a latent region.

Base, Anterior Fossa.—Mental impairment; disturbances of smell and vision; exophthalmus.

Base, Middle Fossa.—Disturbance of vision; oculomotor disturbances; hemiplegia.

Base, Posterior Fossa.—Trigeminal neuralgia; neuro-paralytic ophthalmia; paralyses of face and tongue; disturbance of hearing; crossed paralyses.

Hypophysis.—Disturbance of vision; oculomotor disturbances.

It also is of importance, in cases which may come to operation, to determine whether the growth be cortical or subcortical. Seguin has given certain rules which, however, are not absolute. "In favor of a strictly cortical or epicortical lesion are these symptoms, none of them having specific or independent value: localized clonic spasm, epileptic attacks beginning by local spasm, followed by paralysis; early appearance of local cranial pain and tenderness; increased local cranial temperature. In favor of subcortical location of a tumor: local or hemiparesis followed by spasm; predominance of tonic spasm; absence, small degree, or very late appearance of local headache and of tenderness on percussion; normal cranial temperature."—K.]

General Course of Cerebral Tumors.—The symptoms of these growths almost always cover a long period of time. Exceptionally a tumor remains latent till a hæmorrhage or some similar event takes place in it, giving rise to sudden and grave symptoms, and possibly to an equally sudden termination. The rule is, however, for the phenomena to develop gradually. According to the location of a new growth, either the general or the focal symptoms may come first into prominence. They generally occur in the order named. First of all is an ill-defined, deeply seated headache, and by degrees all the other general and focal symptoms follow after. The symptoms may vary repeatedly and greatly in severity, a fact due mainly to the varying pressure of the tumor on neighboring parts. Repeated mention has already been made of the sudden exacerbations which may come on, especially in case of the vascular gliomata.

The entire duration of the disease is usually at least some months, and may be one or two years or more. The termination is almost invariably unfavorable. Death may be rather sudden, or it may not come till after a long period of wretchedness. Fortunately, however, the lameness, blindness, and marasmus are frequently made less terrible to the patient because of his mental debility. Recovery is possible only where the growth is syphilitic. It is indeed possible that solitary tubercles may also end favorably, but the matter is still in doubt.

Diagnosis.—The main points in support of a diagnosis of cerebral tumor would be the gradual onset and continuous slow increase of the general symptoms above detailed—namely, headache, vertigo, vomiting, convulsions, dementia, etc. The most constant of these symptoms is the headache. They all indicate the development of some chronic brain trouble, a tumor being the most probable if there be no definite ætiology to suggest some other process, such as traumatism resulting in abscess, or syphilis. Much stress may also be laid on the choked disk, which is much less often seen in case of abscess or softening than of tumor.

The general symptoms indicate that a tumor of the brain exists; but, in order to learn the position of that tumor, we have to rely mainly upon the focal symptoms. Their gradual development and the way in which one new symptom is slowly added to another, also give further ground for the opinion that some continuously progressive disease exists, and most likely a cerebral tumor. Of diseases with a similar course, abscess is recognized by the absence of choked disk (an important point), frequently by febrile symptoms, and by its ætiology (trauma). Inflammatory and thrombotic softening, if they come on slowly, usually produce less general disturbance than do tumors; they seldom cause a choked disk, and (unless of syphilitic origin) are much rarer before middle age than tumors of the brain. Sclerosis sometimes simulates cerebral tumor; but here also there is no choked disk; the disease lasts much longer (five or ten years, or more), and, inasmuch as the sclerosis is usually multiple, there is frequently too great a complexity of symptoms to warrant the assumption of one solitary lesion.

[We must, however, bear in mind that, in about one seventh of the cases, tumors are multiple.—K.]

Certain rare cases of chronic circumscribed meningitis can not be differentiated from a tumor. They generally occur at the base, lead to a considerable thickening of the tissues, and they may simulate all the symptoms of a new growth in this region. Occasionally, also, chronic hydrocephalus is confounded with tumor of the brain. We met with a case of dropsy of the fourth ventricle which presented during life a perfect picture of tumor of the cerebellum.

As to the nature of a new growth, we can not go beyond surmises. If the focal symptoms indicate that the tumor is in the substance of the brain itself, our first thought would be of a glioma, because it is by far the commonest sort of growth in that situation; and, as has been stated, certain peculiarities in the course of the disease (especially if new symptoms add themselves abruptly) would make glioma probable. If, on the other hand, the tumor be at the base, it is most apt to be sarcoma, which is the most frequent form of new growths here. When symptoms referable to the optic nerve occur noticeably early, a tumor of the pituitary gland is to be thought of. In all cases, and especially in tumors at the base of the brain, we should bear in mind the possibility of syphilis. The previous history and the entire body of the patient should be closely searched with this point in mind; its therapeutic importance need not be dwelt on.

One special form of tumor deserves a brief mention here—namely, large cerebral tubercles. The growth may be single or solitary, or it may be multiple. It is seen chiefly in childhood, and any chronic cerebral disorder in a child should suggest the possibility of such a growth. It is rendered all the more probable by the co-existence of the signs of tuberculosis elsewhere, as in the lymph-glands, lungs, or bones. The symptoms are analogous to those produced by other tumors. Among the most frequent phenomena are headache and convulsions. The latter are often unilateral. There may also be all sorts of focal symptoms, according to the locality of the lesion.

Prognosis.—Except gummata, all tumors of the brain have a very unfavorable prognosis. It is said that in very rare instances tubercular growths have been arrested or cured; but in practice we can never rely upon any such result. In all other cases recovery is next to impossible. The time intervening between the appearance of the first symptoms and death varies greatly, as has been said. We should therefore be very cautious in predicting the duration of the illness. It seldom, however, exceeds one or two years, and sudden death without any warning may occur at any time.

[Tubercular growths are occasionally found at autopsies, which have become encapsulated, and have apparently existed for years without causing symptoms. In some cases, especially with tubercular growths in children, the symptoms diminish and the patient may live for years, suffering only from the blindness, etc., caused by the growth. Other cases may present only mild symptoms, and the patient may lead a fairly comfortable life for years.—K.]

Treatment.—Inasmuch as the nature of the tumor can not be determined with absolute certainty in any instance, antisyphilitic treatment should always be tried. Forty to seventy-five grains of mercurial ointment (grm. 3–5) should be used by inunction, and thirty to seventy-five grains (grm. 2–5) of potassic iodide should be given internally each day. If the new growth be syphilitic, much benefit may be done in this way. It must be confessed that the treatment is generally of little avail, because the tumors are not of a syphilitic character; although it may be that iodide of potassium sometimes has a temporary good effect upon other forms. A long-continued course of arsenic has also been recommended, in order to check the growth of the tumor. This remedy particularly deserves a trial where there is a suspicion of solitary tubercle.

Beyond what has just been indicated, treatment must be symptomatic. The headache is combated with ice-bags and narcotics; the convulsions require bromide of potassium or the inhalation of chloroform; the vomiting is lessened by rest in bed, opium, and bits of ice. The general care and nursing of the patient are very important, so that bedsores and the like may be avoided if possible.

[Within the last few years it has been demonstrated that tumors of the brain may be successfully removed by surgical interference. Out of forty-six such operations, thirty recovered from the operation, although in some cases the growth recurred. Hence the propriety of such an operation should be considered in every case, but unfortunately the growth must be in an accessible region and the focal diagnosis must be tolerably certain, so that an operation is indicated in less than ten per cent. of the cases. In cases with intense headache, where removal is impossible, relief may be obtained by removing a portion of the skull and tapping the lateral ventricles, and thus taking off pressure. Such an operation will relieve the pain, but it causes cerebral hernia, and probably hastens death.—K.]

APPENDIX.

HYDATIDS OF THE BRAIN.

It was stated on page 441, that the cysticercus cellulosæ, which originates from the tænia solium, may occur in great numbers in the brain. The cysticerci most frequently occupy the pia mater, but generally project downward into the cortex of the brain. The meninges not infrequently exhibit signs of chronic inflammation, and may present hæmorrhages, which are not always minute. If there are numerous cysticerci in the neighborhood of the ventricles, a varying degree of internal hydrocephalus usually develops The individual cysticerci are usually enveloped in a capsule of connective tissue, but may be entirely devoid of such a covering.

No characteristic clinical sketch of hydatids in the brain can be drawn, because the symptomatology of each case differs according to the number and position of the parasites. Sometimes cysticerci produce absolutely no symptoms, and are discovered incidentally at the autopsy. In other instances they cause a long and tedious illness. Epileptiform convulsions seem to be the most frequent symptom, and must be due to the position of the cysticerci in the cortex of the brain. These may, like true epileptic attacks, come on only at certain times when the general condition is otherwise good; or permanent general cerebral symptoms may also appear, such as persistent headache, vertigo, and mental disorder. Focal symptoms are likewise possible, but on the whole are rare.

The diagnosis can never be made with absolute certainty. The presence of cysticerci in the brain may be suspected when the above-mentioned symptoms occur in a butcher or other person who is from his calling especially exposed to infection, or who is known to have had or still to have a tapeworm, or in whom cysticerci have been demonstrated in some other part of the body, such as the skin.

We know of no remedy capable of destroying the cysticerci. Treatment, therefore, can be only symptomatic. [In a few cases, chiefly in Australia, the cysticerci have been successfully removed by surgical operations.—K.]

CHAPTER VIII.

CEREBRAL SYPHILIS.

Ætiology.—The importance of syphilis as an ætiological factor in many chronic diseases of the nervous centers has been repeatedly adverted to in preceding chapters. Although with regard to its influence upon the spinal cord in exciting tabes dorsalis and certain forms of myelitis there is still some obscurity, the brain presents with comparative frequency disorders unmistakably referable to constitutional syphilis.

Cerebral syphilis is almost always a tertiary symptom. It is only in exceptional instances that cerebral symptoms are produced by the end of a year from the date of the initial lesion. Usually the interval is several years, and it may be ten or even twenty.

Liability to the disease does not seem to be essentially influenced by age or sex. Even hereditary syphilis has been proved to cause diseases of the nervous system. But it can not be denied that a predisposition to cerebral syphilitic disease is often engendered by those influences which are apt to promote cerebral disease in general. Just as the position of syphilitic cutaneous lesions is often determined by external irritation at some one place on the skin, so the disease seems more liable to attack a brain which is exposed to certain unfavorable conditions than one which is perfectly normal and vigorous. Such conditions are inherited tendency to nervous diseases, various injurious mental influences, poisons, in a broad sense, and traumatism. It need hardly be said that even a previously sound brain does not enjoy immunity from the affection.

Pathology.—As far as has yet been ascertained, there are two chief forms assumed by syphilis in this organ: (1) a circumscribed syphilitic tumor, the gumma, and (2) a disease of the arteries of the brain, which is usually quite extensive. There is no essential difference underlying these two varieties. They may also occur in combination with each other. The disease of the blood-vessels is really a syphilitic new growth affecting the walls of the arteries.

The circumscribed syphilitic tumors are yellowish or grayish-red, and frequently cheesy in the center. Their most frequent seat is the dura mater or the subarachnoid spaces, whence they spread to the brain-substance; but exceptionally they may originate in the substance of the brain itself. Histologically they are made up of granulation tissue of varying degrees of vascularity, and presenting yellow spots usually visible to the naked eye. These spots are of firmer consistency than the rest of the growth, and have undergone coagulation-necrosis (have become cheesy). Circumscribed gummata in the brain which have become cheesy do not differ essentially in microscopic appearance from a collection of tubercles (see preceding chapter). The new growth sometimes takes on a more diffuse form in the meninges, particularly at the base (gummatous meningitis). Often the originally delicate granulations become transformed into a firm connective tissue, forming extensive cicatricial induration.

The syphilitic disease of the arteries was first fully appreciated by Heubner, who has described it accurately. It is usually most pronounced in the arteries of the base of the brain, and especially in the middle cerebral artery and its branches. Even the unaided eye detects a grayish opacity in the arteries. They feel firm and stiff, and on cross-section their walls are found to be thickened, either uniformly or in some places more than in others. This causes no inconsiderable narrowing of the lumen, or even its obliteration, particularly if the last gap be closed by the formation of a thrombus. The microscope shows that the new growth originates chiefly in the intima of the vessel, where there is a hyperplasia of the endothelium, and a gradual transformation of it into a firm connective tissue. But the adventitia also undergoes a gradual thickening of considerable extent. Syphilitic endarteritis presents no distinctive histological characteristics. Entire certainty that the inflammation is due to syphilis can be attained only by discovering other evidences of syphilis, whether in the brain or elsewhere, or from the personal history and the previous course of the disease.

The great clinical importance of syphilitic endarteritis is due to its cutting off the normal supply of blood from the regions supplied by the diseased arteries. If the occlusion be complete, cerebral softening is inevitable, as in ordinary embolism and thrombosis of cerebral arteries; and, inasmuch as the middle cerebral artery is particularly liable to the disease, syphilitic softening is most often found in the region supplied by this vessel.

Clinical History.—The variety of the pathological processes and of their location produces a corresponding variety in the symptoms of cerebral syphilis. We shall therefore be obliged to limit ourselves to a brief description of some few types of the disease which are oftenest met with (Heubner).

1. The group of symptoms may be mainly those of a cerebral tumor. Here there is a circumscribed new growth, situated either at the base, or upon the convexity of the brain (and in the meninges). If at the base, the symptoms are analogous to those discussed on page 752. The focal symptoms are often preceded for a certain length of time by general cerebral symptoms, such as persistent headache, worse at night, wakefulness, mental depression, and impairment of memory. Then paralysis of the nerves at the base of the brain comes on: the nerves controlling the motions of the eyeball suffer the most frequently, but the facial and other nerves may also be affected.

In the second subdivision, where the syphilitic new growth is mainly upon the convexity of the brain, the picture is a tolerably characteristic one. Often in this case, also, prodromata similar to those just enumerated precede the severer symptoms. Then appear violent epileptiform convulsions. These often come on very suddenly, and may recur at considerable intervals or in quick succession. There are usually still other symptoms of cortical disturbance, especially paresis of one limb or even of one half the body; very frequently slight disturbance of speech (stumbling over syllables), referable to the cortex, and indications of mental impairment. Many of these cases reach a fatal termination comparatively early. The epileptiform convulsions become more and more frequent, and unconsciousness increases into a deep coma ending in death. But it is in cases of just this sort that prompt and energetic treatment may accomplish a great deal.

2. Another common and important variety of cerebral syphilis is characterized chiefly by syphilitic arteritis. Not infrequently there is a prodromal stage; then, as a result of the occlusion of some vessel, which often occurs quite suddenly, there is a pronounced apoplectic attack, followed in most cases by hemiplegia. The intensity of the initial shock may vary greatly; sometimes there is only a slight dizziness, sometimes there is a coma that lasts for days. Sometimes the shock is succeeded by a peculiar condition of mental confusion and dullness, which may persist for weeks. In severe cases, death is speedy, and is usually ushered in by a great rise of temperature. Other patients improve more or less rapidly, especially under proper treatment.

Apoplectic attacks of this sort may recur after temporary improvement has taken place, and may be associated with all sorts of nervous symptoms.

3. In a third class of cases, cerebral syphilis assumes the form of a diffuse chronic disease of the brain, closely resembling multiple sclerosis or certain types of progressive general paralysis of the insane. Perhaps it would be more correct to say that certain cases of cerebral syphilis are identical with general paralysis.

There are gradual impairment of memory and of speech, and various motor symptoms (tremor, ataxia, paralysis of single members). The intellectual powers grow less and less, and, after passing years as a physical and mental wreck, the patient at last dies, unless indeed he has the good fortune to be carried off earlier, in some apoplectiform or epileptiform attack. In such cases the autopsy sometimes also shows specific syphilitic changes, especially in the smaller vessels; but we find chiefly atrophic and degenerative processes in the nerve-fibers, which, in our opinion, should be separated from the special syphilitic new formations under the name of "post-syphilitic changes."

Diagnosis.—While some few of the phenomena produced by cerebral syphilis are rather characteristic—we refer, for instance, to the intense prodromal headache, the epileptiform convulsions, and the apoplexy—still these symptoms alone are never sufficient to establish the diagnosis, for they may be precisely simulated in cases of tumor, softening, hæmorrhage, multiple sclerosis, and other cerebral diseases. The most important diagnostic criterion is in every case the demonstration of the ætiology—that is, a previous infection with syphilis. We can not here describe in detail the methods of determining this fact. The history of the patient, as well as previous specific affections—such as, in women, miscarriages, abortions, etc.—and the objective signs on other parts of the body, are the two sources of information. There may be scars on the skin or mucous membranes, enlarged glands, ulcers, tibial periostitis, or changes in the testicles. Age is also important: thus an apoplectic attack in a young person would suggest syphilis, because the other causes of such an attack operate chiefly upon the aged. The results of treatment often throw considerable light upon the diagnosis. As there is nothing to lose and much perhaps to gain, we should always give specific remedies in doubtful cases. If they prove successful, the diagnosis of syphilis receives strong confirmation.

Prognosis and Treatment.—There are few severe and dangerous diseases where timely and appropriate treatment is attended with so much success as that achieved in many cases of cerebral syphilis. In order, however, both to understand the favorable results and not to be misled by the failures, we need to gain a clear idea of the way in which antisyphilitic remedies can be of benefit. They can accomplish this only by causing the dissipation and absorption of the new growth—that is, the gumma or the swelling of the intima. If this be effected, the surrounding parts are of course relieved from pressure, and the circulation becomes unimpeded. If the tissues still retain functional power, they resume their duties and all symptoms of disease vanish. But when the tissue has already been considerably impaired by the compression, or by the scanty blood-supply, the results are quite different. Even then the nervous trunks at the base of the brain may gradually become regenerated; but such parts of the true cerebral parenchyma as have undergone softening have lost their functional capacity forever. In this case, antisyphilitic treatment is unavailing.

It is therefore obvious that the first essential of success is to begin treatment as early as possible. The sooner a correct diagnosis is reached, the sooner will existing symptoms be relieved, and further danger be averted. The method of treatment which will probably accomplish all that can be accomplished, and in the shortest possible time, is energetic mercurial inunction. At least a drachm (grm. 3–5) of mercurial ointment must be rubbed in every day at first, according to the ordinary method. We should not venture to restrict the diet unless the patient be well nourished and "full blooded." If he be anæmic and feeble, a generous regimen is demanded. Usually the internal administration of iodide of potassium is combined with the inunctions; we should give thirty to forty-five grains (grm. 2–3), or, in severer cases, even a drachm or a drachm and a half (grm. 4–6), daily.

[In this country iodide of potassium is given much more freely. In cases where syphilis is suspected, we may safely begin with doses of thirty grains (grm. 2) three times a day. If the symptoms be urgent, the dose should be rapidly increased until the patient takes three or even four drachms (grm. 10–15) three times a day. If the patient have syphilis, the danger of iodism seems less. The drug must be given in large amounts of water, and it is better borne if given in Vichy or Giesshubler water. Sometimes the stomach will not tolerate the extreme doses.—K.] The same remedy should also be given afterward, for a long time, in smaller doses. Where there is no benefit at all after twenty or thirty inunctions, there is little prospect of any appreciable improvement. In favorable cases, the mercury often begins to produce some effect after the fifth or sixth inunction; and it may cause astonishingly rapid improvement. Potassic iodide alone is sufficient for the milder cases only, where there is merely headache, trigeminal neuralgia, or paralysis confined to the motores oculi.

In many cases some symptomatic treatment is also required. Narcotics, applications to the head, electricity, baths, etc., are employed, as in other chronic cerebral diseases, and they often supplement very efficiently the specific remedies.

CHAPTER IX.

PROGRESSIVE GENERAL PARALYSIS OF THE INSANE.

(*Paralytic Dementia. Paretic Dementia. General Paresis.*)

Preliminary Remarks.—Although the description of mental diseases is not properly a part of the plan of this book, we must nevertheless make an exception of one disease of the sort, namely, the so-called progressive general paralysis of the insane, or paralytic dementia, which in medical parlance is often abbreviated into "general paralysis." We consider it advisable to make this exception, because a great part at least of the symptoms of general paralysis are purely of a physical nature, and also because a knowledge of this disease, which is so common and so fatal in its results, is of the greatest importance for the general practitioner.

We must thank the French alienists Boyle (1822) and Calmeil (1826) for the first clinical descriptions of general paralysis, by which it was more sharply differentiated than previously from other diseases which run a like course. A more accurate knowledge of the different symptoms, and the anatomical changes to which the morbid symptoms must be referred, has, however, only of late years been rendered possible by the introduction of better methods of investigation. Accordingly, we must now say that general paralysis is a disease which may attack the most diverse portions of the whole central nervous system *—the brain and spinal cord—at the same time or successively, but in which, of course, we can make out certain rules as to the predisposition of individual portions to disease, and as to the order of the symptoms. General paralysis begins most frequently in those regions of the cerebrum which have an immediate relation to the regular course of the psychical and certain psycho-motor processes. Mental and motor symptoms accordingly form the introductory features of the disease in most cases. More extensive regions of the central nervous system are gradually involved in the morbid process, which goes hand in hand with a progressive degeneration of all the higher intellectual faculties, while at the same time many physical disturbances dependent upon nervous changes constantly increase.

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* At present hardly anything is known in regard to a primary implication of the peripheral nerves in the whole process of general paralysis. No theoretical objection can be raised to the supposition of such an implication.

Ætiology.—General paralysis is a common disease, and apparently demands a heavier quota from the better and more highly educated classes than from the lower classes. We may assume that, on the average, one tenth of all patients committed to the insane asylums are general paralytics. In most patients the beginning of the disease falls in the period between the thirtieth and fiftieth year. The disease is much rarer in advanced life. In young people under twenty it has hardly ever been observed. There is no doubt that the male sex is much more frequently affected than the female, but the number of cases of general paralysis among women is not very small.

What is the special cause of general paralysis? A generally acceptable answer can not be given; but the theory is constantly gaining ground, and we agree to it, from our own experience, that by far the most important causal factor is a previous syphilitic infection. Such an infection can be made out in at least seventy-five per cent. of all cases. In this regard precisely the same conditions exist, and, of course, the same difficulties in the interpretation of this relation are to be considered, as we have previously mentioned in the account of the dependence of tabes dorsalis on syphilis (see page 632), a circumstance which again is not without significance, since intimate points of connection are to be found between tabes and general paralysis (*vide infra*). If we consider that general paralysis depends upon a previous syphilis, we have an easy explanation of most of the other peculiarities in the onset of the disease, especially the above-mentioned influence of age and sex, the decidedly common occurrence of the disease in persons in certain callings—such as artists and officers—the frequency of the disease in large cities in distinction from its rarer occurrence in the country, etc.

Besides the ætiological factors named, which, in our opinion, are the most important, all other " causes " of general paralysis may well be regarded as merely predisposing. Mental overexertion has the greatest significance, especially if it be associated with psychical irritation. In merchants, civil officers, etc., who suffer from general paralysis, such a previous overexertion can often be made out. In some cases injury to the head or insolation is claimed to be the cause. Hereditary predisposition to nervous diseases plays perhaps a certain part in the origin of general paralysis, but by no means a very large one.

Clinical History.—General paralysis usually begins so slowly and gradually that a definite period for its beginning can hardly ever be given. In addition, it is often clear, at a period when the disease is already fully developed, that certain early symptoms, whose nature was at first not correctly recognized, ought to have been regarded as the initial symptoms.

The first symptoms of the disease in the psychical domain usually consist of the gradual appearance of a change in the whole nature and in the mental individuality of the patient; wherein, however, the mental disturbance usually shows from the start the character of weakness—that is, of a lessened capability of psychical exertion. The patient's ordinary mental work no longer goes on as easily as before. His memory is uncertain, and there are marked forgetfulness and inattentiveness, which were previously quite impossible for him to exhibit. The patient is often disorderly in his dress, and violates the ordinary social rules of decency and morality. Since his judgment as to the value and significance of things is uncertain, he commits purposeless actions, wastes money, commits crimes, is dissolute, etc. In these respects, too, the increasing mental dullness often appears, since the patient becomes incapable of any higher intellectual, æsthetic enjoyment, and since the nobler sway of feeling finally becomes dulled, and can no longer exert any lasting influence upon his actions. Besides all these signs of beginning mental weakness, we often notice, on the other hand, an abnormal irritability. The patient easily becomes agitated, or gets angry; but these humors rapidly pass away without leaving a lasting impression. We easily understand how this change in the whole personality of the patient must distress and alarm his family, since the relatives at first can not understand at all why the patient is now "so different from what he was."

In the first period of the disease a subjective feeling of illness is frequently present. The patient himself notices that his mental capacity, especially his memory, is diminished, and he very often becomes extremely anxious on this account. It frequently happens that certain subjective sensations are also noticed, a feeling of confusion in the head, pressure in the head, dizziness, rheumatoid pains, etc. The sleep is disturbed, as a rule, and also the appetite and the digestion. If such a patient comes to the physician with his complaints, it unfortunately only too often happens that he is in the beginning regarded as "neurasthenic," and is treated accordingly.

Careful observation, however, may even now usually discover the disease with certainty. The beginning mental disturbance is usually more apparent to the family than to the physician, who has not known the patient before and who sees him only cursorily, but it can usually easily be confirmed on a somewhat more searching examination of the patient. We generally succeed best by making the patient reckon; he often makes the greatest mistakes in simple examples in multiplication; especially does he forget to add numbers carried in the mind, etc.

Certain motor symptoms, however, which usually come on in the early stages of the disease, are of the greatest diagnostic significance, especially peculiar disturbances of speech and handwriting. The paralytic disturbance of speech shows itself first in the form of stumbling over syllables (*Silbernstolpern*), or literal ataxia. The individual sound (in distinction from bulbar paralysis) can be pronounced quite correctly, but the combination of different sounds in the whole word causes increasing difficulties. It is a good plan, in order to recognize the first beginnings of this difficulty, to have the patient pronounce a few difficult words, such as "third riding artillery brigade," "representative government," "initiative," "electricity," etc. We often hear "artralleriry" instead of "artillery," and like blunders. In the later stages of the disease the speech is sometimes almost wholly incomprehensible. We also observe other more complicated aphasic disturbances, such as paraphasia, persistent repetition of the same word, etc. In such cases the patients are sometimes no longer able to read any sentence correctly. They at times put in entirely different words, so as to make utter nonsense; but they do not notice it themselves. The abnormal associated movements of the facial muscles on speaking are also often to be observed, and are very characteristic. The voice of general paralytics often loses its power of modulation and becomes weak and rough—symptoms which depend upon a defective innervation of the vocal cords. The change in the handwriting, to be observed in general paralytics, is even more characteristic than the disturbance of speech (see Fig. 106). This is at first purely of a motor nature; the letters are uncertain, irregular, and tremulous. A psychical factor, however, also shows itself; single letters are omitted, the dot on the *i* and the marks of punctuation are forgotten, the patient ceases to keep on the lines, or leave a free margin, etc. As the disease advances, the disturbance in the handwriting gradually increases, so that the writing may finally become wholly illegible, and may consist merely of senseless scratches.

Besides the changes in the speech and the handwriting, which we have just briefly described, other physical disturbances are often quite early symptoms, and prove in how many parts of the nervous system at once the disease may begin its work of destruction. The condition of the pupils especially is of importance in diagnosis. They are often unequal, and also show a reflex immobility (see page 642) in a large number of cases, especially in those in which other tabetic symptoms develop (*vide infra*). Transitory ocular paralyses are at times early symptoms. Not infrequently we find quite early changes in the tendon reflexes, either

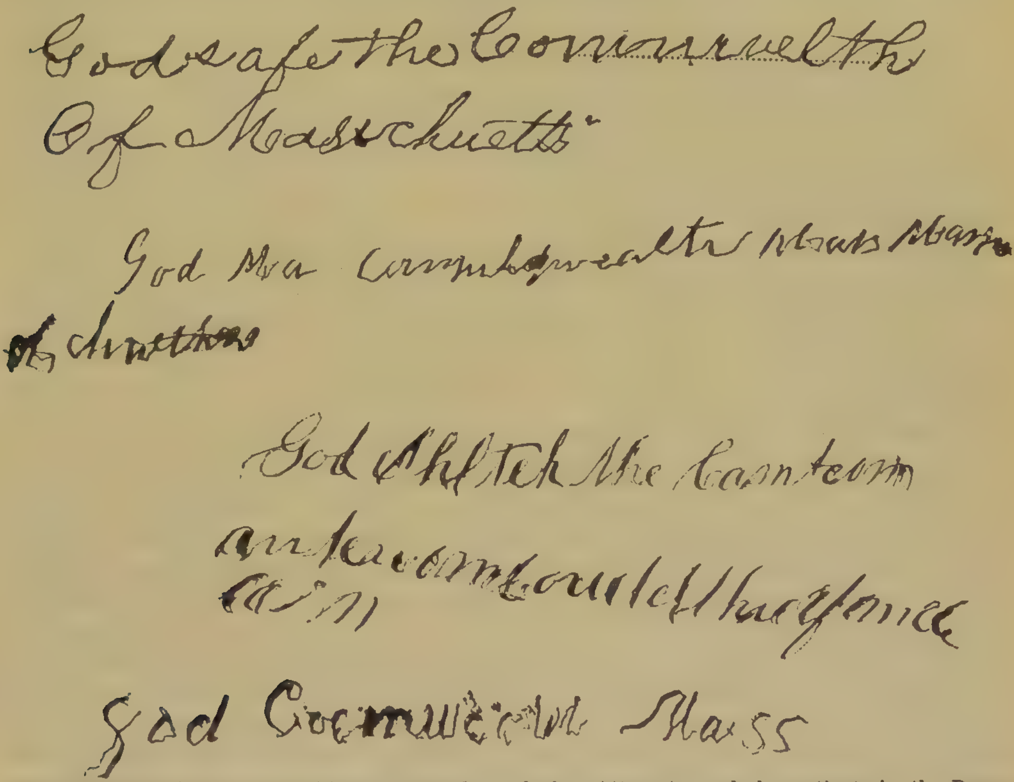
Godsafe the Commwelth
Of Masschuetts
God Ma
God Mass

FIG. 106.—Examples of handwriting in general paralysis. Attempts made by patients in the Danvers Lunatic Hospital to write "God save the Commonwealth of Massachusetts." Besides the motor disturbance, the frequent omission of certain letters will be noticed, e. g., "Masschuetts" for "Massachusetts." In 3 and 4 the handwriting is almost wholly illegible.

absence of the patellar reflex (a tabetic symptom) or an increase (beginning spastic paralysis of the legs). The association of reflex immobility of the pupils with an increase of the patellar reflex is not uncommon. In the distribution of the sensory nerves we may mention as repeatedly observed symptoms, neuralgia, attacks of migraine, and finally optic atrophy, the latter usually as one symptom of a co-existing tabes.

We can not give a generally applicable account of the further course of general paralysis, since it may differ decidedly in this respect. In what follows we can point out only the main features of the different types of the disease, and, in particular, we will sketch only very briefly the groups of mental symptoms.

We often say that those cases belong to the "classical form" of general paralysis where an initial "stage of depression" with a melancholy tendency is followed by a second stage of "maniacal exaltation." This is the stage where the delusions, which are already quite pronounced, assume more and more the character of "grand ideas," and thus exhibit the "delusions of grandeur" which have for a long time been generally regarded as ominous. The first signs are often found in the patient's statements that he is now much better, that he is "very well," that he feels "very strong," etc. These delusions, however, often assume gradually a more exaggerated form; the patient considers himself enormously rich, he owns thousands of palaces, millions of dollars, has made the greatest inventions, considers himself the Emperor Napoleon, Christ, a "higher God," etc. Any judgment as to the absurdity of these ideas, and as to the sad contrast between his claims and the reality, has already become impossible to him; but, of course, there are even now occasional remissions in this condition, when the patient is clearer and recognizes temporarily the morbid character of his delusions.

We must not think, however, by any means, that the delusion of grandeur is necessarily an invariable symptom in general paralysis. In many cases (the so-called "depressive" form of general paralysis) the initial melancholic-hypochondriacal condition continues. The delusions that appear possess the same coloring; the patient claims that he can no longer eat, that he is poisoned, that he no longer has a head or an arm, that he is very small ("*delire micromaniaque*"), etc. Sometimes acute and severe conditions of anxiety come on. In other cases, again (the agitated or maniacal form of general paralysis), there are states of violent excitement, in which the patient raves loudly, cries, and tries to destroy whatever falls in his way. Such states sometimes alternate with delusions of grandeur. Finally, we not infrequently see cases which, in their mental relations, present simply the symptoms of a mental enfeeblement gradually increasing to complete dementia, without ever showing, in any notable form, states of excitement, the development of delusions, etc.

While the intellectual life steadily goes to utter ruin in the ways just stated, the physical disturbances of the disease, as a rule, gradually advance in a more severe degree. In many cases ataxia of the extremities develops, and also loss of sensibility and vesical disturbances—in short, the symptoms of tabes. In these cases the tendon reflexes are almost always lost, and the pupils are often immobile. In other rarer cases, however, there is actual paralysis first in the lower and then in the upper extremities. In these cases the tendon reflexes are often increased, so that the picture of "spastic paralysis" develops. Again, in other cases bulbar symptoms appear, such as disturbances in swallowing or masticatory paralysis, and also ocular paralyses, usually as a part of the tabetic symptom-complex, etc.

Peculiar attacks, which are among the commonest and most characteristic symptoms of general paralysis, are, however, of special interest, and sometimes even of marked importance in diagnosis. These "paralytic attacks" in their milder degrees sometimes appear even in comparatively early stages of the disease. Then they usually consist of attacks of vertigo, an obscuring of consciousness, or even a loss of consciousness, coming on quite suddenly, and lasting from a few minutes to half an hour or more, and they are very often associated with mild hemiplegic or monoplegic symptoms. We very often see, besides the vertigo, a temporary feeling of weakness in the right arm, associated with a marked aphasic disturbance of speech. To this there is often added some slight twitching in the affected extremities or in the face. In the further course of the disease the attacks usually increase, and are termed apoplectiform or epileptiform paralytic attacks, according as the conditions of paralysis or spasm predominate. The epileptiform attacks may often be repeated with great frequency—thirty or forty attacks a day or more—during which time the patient remains in an unconscious state. If the patients gradually return to consciousness, sometimes only after a week or two, we very often see, as a result of such severe attacks, a permanent impairment of the general condition, an increase of the dementia, etc.

The other organs, apart from the nervous system, are only secondarily implicated in the morbid process. It may be mentioned briefly here that formerly, for a long time, stress was laid upon certain changes in the pulse, the pulsus tardus especially being thought to be characteristic: but the numerous investigations of the pulse have so far not given us any significant or sure results.

The temperature as a rule is approximately normal, or often somewhat subnormal, but very marked changes in the temperature occur in connection with the paralytic attacks—sometimes elevations and sometimes very deep declines.

The whole duration of the disease is in some cases only a few months (the "galloping" form of general paralysis), usually it is two or three years, and sometimes much more. The most rapidly fatal form is that in which there is very soon a marked emaciation and a rapid loss of strength as a result of the sleeplessness, the constant unrest, and the refusal of food. In other cases death ensues from the gradual and general loss of strength, or in a paralytic attack; or, finally, as is frequently the case, from the onset of secondary conditions, such as severe bedsores, pyelo-cystitis, tuberculosis, intestinal diseases, etc.

Pathological Anatomy and Nature of the Disease.—Considering the great difficulty of an accurate microscopic examination of the brain, it is not strange that our knowledge of the pathological anatomy of general paralysis is still very defective. If we except occasional immaterial changes in the skull, such as hyperostoses and the like, or in the meninges, such as hæmatoma of the dura or secondary thickening of the pia over atrophied portions of the brain, the first striking and at any rate most important anomaly seems to be the atrophy of the brain which affects chiefly the anterior half, especially the frontal lobes. In this region the convolutions are very much diminished, and the fissures are wider; the weight of the anterior portion of the brain may be reduced to one fourth or one third of the normal. If we examine the convolutions microscopically we find that the diminution of the whole organ depends chiefly upon a loss of nervous elements. The cortex usually shows the greatest changes. In fresh cases we sometimes see the signs of a mild "inflammation"—that is, we find vascular dilatation and little disseminated foci of round cells about the vessels, but the changes in the nervous elements themselves are far more important, and consist essentially in a degenerative atrophy. We must mention especially that in the cortex of the frontal lobes, especially marked in the straight convolution and in the island of Reil, and also in other portions, we can make out with certainty, by the aid of good methods of examination, a very considerable loss of the fine medullary nerve-fibers, mainly those which run parallel with the surface, and hence are termed "association-fibers" (Tuczek); but signs of degeneration and atrophy are very often to be seen in the ganglion-cells themselves. The destruction of the nervous elements is regarded by many investigators as secondary, since they lay the chief stress upon the marked changes in the interstitial tissue, such as increase of the connective tissue, numerous spider-cells, or thickening of the vascular walls, which are almost always to be found in old cases, and hence they speak of an interstitial encephalitis (Mendel). We ourselves, with Tuczek, Wernicke, and others, are much more inclined to the theory that we have to do mainly with a primary process of degenerative atrophy of the nerve-fibers and nerve-cells, to which the increase of the connective tissue is only a secondary addition.

The anatomical affection in general paralysis, moreover, is by no means limited to the cerebral cortex. We can often make out the loss of fibers in the deeper parts also, in the white substance, and the central ganglia. The co-existing changes in the spinal cord, first accurately described by Westphal, and since then recognized as almost constant, are of especial interest. They usually consist of fascicular systemic degeneration of the lateral columns (the pyramidal tract), or the posterior columns. A large part of the physical disturbances of general paralytics, such as tabetic symptoms or spastic paralysis (*vide supra*), are certainly due not to the cerebral disease, but to these accompanying changes in the spinal cord.

Accordingly, we believe that, according to our present knowledge, we can best conceive the nature of general paralysis in the following way: By the action of certain injurious influences, which usually seem to stand in some connection with syphilis (see page 632), there is a gradually progressive destruction of nerve-tissue in the most diverse portions of the nervous system. The clinical symptoms must naturally differ according to the significance and function of the affected fibers or cells. As a rule, certain cortical regions of the cerebrum are first diseased. The disturbances of speech are probably dependent upon the loss of fibers in the left island of Reil, the disturbances of intelligence upon the destruction of fibers in the frontal lobes. We may also make out corresponding anatomical changes, either cerebral or spinal, as an explanation of the later motor, tabetic, and other symptoms; but in many cases the order in which the different sections are affected varies considerably. We have seen in a previous section (see page 645) that the whole process may begin with a spinal disease, especially tabes dorsalis, to which the paralysis is "added" later; but we must understand that the two conditions are wholly analogous to and co-ordinate with each other. Both are parts of the same degenerative process, which can accomplish its work of destruction in the most diverse regions of the nervous system.

No coarse anatomical lesions can be made out, as a rule, to explain the paralytic attacks, but it is very probable that they depend, at least in great part, upon the changes in the motor central convolutions.

Diagnosis.—Since the diagnosis of beginning general paralysis is of the greatest practical importance, we will once more mention briefly all those symptoms which are especially to be considered in diagnosis: Striking alteration in the behavior, rapid and motiveless change in the disposition, disturbances of memory, loss of intelligence (failures in reckoning, etc.), the characteristic changes in the speech and handwriting, and finally the somatic symptoms which often co-exist: difference in the pupils, immobility of the pupils, loss, or, more rarely, increase of the tendon reflexes, and mild paralytic attacks, such as vertigo, disturbance of speech, temporary disturbance of motion in one arm, etc.

We would also mention, as especially common and disastrous mistakes, that the symptoms of general paralysis are often misunderstood at first, and are regarded as the signs of immorality, the failure of the sense of duty, etc. It also frequently happens that general paralysis is at first regarded as simple neurasthenia or hypochondriasis, and treated accordingly.

As a rule, general paralysis can be certainly distinguished from other organic nervous diseases by careful attention; but of course we must add that in some cases cerebral tumors, syphilis of the cerebral arteries, and especially certain cases of multiple sclerosis, may show a type of disease very like general paralysis.

Prognosis.—The prognosis of general paralysis, like that of all chronic degenerative conditions of the central nervous system, is very unfavorable. At present we know of only a small and decreasing number of actual recoveries, but there are many cases where there is a temporary improvement in the condition, a "remission," sometimes of a considerable degree and lasting a long time. The earlier the patient comes under proper care and treatment the sooner may we hope for such a favorable turn. Of course, as we have said, relapses of the disease almost always come on later. Those cases especially are to be regarded as unfavorable in which frequent paralytic attacks come on early, in which other physical symptoms, especially of a spinal nature, soon set in, and in which the whole nutrition of the body rapidly suffers.

Treatment.—As soon as the disease is recognized, the first and imperatively necessary injunction must be to remove the patient from all physical and intellectual exertion as well as from all mental excitement. The patient must therefore, if possible, withdraw from business, which up to that time he may have tried to carry on. His methods of life and his diet must be regulated, and every excess must be forbidden. For the cases which even at first are associated with states of great mental excitement, the commitment to a proper asylum is often most urgently to be recommended, while for cases that during their course show simple mental weakness, care at home is often sufficient.

In regard to the treatment of the disease itself, we should advise inunction with mercurial ointment, especially if we can discover a previous syphilitic infection. As a rule, we ought not to expect much success from this any more than in tabes (see page 647), but we may perhaps check the advance of the disease. We should therefore try the antisyphilitic treatment chiefly in the initial stages of the disease. We may combine the internal use of iodide of potassium with the inunction.

Furthermore, we should try tepid baths, with cool sponging, and also a cautious application of electricity (galvanization of the head and spinal cord), and, of internal remedies, ergotine especially. We need not go more fully here into the numerous symptomatic details.

CHAPTER X.

CHRONIC HYDROCEPHALUS.

Ætiology and Pathology.—Repeated mention has been made in preceding chapters of the occurrence of dropsy in the ventricles as a sequel to other cerebral diseases, such as meningitis and tumors. Besides this "secondary hydrocephalus," a collection of fluid in the ventricles may be a symptom of an apparently idiopathic primary disease. This is observed most of all in the newborn, or at least in young children.

Little is known with certainty about the causes of chronic hydrocephalus. The assumption is very frequently made that the condition is the result of an inflammation of the ependyma of the ventricle, which itself occurs either before birth or very soon after, but the autopsy often fails to support this idea. There is equally slight objective evidence that there is a stasis due to mechanical obstruction. Syphilis and dipsomania have been regarded as predisposing causes; whether justly or not, is uncertain. It has been repeatedly observed that the disease has attacked several children of a single family.

The most important physical sign of hydrocephalus in children is enlargement of the head. The circumference of the skull may even in the first year of life be sixty to eighty centimetres. Usually the frontal bones and the parietal eminences are especially prominent. The cranium becomes gradually almost as thin and translucent as paper. The fontanelles and sutures gape widely. The brain is flattened out, so as to seem almost like a bag, filled with the hydrocephalic fluid. In well-marked cases the entire thickness of the hemispheres is frequently not more than an inch. The space within, containing the serous effusion, represents the enormously distended ventricles, particularly the lateral ventricles, although the third and fourth ventricles are quite often distended also. The walls of the ventricles are often strewn with minute granulations, or they present a reticular hypertrophy. The hydrocephalic fluid usually has the appearance of colorless serum, and contains a very slight amount of albumen, if any. The specific gravity is about 1004 to 1006. The amount of fluid may be a quart or more; but, of course, there is great variation in this respect in different cases.

Congenital hydrocephalus is often associated with other peculiarities or defects in the structure of the brain, which we can not mention here.

Clinical History.—Sometimes a child is born with hydrocephalus so far developed as to occasion dystocia. Usually, however, the parents notice nothing peculiar about the child for some weeks. Then they are alarmed by the gradual swelling of the head. As a basis for determining abnormal size, we may mention that under normal conditions the circumference of the head at birth is about forty centimetres, at the end of a year about forty-five centimetres, and from that age to puberty there is a gradual approach to a circumference of about fifty centimetres. The possible dimensions in chronic hydrocephalus have been already stated. The increase in circumference is often quite rapid, amounting in a fortnight or three weeks to one or two centimetres. Usually the swelling is tolerably symmetrical; but sometimes the greater increase is in the antero-posterior diameter, making the skull dolichocephalic. At times the rate of expansion may be particularly rapid, and then at other times it may seem to be suspended. That the fontanelles and sutures remain widely open has already been mentioned; sometimes it is even possible to get fluctuation through them. An intravascular murmur can now and then be heard in the head, but it has no great importance with regard to diagnosis. The veins are often so greatly distended as to form a bluish network underneath the scalp. The face remains small, in striking contrast with the great, heavy cranial portion of the head. The head almost always hangs over forward from its weight. The eyes generally look down, partly because the roof of the orbit is depressed, and partly because of impairment of the nervous supply to the motores oculi.

A very important symptom is the defective intellectual development of hydrocephalic children. They can not learn to talk well, if at all. If they play, it is in a silly manner. They can not concentrate their attention upon anything, and they are heedless and dirty. It must, however, be mentioned that sometimes, in spite of considerable hydrocephalus, the patient now and then evinces an unexpected activity of mind—thus, he gradually becomes able to distinguish the different objects and individuals about him.

There is almost invariably motor disturbance also. The legs, more rarely the arms, are decidedly paretic, or there may be even complete paraplegia. There are usually spastic symptoms and increased tendon reflexes. Few patients learn to walk or stand alone. The arms seldom present any great paresis, but their movements often betray an awkwardness and uncertainty suggestive of ataxia. It is noteworthy that sensation almost always remains intact. At least the patient reacts vigorously to the prick of a pin, etc. Of the special senses, sight is most frequently affected; choked disk and atrophy of the optic nerve have been observed repeatedly. Symptoms of motor irritation are of very frequent occurrence, such as general convulsions and spasm of the glottis. General nutrition is pretty well maintained in many cases, but, as a rule, hydrocephalic children are atrophic and ill developed.

The chronic hydrocephalus of children almost always terminates unfavorably. Only a few patients survive the fifth year, although now and then striking exceptions occur. Death is generally the result of marasmus; or a convulsive seizure may prove fatal. The possibility of recovery has not yet been demonstrated. The progress of the disease may, however, be arrested, and the child continue for years *in statu quo*.

Hydrocephalus in adults is a very rare, chronic, and apparently idiopathic disease. Its cause again is assumed to be a chronic inflammation of the ventricular ependyma. The symptoms are sometimes very like those of a tumor of the brain, and sometimes there is a remarkable absence of characteristic cerebral disturbance, except that spastic paralysis of the extremities (compare page 663) is gradually developed.

Diagnosis.—A pronounced case of congenital hydrocephalus can be recognized without difficulty, inasmuch as the excessive size of the head betrays the disease upon the first glance. Less extreme cases may indeed be somewhat obscure, and we have especially to avoid confounding the condition in question with rachitic enlargement of the skull. We should always, therefore, take into consideration the intellectual powers, the presence or absence of motor disturbances, and other similar symptoms, as well as the cranial peculiarities. In the hydrocephalus of adults there is often no enlargement whatever, so that a diagnosis can hardly ever be declared positively.

Treatment.—Thus far, no remedy has been applied with success in chronic hydrocephalus. The following may be tried: Applications of mercurial ointment and of tincture of iodine to the scalp, methodical compression of the skull, and iodide of potassium internally. The hydrocephalic fluid has often been drawn off, to a certain extent, by tapping, but with merely temporary benefit, if any.

Hence we confine ourselves chiefly to hygienic and symptomatic treatment.

CHAPTER XI.

MÉNIÈRE'S DISEASE.

(*Vertigo ab aure læsa. Labyrinthine Vertigo.*)

In 1861, Ménière, a French physician, first called attention to a peculiar affection which sometimes results from chronic aural disease and is characterized mainly by excessive vertigo and loud tinnitus aurium. At first the symptoms appear in distinct paroxysms. These are ushered in by a shrill ringing in the ears, which is often compared to the whistling of a locomotive, and which is perceived in but one ear. At the same time, or shortly after, comes on a very pronounced dizziness, of a unique sort. The patient has a feeling as if his whole body were moving, as if he were falling forward or were whirling around. Consciousness is unimpaired, but the patient feels very bad, the skin is pale and cool, and the face is bathed in cold perspiration. Frequently there is vomiting toward the close of the attack. The first paroxysms are of brief duration.

As the disease progresses the attacks become more and more frequent, and at last the vertigo may be constant, being extremely annoying to the patient and perhaps confining him to bed. Even now there are occasional paroxysmal exacerbations of the disorder, usually ushered in by the shrill tinnitus. The tokens of chronic aural disease on one side, or less frequently on both sides, also persist. Sometimes there is purulent otorrhœa; often the aural speculum reveals lesions of the drum or of the middle ear; and almost invariably there is more or less deafness on the affected side. This condition may persist for years, until finally it ceases of its own accord, after the deafness on that side becomes complete.

We possess scanty information as to the origin of these subjective phenomena. That they are due to a disease of the internal ear (labyrinth) can scarcely be doubted; and it is further probable that in every case the semicircular canals are involved. Numerous experimental investigations have demonstrated that these last-named structures bear a part in maintaining the equilibrium of the body. Acquaintance with this variety of vertigo is valuable to a nervous specialist, inasmuch as Ménière's disease has been more than once confounded with epilepsy and disease of the cerebellum and other parts of the brain.

Treatment is not wholly unavailing. Charcot has discovered that the persistent use of quinine almost always gives great relief, and may even completely cure the disease. Eight to fifteen grains (grm. 0·5–1) of quinine should be given daily, in two or three doses, and continued for at least several weeks. The particulars of such special treatment of the ear as may be necessary must be sought elsewhere.

VI.—Neuroses without known Anatomical Basis.

CHAPTER I.

EPILEPSY.

(*Falling Sickness. Morbus sacer.*)

Ætiology.—Epilepsy is a peculiar disease of rather frequent occurrence, the main symptom of which is paroxysmal loss of consciousness. In typical cases the unconsciousness is associated with violent general convulsions; but there are many anomalous and rudimentary forms of epilepsy without any symptoms of motor irritation. "Genuine epilepsy" is a functional neurosis—that is, with our present means of investigation we can discover no constant objective lesion of the nervous system as its basis. It is, indeed, true that attacks similar to those of true epilepsy not infrequently occur in the course of tumor, syphilis, and other diseases which do present an anatomical lesion; but such attacks are merely symptomatic, and are therefore termed "epileptiform," in distinction from the genuine epileptic paroxysms. [Convulsions may also be due to lead encephalopathy (*vide infra*). —K.]

Of the actual causes of epilepsy we know nothing. We are acquainted only with certain factors which are favorable to the development of the disease, and are to be regarded, therefore, as predisposing or exciting causes. Heredity is decidedly the most important of these. About one third of all cases of epilepsy occur in persons who have inherited a nervous diathesis, and one or more of whose blood-relations have suffered from diseases of the nervous system. It should not be understood that we must find other cases of genuine epilepsy in the family, in order to establish the fact of congenital predisposition. The question is merely whether the ancestors have exhibited a general tendency to nervous disease. The more accurate and careful our investigations, the oftener shall we find among the relatives of the patient instances of nervous trouble—sometimes genuine epilepsy, sometimes insanity, hysteria, or general "nervousness." Of course these "nervous families" present, besides those that are actually ill, others who are more or less peculiar and odd, and yet others who have extraordinary talents, although frequently somewhat ill-balanced. It is said that the children of parents who are related to each other are somewhat predisposed to epilepsy, as well as to other nervous diseases; but certainly this factor is very rarely of importance. Perhaps dipsomania in the parents is somewhat more prejudicial in this regard. It is said to have been repeatedly observed that children begotten while the father was intoxicated became epileptic.

There are other influences which are assumed to have ætiological importance, but whether justly or not is difficult to decide. Alcoholic excesses can seldom act in this way (although epilepsy is said to attack absinthe-drinkers in France quite frequently). Venereal excesses probably have still less importance. It should also be borne in mind that not infrequently excesses in these directions are the result of neurotic tendencies already existing. Syphilis has no direct connection with genuine epilepsy. Epileptiform convulsions may, as we have seen, be symptomatic of syphilis, being due to the cerebral lesion caused (*vide* page 758) by this latter disease. Certain factors may determine the onset of epilepsy, although they can not be said to cause the disease. Such are overexertion of mind or body, repeated emotional disturbance, certain general conditions of the system, such as anæmia or malnutrition, on the one hand, and plethora on the other; and, in particular, acute febrile diseases, such as scarlet fever, measles, and gastritis. [In some cases abnormal putrefactive processes in the small intestines seem to act as the exciting causes of the individual attacks.—K.] Another important point is that the first attack is sometimes brought on by great mental excitement, especially fright. But here, too, it is probable that the terror is merely the exciting cause, a tendency to the disease already pre-existing. We must also be on our guard against confounding genuine epilepsy with the convulsive form of hysteria (*q. v.*), which very frequently develops after fright.

In some instances there is an evident connection between epilepsy and a previous injury to the head, as from a fall, blow, or cut. At a certain interval after the trauma, attacks begin which seem precisely like those of genuine epilepsy. This is known as "traumatic epilepsy." But these are not cases of genuine epilepsy, inasmuch as there is really some anatomical lesion of the cortex cerebri which, in some way as yet unknown, causes irritation of the motor centers of the cortex (*vide infra*). It is often the case that this sort of epileptiform attacks is peculiar in that the convulsions are at first unilateral, or confined to a single limb, corresponding to the seat of the injury in the opposite cerebral hemisphere.

"Reflex epilepsy" remains to be mentioned. This name is applied to cases where each convulsive attack seems to be excited by reflex influence originating in some one part of the body. Most cases have followed traumatic injury of peripheral nervous trunks (retained splinters, or scars), and have ceased upon removal of the exciting cause. Other causes are new growths in the nerves, foreign bodies in the ear, otitis, intestinal parasites, and, apparently, diseases of the female sexual organs. But it seems probable that sufferers from these attacks have had a tendency to disease of the nervous system. We must hesitate to rank reflex epilepsy in the same class with the genuine form. In former treatises "reflex epilepsy" has certainly very often been confused with traumatic-hysterical spasms (*vide infra*, the chapter on traumatic neuroses).

Both the traumatic and the reflex varieties of epilepsy have repeatedly been the object of experimental investigation. Brown-Séquard has shown, by a great number of experiments, that epilepsy can be excited in rabbits by injuries to the medulla, the spinal cord, and the sciatic, as well as other peripheral nerves. A certain time after the operation the animals undergo spontaneous convulsive paroxysms. These occur at frequent intervals for a long time, and may be voluntarily excited at any time by irritation of a certain portion of the skin called the "epileptogenous zone." An interesting observation in this connection has been made by Brown-Séquard, which is that sometimes the progeny of these animals, who have been made epileptic, suffer from spontaneous epilepsy. Westphal induced epilepsy in guinea pigs by blows upon the skull. Immediately after the blow general convulsions occurred, but soon entirely ceased. Afterward, however, there were repeated epileptiform attacks. Westphal thought that the causative lesion in these instances was the minute hæmorrhages which were found in the upper part of the cervical division of the cord and in the medulla.

Further experiments bearing upon the same subject will be discussed later on.

Clinical History.—In describing the symptoms of epilepsy, we shall first consider the various forms of the epileptic paroxysm, and then describe the general course of the disease.

1. The typical epileptic paroxysm is usually described, for the sake of greater clearness, as made up of several stages. First is the prodromal stage, or, according to Galen's expression, still in vogue, the stage of the epileptic aura (*aura*=

breath). Not infrequently, however, there is no aura whatever, the convulsions coming on without warning. But in many cases the prodromata are well marked, and are repeated with remarkable regularity and similarity before each individual attack, although the different cases of epilepsy differ greatly as to the special phenomena of the aura occurring in each.

The best manner in which to distinguish the various forms of aura is according to the nature of the nervous phenomena, whether sensory, motor, vasomotor, or psychical. Of these the most frequent is, beyond a doubt, the sensory. Here we have peculiar paræsthesiæ, beginning in an arm or leg, or perhaps in the region of the heart or stomach, and thence usually "mounting to the head." It is seldom that the peculiar sensation is actually like a "breath" or puff of air. The aura which proceeds from the epigastrium is sometimes associated with a very disagreeable feeling of oppression and anxiety, and often also with nausea and vomiting. The aura may be referred to the nerves of special sense. In certain instances the patient perceives an unpleasant odor, which he likens to some familiar one. An aura of taste also occurs, but it is very rare. An optical aura is much more frequent, consisting in a subjective sensation of color or light (usually a sensation of red appears first), in an apparent increase or diminution of the size of surrounding objects, or finally in actual hallucinations of vision, such as beholding all sorts of human or brute shapes. An auditory aura is not very rare: it produces a sudden feeling of deafness in one ear, or various subjective sounds, like whistling, humming, or roaring.

The motor aura takes the form of mild premonitory contractions, affecting the head, face, arm, or leg. There may be aphasic disturbance at the same time; or we may observe symptoms of irritation of the unstriped muscles (strangling, or a desire to go to stool). Sometimes there are prodromal vasomotor phenomena, where the aura consists in a sensation of cold or warmth, often associated with excessive pallor or redness of the face or hands. An attack may be ushered in by chilliness, perspiration, or palpitation.

Finally, the name of psychical aura is applied to an initial impairment of consciousness, with vertigo, confusion of thought, etc. A particularly frequent form for this to assume is excessive mental uneasiness and excitement.

Various forms of aura are not infrequently seen in combination.

The aura lasts sometimes only a few seconds. It may persist long enough for the patient, who knows from experience what is coming, to lie down or take other precautionary measures (*vide infra*). In some few cases the aura may last hours, and even days. This is especially true of the psychical variety. Sometimes the aura passes away without being succeeded by any true epileptic fit; but it is usually followed by the second stage of the attack—the convulsive stage.

The convulsions almost invariably begin abruptly. Perhaps there is no aura, or only a very brief one, before the patient falls suddenly to the ground, usually on his face, although sometimes on the side or back. Consciousness is entirely suspended. Insensibility is complete, and often the patient sustains severe injury from his fall. Some patients utter a loud "epileptic cry" at the commencement of the attack, but they are already entirely unconscious.

The convulsive stage begins with a brief period of general tonic spasm of the muscles. The head is usually strongly extended, the teeth are pressed firmly together, the trunk is curved backward in opisthotonos, the extremities are extended, and the fingers are clinched over the flexed thumb. Inasmuch as the respiratory muscles participate in the seizure, breathing stops, and the original pallor of the face soon gives place to deep cyanosis. This general tonic convulsion ordinarily is but brief, perhaps fifteen to thirty seconds. It is followed by the second period of the convulsive stage—that of the clonic convulsions. The facial muscles now exhibit the most violent contortions; the eyeballs roll, or occasionally present a conjugate deviation toward one side; the tongue is thrust out and retracted convulsively; the head beats violently against the floor; and the muscles of the arms, legs, and trunk undergo the severest clonic spasms. The pupils are probably contracted for a short time at first, but during the convulsive stage they are widely dilated and do not react at all. The pulse is somewhat accelerated, but not greatly. The temperature is normal, or elevated a small fraction of a degree. The cutaneous reflexes are still suspended directly after an attack; but the tendon reflexes are generally somewhat exaggerated, although sometimes they also are diminished or absent. Not infrequently an involuntary dejection takes place during the fit, or the bladder is emptied; and in men there may be a seminal emission. During these violent convulsions the body is often severely injured. The tongue is frequently bitten. The venous stasis is so extreme that sometimes minute hæmorrhages occur into the conjunctiva, the skin of the face, and other parts.

The convulsive stage usually lasts several minutes. Then the contractions cease, often after a deep, long-drawn sigh; and the patient passes into the third stage, of post-epileptic coma. He lies unconscious, but his respiration grows quiet, and the cyanosis vanishes. Gradually the coma yields to slumber, which may persist for some hours; but some patients remain only a very brief time in this stage, and recover from their attack with surprising rapidity. It is, however, not infrequently the case that for some days after-pains are felt; there are headache, languor, and exhaustion, and mental despondency and irritability. For some time there may be severe pain in the muscles, particularly those of the trunk. There may be slight paresis of one limb or one side of the body after an attack, but this speedily vanishes again in cases of pure epilepsy. In the urine first passed after the seizure is often found a trace of albumen, and perhaps a few hyaline casts. Not infrequently there is also decided polyuria for some time subsequent to the fit.

2. *The Milder, Rudimentary Forms of Epileptic Seizure. Petit Mal.*—Besides these violent paroxysms just delineated ("*grand mal*"), there are very often witnessed in epilepsy milder attacks of so-called "*petit mal.*" Sometimes there is only a transitory dizziness, or slight faintness, or perhaps a brief loss of consciousness, but without accompanying symptoms of motor irritation. These milder attacks may or may not be preceded by an aura. Cases have been seen repeatedly where the patient suddenly pauses in the midst of conversation, card-playing, piano-playing, or other occupation, stares absently for a moment, and then, with equal abruptness, goes on with what he was doing, as if nothing had happened. In other instances the patient pursues his occupation during this brief suspension of consciousness. For example, if seized while upon the street, he walks on mechanically, but takes the wrong turning, or goes into a strange house, when suddenly he comes to himself and wonders to find himself where he is. Cases of "sudden somnolence" are also almost all of them ascribable to epilepsy. There are all sorts of transitional forms between the slight attack of vertigo and the typical epileptic fit. Not infrequently the patient falls down unconscious, but he has only a slight twitching of the face or arm, and in a few minutes is entirely himself again.

[The medico-legal bearings of epilepsy, in its mild as well as in its severe form, are very important, but they can be only alluded to here.]

3. *Epileptoid Conditions.*—Cases of petit mal are generally rudimentary forms of the typical epileptic paroxysm, and consist merely of a simple impairment of consciousness, possibly associated with slight motor symptoms; but in the epileptoid state the character of the typical epileptic attack is almost indistinguishable.

The disturbance is paroxysmal, and it can often be shown to be connected with genuine epileptic seizures, else its undoubted relation to epilepsy would never have been recognized. The greatest practical importance attaches to Samt's "psychical equivalents of epilepsy." These are attacks of mental disturbance, which either immediately succeed a typical epileptic fit ("post-epileptic insanity") or occur independently. The patient is completely deranged, and may do the strangest things—may strip himself, steal, jump into the water, or commit incendiarism. Here the mind may be said to be only clouded, as compared with other instances where there is violent psychical excitement, associated with terror, frightful hallucinations, and resultant maniacal excitement. Not infrequently the patient is led to acts of violence against those about him. In the young the attack may take a peculiar form: the child runs about in a peevish way, collects all sorts of things together, makes strange motions, etc. Almost always the patient, on recovering consciousness, remembers nothing, or almost nothing, of what has happened. Numerous and valuable particulars upon this subject, and a consideration of its great medico-legal importance, must be sought in text-books on insanity.

Another form of epileptoid attack is the epileptoid sweating of Emminghaus a spontaneous outbreak of excessive perspiration in epileptics, which may or may not be associated with impairment of consciousness.

General Course of the Disease.—In a large majority of cases epilepsy begins before the thirtieth year. Often the disease appears in early youth, and sometimes in even the earliest years. Many a child has "convulsions from teething," which later on are seen to have been epileptic. It is only in rare instances that the first appearance of trouble occurs in advanced life.

It is impossible to give any general rule as to the frequency of the paroxysms. Different cases differ very much. There are persons who during their whole life have no more than three or four seizures, at intervals of ten or fifteen years, while in most cases there is an attack every two to eight weeks. In severe types the fits may even recur daily. One very often sees certain variations in the course of the disease; at some periods the intervals between the attacks will be longer, and at others shorter. In severe cases the patient may have for several days very frequent seizures, so that he does not regain consciousness at all between them. This is termed the epileptic state (*état de mal, status epilepticus*). The condition is quite rare. It is often fatal, death being ushered in by a great rise of temperature.

External influences sometimes affect the frequency of epileptic attacks. Alcoholic or sexual excess, mental excitement, and physical overexertion almost always exert a malign influence. An opposite effect is often experienced where a quiet life is led, with every attention paid to hygiene and pure air. In women, the appearance of the catamenia is not infrequently the signal for the occurrence of an attack. In many instances the disease begins with the first establishment of menstruation. Sometimes, however, epileptic girls grow better when they arrive at puberty. Pregnancy sometimes increases and sometimes diminishes the frequency of the paroxysms. Intercurrent diseases seem frequently to exert a beneficial influence upon the frequency of the attacks.

There is a practical distinction between diurnal and nocturnal epilepsy. Many patients have attacks only during the day, while others again have them only at night. A case of purely nocturnal epilepsy may go on for a long while unsuspected, particularly if the patient sleep alone. He seldom has, on the next morning, the slightest recollection of his attack during the night. He usually perceives, however, from a confused feeling in his head, or from certain injuries such as a bitten tongue, or from the disordered state of the bed, that something must have happened to him during the night. In some cases of nocturnal epilepsy the patient wakes up out of sleep before he enters into the epileptic state of unconsciousness. Probably he is aroused by the aura. Besides cases where the fits occur during the day or the night only, mixed forms are frequently seen.

With regard to the occurrence of the different varieties of epileptic seizure, all sorts of combinations are possible. Many cases never have any but the typical convulsions; but in many others there are also a greater or less number of attacks of petit mal. The latter may even be for a long period the sole indication of the disease. Often there are no epileptoid conditions whatever, while in other instances the "psychical equivalents" are the most prominent feature of the disorder.

During the interval between the individual attacks many epileptics seem perfectly well, both physically and mentally. They are not infrequently, to be sure, somewhat peculiar and nervously excitable, or again dull and lethargic; but this does not by any means apply to them all. Many epileptics, and particularly such as have comparatively infrequent paroxysms, are very capable; and history furnishes numerous examples of eminent men who suffered from this disease—for instance, Cæsar, Mahomet, Rousseau, and Napoleon I.

Much effort has been devoted to the discovery of "signs of physical degeneracy" in epileptics. Relying upon numerous measurements, Benedikt believes that a majority of epileptics exhibit craniometric anomalies, such as asymmetry of the cranium, macrocephalia, or steepness of the vertex. It is also not unusual to meet with anomalies of the auricles, teeth, or hands* in such cases. And, indeed, all peculiarities of this sort are, in general, more frequently observed in neuropathic families than in healthy ones.

When the disease has lasted some time, and particularly if the attacks come at very short intervals, the general condition of the patient often undergoes a gradual but marked change. This rule is by no means invariable. The mind becomes more and more affected; the intellect grows feeble; memory grows weaker, and occasionally there is at last dementia. In such cases the body also suffers. There are emaciation, paresis, tremor, and other persistent disturbances of cerebral origin.

Duration.—Epilepsy must be termed a life-long disease. To be sure, it is no rare thing for the paroxysms to cease and not return for years. But one can never rest satisfied that all trouble is at an end; some cause or other may excite another attack after a long interval. In general, an epileptic has a shorter life-expectancy than healthy persons, especially as he may be carried off by chronic pulmonary or other intercurrent disease.

The prognosis is obvious from what has been already said. The individual seizure is only exceptionally dangerous of itself. Often the so-called "*status epilepticus*" ends fatally, as above stated. In general, those cases may be called the most favorable where the separate paroxysms are infrequent and mild; but even here the disease may suddenly assume an aggravated form. With regard to the distinction between nocturnal and diurnal epilepsy, the nocturnal is, in our opinion, the milder of the two.

Pathology.—The very fact that in the intervals between attacks the patient often betrays no sign of disease, shows that epilepsy can not be due to any persistent macroscopic lesion of the tissues. Indeed, in many cases nothing is found at the autopsy, or, at most, changes which can not be regarded as essential, such as osteosclerosis of the cranium or thickening of the cerebral meninges. Epileptic subjects who were during life decidedly demented usually present atrophy of the hemispheres. Meynert states that changes in the pes hippocampi major are noticeably frequent in epilepsy; but these changes are not at all constant, and their significance remains to be established.

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* We have lately seen an epileptic man who had six fingers on each hand.

We must therefore, for the present, be content to assume that the cause of the epileptic seizure is an intermittent functional condition of irritation. A natural question is, Where shall we locate this irritation, and what may be its nature? The opinion was long current that the medulla oblongata must be regarded as the true seat of the disease. Schröder van der Kolk was the first to express this opinion. It afterward received support from the experimental investigations of Nothnagel, who demonstrated that irritation of a particular spot ("convulsive center") in the pons, in rabbits, invariably excites general convulsions. Nevertheless, most pathologists have now abandoned this view, because experiment and clinical observation indicate with increasing distinctness that the origin of epileptic convulsions is to be sought in the cortex cerebri. The clinical evidence is the invariable combination of convulsions and impairment of consciousness; the circumstance that the milder and the masked forms of epilepsy, now known to be intimately related to the true epileptic convulsions, also, almost without exception, indicate psychical disturbance; that attacks, the symptoms of which are perfectly analogous with those of epilepsy, are often found to be the result of anatomical lesions of the cerebral cortex; and, finally, that these convulsions in man and the convulsions of "cortical epilepsy" (*vide infra*), experimentally produced in animals, extend over the different groups of muscles in a way which corresponds precisely with the anatomical position of the different motor centers in the cortex (Hughlings Jackson). For example, if the convulsion begins in the distribution of the facial nerve, it extends from this point to the arm before it affects the leg.

There is also experimental evidence that epileptic paroxysms are of cortical origin. A great number of observers (Hitzig, Ferrier, Albertoni, Luciani, Franck, and Pitres) have proved that electrical irritation of the motor regions of the cortex in animals will produce epileptiform convulsions. Unverricht has made some of the latest and most thorough investigations in regard to this point upon dogs. He found that when a motor center is stimulated the convulsions spread from the corresponding group of muscles to others in a way which corresponds precisely to the anatomical position of the separate centers. If one of the centers in the cortex be destroyed, the convulsions of the corresponding muscles cease at once. This proves that the motor centers must be intact in order to render the occurrence of epileptic seizures possible. Just how the stimulation extends from one center to another we have as yet no certain information. Probably it travels horizontally through the cortex.

We see, therefore, that in all probability the seizures in man also originate in the cortex of the brain. The phenomena of the aura are likewise referable to some stimulation of the cortex, probably of the sensory region in most cases, as in the optical aura. The starting point of the irritation is probably usually in the motor cortex, but it does not seem impossible that an irritation developing in the posterior cortical regions may extend secondarily to the anterior motor portion of the cortex (Unverricht). The manner in which the irritation is created is as yet entirely conjectural. Kussmaul and Tenner proved that epileptiform convulsions can be excited by a general cerebral anæmia; and this fact was the main foundation for the assumption that the genuine epileptic convulsions are also due to a temporary cerebral anæmia, caused, it may be, by local vasomotor constriction. Definite proof has not yet been furnished on this point. In the artificial epilepsy which Unverricht produced, and that also which Magnan excited by absinthe, the cortex of the brain was not strikingly anæmic.

Diagnosis.—Most cases of epilepsy are easily recognizable. It needs only to be borne in mind that epileptiform convulsions may also occur as a symptom of cerebral diseases which do have an anatomical basis, such as tumor, abscess, multiple sclerosis, and hydatids. As a rule, however, such diseases are readily distinguished by the state of the patient between the seizures, or by the further course of the illness. It should also be understood that unilateral convulsions, or such as are confined to a single member (Jacksonian epilepsy, *vide supra*, page 714), are usually not true epilepsy, but symptoms of some circumscribed affection of the cortex. The differentiation from hysterical convulsions (*q. v.*) is seldom difficult. Weight should be laid on the general character of the attack, the complete loss of consciousness in epilepsy, the dilatation of the pupils, which do not react to light, the initial pallor not infrequently observed, and the late cyanosis of the face. It is mainly the same factors which will enable us to detect simulated epilepsy. Here, also, there is an absence of those bodily injuries, such as a bitten tongue, which are often so characteristic of the genuine disease.

Treatment.—Although no remedy is capable of working a certain and permanent cure of epilepsy, yet a favorable influence can be exerted upon the disease in many ways, so as to lessen the frequency and severity of the paroxysms, and to avert many of their evil results.

In the first place, regimen is of great importance. Any excessive exertion of mind or body must be forbidden. Temperance must be exercised in eating and drinking. Alcohol, strong coffee, and tea, can be used only moderately; nor is too much smoking permissible. The diet should be simple and unirritating, and vegetable rather than animal. It is said that in some cases decided improvement has been brought about by confining the patient to milk and vegetables. In summer the patient should live quietly in the country or the mountains. We have also the individual constitution to consider: a weak and anæmic person must have iron and abundant nourishment; and a full-blooded, corpulent individual should drink the natural aperient waters, and live abstemiously.

Proceeding to the treatment of the disease itself, we shall rarely find any cause to remove; although, in a few cases of reflex epilepsy, the excision of old scars, the extraction of foreign bodies, or trephining the skull, where the disease has followed an injury, have brought about permanent recovery. In genuine typical epilepsy we have no such indications to fulfill, and we must have recourse to such treatment as experience shows can influence favorably the outward manifestations of the disease. [In Jacksonian epilepsy the cortical center of the initial spasm of the convulsion has been excised a number of times, even when, after trephining, it appeared healthy. The center should be carefully determined, after the brain is exposed, by faradic stimulation of the cortex. In some cases relief has been obtained, and the convulsions have ceased for a considerable period. It is too early to speak of the ultimate results, but in some cases the attacks have finally recurred in equal severity.—K.]

Among symptomatic remedies, potassic bromide has an undisputed right to the first place. It was first recommended by Locock in the year 1853. It should be the first thing tried in any severe case. Apparently it acts by directly lowering the sensitiveness of the motor centers of the cortex to irritation. Rather large doses are requisite. Beginning with about one drachm (grm. 4–5) a day, we may find it advisable to increase up to two or two and one half drachms (grm. 8–10). It may be prescribed in water (1 to 10 or 15), or in powders which the patient himself is to dissolve in water, sweetened if desired. The remedy in almost all cases needs to be used for months and years, and it is therefore often advisable for the patient to buy a half pound or a pound and weigh it out himself into the proper doses. It should always be taken in a good deal of water, say half a tumbler or more, to avoid irritating the stomach. The total amount for the day is usually divided into two or three portions; but the whole may be dissolved in a large amount of water and drunk gradually through the day. The bromides of sodium and ammonium are also frequently employed. They have the advantage of disturbing the stomach less than does the potassium salt. It is a good way to combine the different bromides—for instance, bromide of sodium and bromide of ammonium, of each 10 parts; distilled water, 200 parts: three tablespoonfuls a day in water. It is well to combine the various bromides. Erlenmeyer strongly recommends a mixture of two parts of potassic bromide with one each of sodic and of ammonic bromide.

In using the bromides, persistence is necessary for at least months, and often, with occasional interruptions, for years, if benefit is to be obtained. In case there are unpleasant symptoms due to the remedy, such as excessive acne, muscular lassitude and tremor, cardiac weakness, dyspepsia, impotence, or melancholy, we must diminish the dose, or even omit the medicine for a time. Many patients are greatly annoyed by pustules due to the bromide; this can sometimes be avoided by giving Fowler's solution at the same time. If the attacks are decidedly abated, the dose may be gradually diminished, to be increased again, however, if there be any tendency to a relapse.

Recourse to other remedies is seldom had, unless potassic bromide fails, or for some cause must be discontinued. We may then try valerian in powder, 8–30 gr. (grm. 0·5–2) several times a day, or as an infusion. It is a very good plan to give patients who are taking bromide a cup or two of valerian tea at bedtime. Belladonna may also be exhibited, or a pill of atropine, gr. $\frac{1}{120}$ (grm. 0·0005), three to five times a day; or zinc oxide in the dose of one to three grains (grm. 0·05–0·20), perhaps combined with a grain of extract of belladonna, and fifteen grains of valerian, as a powder, three times a day. There are also many other remedies of doubtful efficacy: curare, hyoscine, the root of artemisia vulgaris, ammonio-cupric sulphate, nitrate of silver, and arsenic.

Electricity is apparently beneficial in occasional instances, and may be tried in connection with other remedies. The galvanic current should be cautiously applied to the head and the sympathetic nerves. Still greater benefit is sometimes obtained from a carefully conducted cold-water cure. Cold sponging with friction at night helps most cases, and it is sometimes very advisable to send the patient in summer to some appropriate establishment for cold-water treatment.

Treatment during the Paroxysm.—In most cases we can do little during the seizure except to take such precautionary measures as common sense suggests. We possess no means of suppressing an attack when once under way; nor, indeed, is it often dangerous. In individual instances the patient finds out from experience some method to cut short the paroxysm during the aura. For instance, there are cases where tightly bandaging or vigorously rubbing the part in which the aura originates will avert the convulsions. A number of cases have also been known where the ingestion of a generous quantity of common salt during the aura (usually in these instances starting from the epigastrium) has had the same effect. A patient of our own, whose attacks began with a feeling of rectal tenesmus, maintained that she could almost invariably suppress the convulsions by promptly going to stool, if she had time and opportunity. It was formerly a frequent manœuvre to seek to ward off the attack by compressing the carotids; but this usually fails. Berger recommends the inhalation of nitrite of amyl at the commencement of the fit, having repeatedly seen benefit follow its use.

In the "status epilepticus," narcotics are the most deserving of trial, and in particular chloroform or ether given by inhalation. Amyl nitrite may also be of service.

[It is sometimes desirable to withhold the knowledge that he is an epileptic from the patient, whose ordinary life should be interfered with as little as is possible.

Especially in cases characterized by headache and heat in the head, Brown-Séquard finds the application of ice directly to the back of the neck and between the shoulders useful.]

APPENDIX.

INFANTILE CONVULSIONS (ECLAMPSIA INFANTUM).

Convulsions in childhood are of such frequency and importance as to justify brief special mention here.

Every practitioner learns from experience that the young are especially predisposed to convulsions. Probably this is partly due to excessive reflex excitability of the brain in childhood. Thus children not infrequently undergo convulsions under circumstances in which adults would very rarely have them. They sometimes are seen in children in the beginning of acute febrile diseases, such as pneumonia, scarlet fever, and measles. They also occur from indigestion, particularly when the stomach has been overloaded; sometimes on account of teething; or because of intestinal worms. Here they are in all probability of reflex origin.

Convulsions may occur in very early life without ascertainable cause. In many cases they are really the commencement of epilepsy, as is seen afterward. Again, they may be due to some actual lesion in the brain. For example, if one recalls the initial stage of the acute poliomyelitis and acute encephalitis of children (*vide* pages 668 and 744), it will not seem unlikely that many rapidly fatal cases of "convulsions" are really instances of the diseases mentioned. This point has not yet been at all satisfactorily investigated by pathologists. At any rate, it does not seem satisfactory to us to regard the "œdema of the meninges" found in such cases as an independent disease and the sufficient cause of death. Often convulsions occur suddenly in children and then cease, never to recur, without our being able to find any explanation of the attack. Experience shows that rachitic children are especially liable to suffer from eclampsia—possibly because of cranial rachitic changes (?).

The symptoms of the eclamptic attacks are on the whole analogous with those of epileptic paroxysms. The child's eyes become staring and fixed, and there are tonic and clonic spasms of the face, trunk, and extremities. Such seizures may continue for days with brief intermissions. In such cases the prognosis is dubious, particularly if the child be weakly, but it is by no means absolutely bad. The cause and the significance of the convulsions can seldom be determined immediately.

Symptomatic treatment consists in applying cold to the head, wet packs, sinapisms on the chest and the calves of the legs, and perhaps an enema (to which vinegar may be added). These measures generally answer for mild cases. If the fits are very frequent and violent, we may allow even small children to inhale chloroform, often with great advantage. A dessertspoonful is to be poured upon a handkerchief and administered cautiously.

Of course, we must also try to discover and remove the cause. The attacks due to overloading the stomach usually occur in not very young children, and are apt to be greatly benefited by a prompt emetic or purge.

[A bath at a temperature of 90°–95°, while cool water is applied to the head, seems often to be of service; if the child be exhausted by diarrhœa, the cold to the head should be omitted. In a teething child it can do no harm, and it sometimes has a very marked beneficial effect to lance the gums thoroughly. Enemas containing chloral with or without bromide of potash are more used in this country than is chloroform by inhalation.

A drop of nitrite of amyl by inhalation is said by Eustace Smith to exert a controlling effect on the muscular movements.]

CHAPTER II.

CHOREA.

(*Chorea Minor. St. Vitus's Dance.*)

Ætiology.—Centuries ago the name *chorea* (dance) was applied mainly to those strange states of "dancing mania" which were endemic in certain places, being due to excessive mental excitement and to the innate propensity to imitation. The specific for this condition was held to be a pilgrimage to some shrine of St. Vitus. At the present time, however, chorea is used to designate a perfectly definite disease, of which the characteristic symptom is the appearance of certain peculiar motor phenomena due to irritation of the nervous centers. It is sometimes called *chorea minor*, in contradistinction from what was formerly termed *chorea major* or *magna*. This latter is, however, not a genuine, independent disease, but a manifestation of hysteria (*q. v.*), or apparently in many instances of epilepsy.

Chorea proper is mainly a disease of children. It occurs most often between the fifth and fifteenth years, although it may be seen both earlier and later. There is a slightly greater liability to it in girls than in boys. Hereditary neuropathic tendencies are also a factor in its ætiology, but not a very important one.*

As to causation, in many cases nothing definite can be made out. Mental excitement, as from fright, seems in some few instances to favor the onset of the trouble. It is also certain that the imitative impulse will often lead to choreic movements in healthy children who come in contact with choreic patients, but it is doubtful whether this "imitative chorea" can be regarded as true chorea. There is a very interesting connection between chorea and acute articular rheumatism. Although the statement of certain authors, that almost every case of acute articular rheumatism in children is followed by chorea, is far too strong, yet this sequence is comparatively frequent. Chorea is sometimes seen also in children who have a mild form of chronic rheumatism, or in such as have valvular cardiac disease, whether preceded by articular rheumatism or not. [Some observers claim that there is an increased amount of uric acid in the urine of patients with chorea, the acid diminishing as the chorea subsides.—K.] Here chorea is seen as a sequel to an infectious disease; perhaps this is a hint of the light in which we should view apparently idiopathic cases of chorea. [Lewis, having studied 1,383 attacks of chorea and 673 of acute articular rheumatism, finds that the fewest attacks of chorea occur in October and November, the greatest number in March and April. The greatest number of attacks of rheumatism occur in April, the smallest in the autumn. The frequency of attacks of both diseases seems related to the number of storm-centers which passed within four hundred miles of the localities studied, and to the mean actual barometer and mean relative humidity. Over-study, acting with meteorological conditions, may be a predisposing but not an exciting cause of chorea.—K.]

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* The so-called hereditary or Huntington's chorea is a special disease. Choreic movements appear in many members of the same family in several generations, and at an advanced age, from thirty to forty. The disease is incurable, and often leads to mental deterioration.

Women are particularly liable to chorea during pregnancy. *Chorea gravidarum* is most frequent in youthful primiparæ.

Clinical History.—Chorea usually begins gradually, and without any special prodromata. Sometimes, however, there are prodromal symptoms, chiefly a certain mental depression and irritability, with indisposition to intellectual effort. There may be slight rheumatic pains or anorexia, and other evidences of constitutional disturbance.

Ordinarily, the peculiar motor disturbances are the first thing to attract the attention of the patient or its parents. There are involuntary and irrepressible movements in the most diverse groups of muscles. Both single contractions and also complicated movements occur, independently of the will, and in all parts of the body, now in one place, now in another, sometimes in a single member, and sometimes in several at once. The movements may be made in rapid succession, or may be separated by long intervals of quiet. The facial muscles may be involved, causing an occasional wrinkling of the brow or distortion of the mouth. The eyes or the eyelids may also exhibit involuntary movements. The pupils are frequently dilated. If the patient be asked to protrude his tongue and keep it quiet, it will often be involuntarily withdrawn into the mouth or thrust to one side. The tongue may even be sufficiently affected to impair speech. The laryngeal muscles have also been observed to make choreic movements. The arms are frequently the most affected of any part; they are twisted, flexed, elevated, put behind the back—in short, moved in every conceivable way. The trunk is generally but little disturbed in the milder cases, but in severe ones the whole body participates. The patient stands up, lies down again, turns upon his side, etc. The legs are seldom as much disturbed as the arms and face, but slight movements of the lower limbs are very frequent—the foot is thrust forward or extended, the knee is flexed, and so on. In general, it may be said to be characteristic of chorea that the abnormal motor irritation usually affects a considerable number of muscles simultaneously, thus exciting all sorts of combined movements; and, secondly, that choreic movements, for the most part, are not short twitches, but take place in a manner decidedly similar to that of voluntary movements.

The vigor of the movements varies greatly in different cases. At first they may be too slight for the unpracticed eye to catch. Many children in an incipient stage of chorea are unjustly punished at school because they write ill or do not sit quietly. Many cases are mild throughout, never having very severe disturbance. Others, though considerably annoyed, can nevertheless walk or stand alone. In the severest cases, however, the whole body is continually in active motion. The patient throws himself about in bed, and all the extremities exhibit constant violent and irregular movements. The ingestion of food is extremely difficult, sleep is disturbed, and the patient's flesh and strength are rapidly and greatly diminished.

Further, each individual case presents variations in severity at different times. If the patient be left quietly to himself, the contractions are comparatively very slight. As soon as he is conscious of being watched or as soon as any one speaks to him, his condition usually becomes much worse. During sleep the choreic movements cease altogether.

In many cases all the voluntary muscles are involved; but sometimes the disease is limited to certain groups of muscles. Very frequently the disturbance is mainly unilateral (hemichorea); the opposite side of the body then exhibits few involuntary movements, or, it may be, none. As already stated, the face and upper extremities are often more affected than the trunk and lower limbs.

These motor disturbances often constitute the sole or the predominant symptom of chorea. There is hardly ever muscular weakness or paralysis. It is remarkable how little feeling of fatigue there usually is, despite the incessant activity. In a few cases only, of genuine chorea, have we seen actual paresis, affecting, for instance, one arm, or in hemichorea the same half of the body. Sensation is unimpaired. The reflexes do not exhibit striking peculiarities. The tendon reflexes we have sometimes found to be noticeably diminished, although in other instances they were normal. There may be isolated spots in the spinal column tender on pressure, but this is not at all constant. That chorea may be complicated by arthritis and valvular cardiac disease has already been stated. Some caution should be exercised in making a diagnosis of cardiac lesion here, for experience shows that choreic patients are apt to have functional murmurs and slightly irregular cardiac action. [Osler has found that the endocarditis of chorea is very apt to lead to organic valvular disease. Of 110 choreic patients that he examined more than two years after the attack, 54 presented signs of organic heart disease.—K.] The temperature is not elevated, in spite of the constant muscular contractions, nor is the amount of urea excreted by the kidneys increased.

Slight mental disturbance is frequently observed. The patient is often rude, peevish, capricious, incapable of mental exertion, irritable, and inclined to tears; but any great or permanent impairment of intellect is very exceptional indeed.

The entire process generally occupies several months. In mild cases, however, recovery may ensue at the end of a few weeks, while, on the other hand, cases may last a year or even longer. Variations in the intensity of the chorea are often witnessed. These are sometimes spontaneous, and sometimes are due to outward influences. Even when the disease is apparently extinguished, we must be prepared for a possible relapse. The disease may appear repeatedly in the course of a few years, in such a way that it is not easy to determine whether the different attacks are relapses or new illnesses. The protracted cases are, as a rule, comparatively mild; and many cases that begin with great violence end comparatively early. In adults, however, we have met with some rather severe cases which were very chronic, and seemed at last to become stationary.

The termination of chorea, in the great majority of cases, is favorable. Now and then severe cases do occur which end in death. In these the choreic movements are extremely violent. The patient is tossed about in his bed, and can eat little and sleep none. We have ourselves observed three such cases, in girls fourteen to seventeen years of age, which proved fatal within the first two or three weeks. Two died from general exhaustion and collapse, and the third from gangrene affecting numerous cutaneous traumatic lesions, which had occurred despite every possible precaution.

Nature of the Disease.—All cases of genuine chorea thus far examined by pathologists have failed to furnish any lesions which can be regarded as essential. In the three cases above mentioned the autopsy revealed absolutely nothing abnormal in the central nervous system. We are at present, therefore, obliged to classify chorea as a "neurosis"—that is, as a disease that produces functional disturbances, for which latter there is no anatomical basis known to us. The symptoms themselves show that the disorder must affect principally some motor region of the nervous system; but just which motor region is involved can at present only be conjectured. It seems very probable, however, that the true seat of chorea is to be sought in the brain. In the first place, the frequent occurrence of hemichorea would indicate this; as would also the fact that slight mental anomalies are frequently combined with chorea; and, finally, "choreiform" movements may occur as the sole symptom of undoubted cerebral disease, as in post-hemiplegic hemichorea. We have, however, no hint as to whether the motor regions involved are those located in the cortex, or others. The surmise has quite often been expressed that chorea is due to embolism of a mild type; but, in our opinion, this view lacks proof entirely, and is even improbable. As already said, it may be that the connection existing between chorea and acute articular rheumatism will throw some light upon the nature of the former disease.

[Money has produced choreic movements in dogs by the injection of starch into the carotids, thus causing embolism of minute cerebral vessels.]

Diagnosis.—The diagnosis is almost always easy, and it can often be made at a glance. The motor symptoms of athetosis, paralysis agitans, and of alcoholic, senile, saturnine, and mercurial tremor are so characteristic as to be readily distinguished from the movements of chorea. It is not difficult to perceive the difference between genuine idiopathic chorea and the symptomatic choreiform movements occasioned by some other cerebral lesion.

Prognosis.—As has been stated, the prognosis is almost invariably favorable, although the disease may prove very tedious. The possibility of relapses should be borne in mind. The prognosis is doubtful only in the worst cases of acute chorea, where there is great and rapid failure of the general health.

Treatment.—Even in mild cases the patient must be kept from school and at home, in order to avoid all unnecessary excitement from ridicule and the like. If the chorea be only moderately severe, it is not necessary that the child should be in bed; we may even allow moderate exercise in the open air. Where the motions are violent, we should seek to guard the patient from self-injury by means of pillows and cushions.

Among the remedies recommended for chorea, the chief place is occupied by arsenic and potassic bromide. Arsenic in particular seems often to be of value. We give Fowler's solution in water; beginning with five drops, two or three times a day, we gradually increase to eight or ten drops. [In severe cases much larger doses may be given. It should be pushed up to twenty drops or more, stopping for twenty-four hours if symptoms of poisoning appear, and beginning again at the same dose. It should be freely diluted.—K.] In children under six, the dose should be made somewhat smaller. If the child be anæmic, iron may be given in addition; or, if there be great restlessness and loss of sleep, narcotics may also be administered. Antipyrine, in doses of seven to fifteen grains (grm. 0·5–1·0) several times a day, is sometimes of distinct service in severe cases. We may also, especially if arsenic fail, try bromide of potassium in large doses, a drachm (grm. 3–5) a day. Numerous other drugs have been recommended: oxide of zinc, valerianate of zinc, nitrate of silver, and sulphate of copper. At present they are very rarely used; but Riess has lately recommended physostigmine, one sixth to one third of a grain a day (grm. 0·001–0·002), best subcutaneously. [Sulfonal, three to five grains (grm. 0·2–0·4) three times a day, acts well, as an adjuvant to arsenic, in cases where there is much excitement. Exalgine, two or three grains (grm. 0·1–0·2) three times a day, has recently been recommended.—K.] If the disease occur as a sequel of articular rheumatism, we may try salicylic acid. Narcotics should be employed very cautiously in chorea. Although chloral has been recently recommended for grave cases, there are instances known where this remedy has been followed by unfortunate results.

Hydrotherapeutics of a mild kind do good, and can easily be carried out in most instances. Thus, we may use lukewarm baths, a wet pack, or gentle sponging with water at 72°–82° (18°–22° R.) to great advantage.

Electricity may also be tried. A feeble current of galvanic electricity is applied to the head (in the region of the motor centers), or the spinal cord is galvanized. If there be points along the spinal column where pressure causes pain, it is said to be an excellent plan to apply the anode to them; but the results of electrical treatment are seldom very brilliant.

In the chorea of pregnancy, which sometimes is a very violent disease, the same remedies may be employed. If they do no good, artificial delivery may be required; after which, as we have ourselves once observed, there may be a rapid abatement of all symptoms.

CHAPTER III.

PARALYSIS AGITANS.

(*Shaking Palsy. Parkinson's Disease.*)

Ætiology.—In 1817 Parkinson described for the first time a disease which he named the "shaking palsy." It is not of very frequent occurrence, and as yet little has been ascertained with regard to its ætiology. In most cases it develops very gradually, without any demonstrable cause. It almost always attacks elderly persons, being very rare before the thirty-fifth year. Sex does not seem to exert any great predisposing influence. Hereditary neuropathic tendencies can indeed be traced in some instances, but are certainly of less potency in paralysis agitans than, for instance, in epilepsy. Special exciting causes have sometimes been observed, such as catching cold, violent emotional excitement, and traumatic influences, among the latter injury to the nerves, burns, etc. Berger reports two cases where the first symptoms appeared after an acute febrile disease (typhoid fever).

Clinical History.—Paralysis agitans has two characteristic symptoms, viz., (1) peculiar evidences of motor irritation, evinced by tremor, and (2) a condition of stiffness and persistent shortening of certain muscles, consequent upon which is a series of peculiar motor phenomena.

The trembling is generally the earliest symptom to attract the patient's attention. It usually begins in the hands, especially in the right hand, and then gradually involves the arm and leg on the same side, next the other arm and leg, and finally, in well-marked cases, the entire body. The form of tremor is very characteristic. There are rapid, uniform, oscillatory movements of varying extent. The tremor is usually greatest in the hands and arms. At the same time the thumbs and half-flexed fingers exhibit a movement which suggests spinning or pill-rolling. The forearm is generally flexed and extended in rapid alternation, but it is always very difficult to determine just what muscles contract. With regard to the trunk, it is often a question whether its tremor is of independent origin, or due merely to the motion of the extremities. Charcot states that the head and the facial muscles are never implicated, but there is doubt about this point. We ourselves, as well as other observers, have repeatedly seen independent tremor of the head. As to the face, the muscles about the chin seem to suffer chiefly.

The trembling of paralysis agitans is almost continuous. It may, indeed, cease for a moment in a limb, but only to recur immediately. The quieter the patient is, in mind and body, the less violent are the movements. If he is excited, or begins to speak, or is watched, the tremor at once becomes exaggerated, and may be violent enough to jar the whole body vehemently. Active motion does not intensify the tremor. On the contrary, it may often be observed that the trembling abates when the muscles undergo vigorous voluntary contraction, as when a weight is lifted, or the hand of another is firmly grasped.

The second symptom is almost more characteristic than the first. It consists of a peculiar rigidity of the muscles. We generally notice, even in the face, peculiar tension of the muscles. This often produces a stolidity of expression, so that the emotions are less clearly depicted than upon the countenance of a healthy person. The head gradually becomes more and more flexed. When the disease has lasted some years, the chin may even rest upon the sternum. The muscles of the trunk and extremities also stiffen gradually, and lead to peculiar and almost pathognomonic appearances. The body is bent over forward; the arms cling to the trunk, and are flexed at the elbow-joint; the fingers are flexed, especially at the metacarpo-phalangeal joint; the thumbs rest against the fingers, as if holding a pen, or else are flexed inward upon the palm; and the legs are somewhat bent at the knee. The accompanying picture (Fig. 107) is from the photograph of a patient who was for a long time under observation at the clinique in Leipsic, and gives a good representation of the characteristic posture.

The stiffness of the muscles also operates to impair motion in various ways. In particular all movements of the trunk are greatly impeded. In advanced cases the patient can not get upon his feet, if he is lying in bed, without help. Inasmuch, however, as the muscular strength usually remains good (*vide infra*), he requires to exert but a slight traction upon some helping hand in order to attain an erect posture. On the other hand, the patient is often utterly unable to turn in bed from one side to the other. In severe cases, therefore, it is often necessary to alter the patient's position several times in a night, especially as lying long in one attitude makes him feel very uneasy. If the patient is in a chair, he can not get up of himself, because it is impossible for him to bend his body forward in the necessary manner; but with just a little help he can stand up, and is then able to walk alone or even to run. Then, since the flexion of the trunk forward brings the center of gravity forward also, and the trunk can not be sufficiently bent backward, he is apt to "get a-going," so that he can not stop until he brings up against a post or a wall. If such a patient, with a considerable degree of anteflexion and rigidity of the trunk, is slightly pushed from behind, he will have to start into a run to avoid falling. This phenomenon is termed "propulsion." A push backward, which brings the center of gravity behind the point of support, is very likely to make such a patient fall, as he will seldom succeed in moving backward fast enough to recover his balance (retropulsion). Both propulsion and retropulsion are conceived by Charcot to be "forced movements" (see page 541) in the strict sense of the term. We are, however, convinced, as the result of numerous observations, that these phenomena can in every case be explained simply by the mechanical conditions arising from displacement of the center of gravity. Again, the reason why many patients are prone to keep their arms behind them as they walk is that such a position contributes slightly toward bringing the center of gravity farther backward.

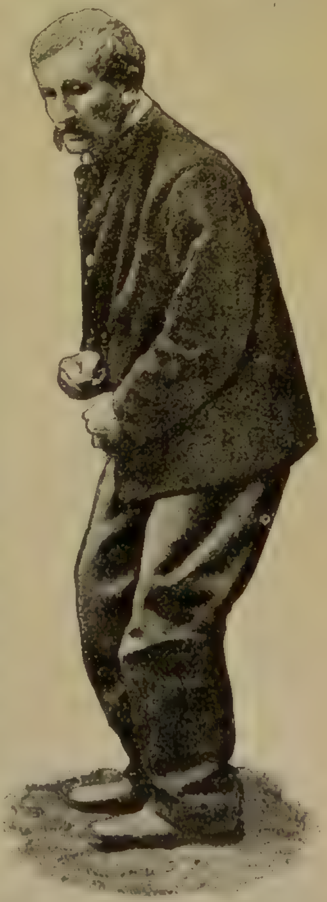
Fig. 107.—Characteristic position of the body in paralysis agitans.

The movements of the extremities are less impaired than those of the trunk, but they often betray a certain slowness and stiffness of motion. The strength of the muscles may be preserved for a long while, but often there is at last evident paresis. Even in the early stages of the disease the muscles may become easily fatigued. The impairment of facial expression has already been referred to. In many cases the muscles of the eye also seem to participate in the rigidity, making it difficult for the eye in reading to follow rapidly along each line, or to pass from the end of one line to the commencement of the next. [The voice is often affected; the patient talks in a high-pitched monotone.—K.]

The muscular rigidity is almost more characteristic of paralysis agitans than is the tremor. Indeed, there would seem to be cases, as we have ourselves observed, where, at least for a time, the posture of the patient is typical, and yet there is no trembling. Such cases might be called *paralysis agitans sine agitatione.* In uncomplicated cases all other nervous functions remain perfectly normal. Sensation is never impaired. Sometimes some pain is felt at the commencement of the disease, particularly in the shoulders. There is no striking disturbance of reflex action nor of the bladder. In a few cases of paralysis agitans, cerebral and mental symptoms have been observed; but they are so rare that it is impossible to say whether they belong to the disease or are merely accidental complications. It is also noteworthy that many patients complain of a subjective feeling of excessive warmth. The internal temperature is normal; but it is said that the temperature of the surface of the body is frequently somewhat elevated. Sometimes there is a tendency to excessive perspiration. [Mossé and Banal have found that the excretion of urea and the total excretion of phosphoric acid is increased in paralysis agitans, but that the excretion of imperfectly oxidized phosphoric acid is diminished, thus showing that there is increased combustion.—K.]

The disease runs a very chronic course, perhaps for twenty years or more. From the first, it keeps on slowly but gradually developing. The symptoms rarely exhibit marked alterations of mildness and severity, but for long periods the progress of the disease may be apparently arrested. Recovery has never yet been observed. The final and fatal termination is not brought about by the disease itself, but is due to some intercurrent affection or to general marasmus. The original of the above picture came to a pitiable end by tumbling face downward into a puddle of water. He could not get up, and was drowned.

Nature of the Disease.—The true nature of the disease is unknown. Inasmuch as the disorder is purely a motor one, the corresponding lesions must be sought somewhere in the motor system. As yet, however, post-mortem examinations of the nervous system, even with the microscope, have revealed no definite changes. We must, therefore, confess that we have even had a doubt whether it is justifiable to claim that paralysis agitans is an affection of the nervous system at all, or whether it may not possibly be of purely muscular origin. Certainly it would not be impossible for abnormal processes in the muscles to excite the tremor and tonic contraction. But, as has been said, there is at present no ground for deciding this question; we would merely suggest it.

[Borgherini has found, at the autopsy of a case of paralysis agitans, an increase in the nuclei of the vessels of the brain and cord, with changes in their caliber, and hyperplasia of the interstitial tissue affecting the nerve-elements, the hyperplasia being most marked in the motor tract. The gray axis and the white matter of the pons, medulla, and cord were affected. An increase of connective tissue was noted also in the muscles.—K.]

Diagnosis.—Any typical case of paralysis agitans can be easily and certainly recognized. The important factors are the peculiar tremor, the characteristic carriage, and the rigidity of the muscles of the trunk and extremities. It was formerly a difficult matter to distinguish between paralysis agitans and multiple sclerosis; but to-day the peculiarities of the two diseases are better known, and confusion is seldom possible. The character of the tremor varies in the two. In paralysis agitans it persists even when the patient is quiet, and it is decidedly oscillatory. The motion in multiple sclerosis (*q. v.*) is almost always an intention tremor only; and, what is of still greater importance in distinguishing between them, the general appearances of the two diseases are essentially unlike.

Treatment.—As has already been implied above, we possess no means of controlling the disease. In most cases, therefore, the treatment is confined to general hygienic measures. Good may be done by lukewarm baths of considerable duration, and by gentle massage of the muscles. Among internal remedies Erb has lately recommended hyoscine, as having the most palliative and soothing influence. It is best given subcutaneously, and we must always begin with very small doses, as otherwise unpleasant symptoms, such as vertigo, headache, etc., may arise. We inject a twentieth to a twelfth of a grain (mgr. $\frac{1}{3}$–$\frac{1}{2}$) in aqueous solution (solution of hyoscine muriate, 1 to 1,000, seven to fifteen minims). If well borne we can cautiously increase the dose. [Atropine has recently shown good results, especially when given subcutaneously.—K.] Arsenic, ergotine, potassic bromide, physostigmine, and curare may also be tried. If electricity is to do any good at all, the case must be a recent one. It is said that in some instances stretching of the nerves has diminished the tremor considerably; but our own observations would not lead us to recommend the procedure in this disease.

CHAPTER IV.

ATHETOSIS.

In 1871 the American neurologist Hammond described under the name of athetosis (*ἄθετος*, without fixed position) a peculiar symptom of irritation of the motor centers, differing in a characteristic manner from all other forms of involuntary movements, including the epileptiform and choreic. The movements of athetosis (see page 541) are often very complicated and peculiar. The part affected by them is in continuous unrest. If the facial muscles (usually those of the lower division of the facial nerve) and the muscles of mastication be attacked, the face and mouth are constantly being twisted and distorted. If the tongue suffer, as in one case which we saw, speech is difficult and indistinct. If the muscles of the back of the neck be implicated, the head is usually drawn backward or to one side, and is turned and twisted in all sorts of ways. Most characteristic of all, however, are the movements exhibited by the hand and fingers when affected. The fingers are incessantly being separated, extended, flexed, and intertwined, assuming the oddest positions. The accompanying pictures may serve to illustrate this (*vide* Fig. 108). The character of the movements reveals that the interossei must be chiefly involved. It is a very frequent result of the unceasing stretching of the articular ligaments of the fingers that at last the articulations become relaxed to such a degree as to permit of hyperextension of the fingers, which it is impossible for a healthy person to imitate. The arms are generally less severely affected than the hands; and in the lower limbs the trouble is not often so severe as in the upper. The toes may, however, exhibit motions analogous to those of the fingers.

Although in general the movements are continuous, their vigor frequently varies. Thus they almost always are aggravated if the patient becomes excited. During sleep they generally cease, although in certain instances they have persisted even then, only being diminished. When voluntary motions are being made they ordinarily grow feebler; but they may, on the contrary, become exaggerated, taking the form of sympathetic movements.

We must distinguish between genuine idiopathic athetosis and a symptomatic form, which also occurs.

Symptomatic athetosis is seen in various nervous diseases. The first observations reported by Hammond were, most of them, made in cases of epilepsy, or the severe psychoses, and the like. By far the most frequent source of the phenomenon, however, is hemiplegia, producing what is known as post-hemiplegic chorea, or better, post-hemiplegic hemi-athetosis. This is, to be sure, a very rare sequel to the ordinary hemiplegia of elderly persons, but follows rather infantile paralysis of cerebral origin (*vide* p. 744). Some traces of athetosis are seen in a majority of the cases of infantile hemiplegia.

Idiopathic athetosis is rare. Here the peculiar movements are the chief, if not the sole, symptom of disease. A few cases of this sort have been reported where the athetosis began without known cause, and usually was limited to some one region. It attacked elderly individuals who were previously healthy. Of especial importance is an apparently congenital form of athetosis, dating from the earliest infancy. Of this we have ourselves seen several instances, which closely resembled one another. The condition is a permanent one, not progressive, nor, on the other hand, capable of any great amelioration. The movements are almost always most pronounced in the face, head, and fingers. There are no other nervous disturbances,

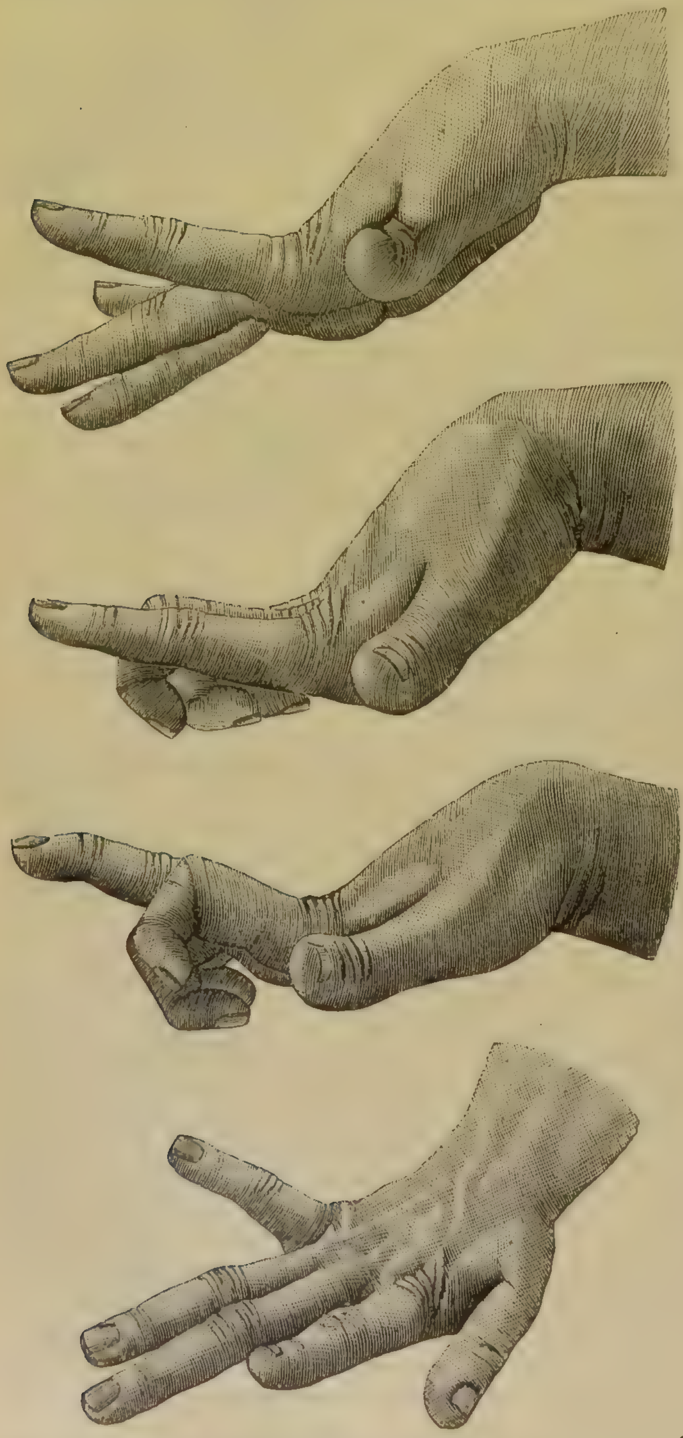
Fig. 108.—Example of the position of the fingers in the movements of athetosis (personal observation).

neither paralysis nor impairment of sensation. The intelligence of the patient may or may not be below par.

Of the nature of athetosis, or the locality or nature of the irritation, we possess no information as yet. It is extremely probable that the lesion is in every case a cerebral one. Perhaps it is in the cortex. In symptomatic athetosis we find post mortem the changes caused by the original trouble. In idiopathic athetosis no changes have thus far been reported. In a case of our own which came to autopsy, absolutely nothing abnormal was found in the brain. The patient was an elderly female, who displayed typical movements of the arm and hand.

It is not yet known whether recovery is possible. A certain amount of improvement sometimes follows the administration of Fowler's solution, bromide of potassium, or galvanism.

CHAPTER V.

TETANY.

(*Intermittent Tetanus. Tetanilla.*)

Ætiology.—Tetany, a name originating with Corvisart, is applied to a peculiar neurosis, characterized mainly by paroxysmal tonic convulsions in certain groups of muscles. The disease attacks by preference children and young adults between fifteen and thirty years of age. The physiological processes peculiar to the female sex seem to have an especial tendency to excite the disorder. It is comparatively so frequent in nursing women that Trousseau has called it "*contracture des nourrices.*"

Among exciting causes, catching cold deserves particular mention. Hence the earlier observers describe the disease as "intermittent contracture of rheumatic origin." In other cases the disorder has appeared as a sequel to other acute diseases, such as typhus or typhoid fever, smallpox, and intestinal troubles. A very remarkable fact was pointed out by N. Weiss—namely, that tetany is apt to follow operative extirpation of goître. No explanation of this has yet been discovered. Reports from various quarters give color to the idea that tetany may sometimes be, to a certain extent, epidemic. It must be confessed, however, that it is somewhat doubtful whether the attacks referred to were genuine tetany. We are also inclined to believe that endemic influences may promote its occurrence. At any rate, the published accounts would seem to indicate that tetany is much more frequent in Heidelberg (Erb, F. Schultze), Breslau (Berger), and Vienna (N. Weiss), than in Leipsic, for instance, where it is one of the very rarest nervous diseases.

Clinical History.—The paroxysm of tetany usually has certain prodromata, consisting of slight general discomfort and pain, and of a feeling of weakness and stiffness, most marked in the arms. These symptoms last some hours (at least) before the true convulsive stage begins. The upper extremities, and more particularly the fingers, almost always suffer first; and then, after the arms, the lower extremities become involved. The spasm usually affects the toes before it seizes upon the other parts of the leg. The symptoms are almost invariably bilateral and symmetrical. Exceptionally, the disturbance commences in a lower limb, or is confined to one side of the body. In most cases the flexor muscles are predominantly affected, giving rise to very characteristic postures. The fingers are in apposition with one another, and placed as if holding a pen, or, as Trousseau says, as if the hand were about to be thrust into the vagina, during labor.

The hands are flexed, the elbows are also slightly flexed, and the upper arm in severe cases is pressed against the chest. In the lower extremities, the toes are flexed, and the feet are in the posture of talipes equinus. The muscles of the thigh rarely suffer. The same is true of the trunk, face, and diaphragm. The main characters of a typical attack, such as has just been sketched, apply to all but a few cases.

The intensity of the tonic spasm is very great. The affected muscles feel as hard as a board, and are usually rather sensitive to pressure. The attack sometimes continues only a few minutes, but not infrequently it may occupy several hours or days. As a rule, there are no disturbances of sensation or other additional nervous phenomena. There is no impairment of consciousness. In a few instances slight œdematous swelling has been observed, and also profuse perspiration. The temperature is normal or slightly elevated, but the pulse is often quite rapid.

When the attack ceases, which it always does gradually, and never suddenly, the patient feels perfectly well, save for a slight pain and stiffness in the muscles. But even in the interval between the paroxysms there are usually some few objective symptoms, which have a most important bearing on the pathology of tetany. In the first place, the peripheral nerves are generally abnormally sensitive to electricity. The complete demonstration of this fact we owe to Erb. The weakest current will produce frequently violent contractions. In an analogous way, the nerves react to unusually slight mechanical stimulation. This is often peculiarly marked in the facial nerve, as Chvostek and N. Weiss have shown. Thus, if the face be vigorously stroked from above downward, almost all of the muscles contract energetically, one after the other. The direct mechanical excitability of the muscles, on the other hand, is not increased (F. Schultze).

Another very characteristic symptom was discovered by Trousseau—"Trousseau's sign." It is found in most cases, although not in all, and is this: a fresh paroxysm can at any time be artificially excited by pressure upon the larger arteries and nerves of the arm (particularly the median nerve and the brachial artery). It is not definitely known how compression accomplishes this. Berger found that mechanical or electrical irritation of certain painful points situated along the spinal column produces the same result.

The frequency of the attacks varies greatly in individual cases. As a rule, there are several paroxysms daily; but the intervals may last for days, or again may be almost inappreciable. The entire duration of the disease is generally several weeks. It is noteworthy that when the paroxysms grow less frequent and violent there is also a gradual diminution in the hypersensitiveness of the nerves and in the reaction to Trousseau's test. As long as these symptoms persist, spontaneous attacks are also possible.

The termination of tetany is almost always favorable. No essential anatomical lesions have yet been detected. The symptoms leave us in doubt whether the disease affects the peripheral nerves or the nervous centers.

Diagnosis.—The diagnosis is not difficult if we only consider carefully the symptoms presented, the nature of the paroxysms, and the other phenomena above enumerated. Similar conditions may result from ergotine-poisoning, or from certain occupations, as in "cobbler's cramp," but the differential diagnosis is usually easy. The peculiar tonic spasms of young children are not, in our opinion, to be regarded as tetany; they have already been described (*vide* page 576) under the name of arthrogryposis, and are characterized by persistent tonic contracture, not paroxysmal, and most pronounced in the distribution of the ulnar nerve on both sides; and in them there is no abnormal sensitiveness of the nerves to mechanical stimuli.

Treatment.—The main treatment, besides general hygienic measures, is electricity. The stabile current is passed upward through the nerves affected; the galvanic current is also applied to the spinal cord, and the anode is applied to the various nerve-trunks, with the cathode on the sternum. This last procedure sometimes dissipates a spasm actually present. Internal remedies, such as bromide of potassium, arsenic, and belladonna, rarely produce brilliant results. Berger was successful in some cases with subcutaneous injections of curare. Tepid baths, and cold sponging with friction cautiously employed, and applied especially to the back, often aid treatment materially.

CHAPTER VI.

TETANUS.

(Lock-jaw.)

Ætiology.—There are two chief exciting causes of this disease, as are indicated by the names rheumatic tetanus and traumatic tetanus. The rheumatic variety results from catching cold, or getting a thorough wetting, or some similar mishap. The other occurs in persons who have some open wound, whether from injury or operation. There is no ground for establishing tetanus neonatorum as a third distinct form. Cases to which this name has been applied are invariably connected with the falling off of the cord, and are therefore instances of traumatic tetanus. In a few instances it is impossible to discover what has been the immediate occasion of the disease; such cases are classed as idiopathic tetanus.

With us tetanus is a comparatively rare disease. It is noteworthy that people who have to do with horses are quite often affected (Verneuil). In the tropics it is much more common. Negroes are well known to be peculiarly liable to it. Men are much oftener attacked than women. Tetanus has been seen as an endemic and also as an epidemic. This has been most frequent in times of war, and is in part due to the unfavorable influence of certain external circumstances, such as bad weather or bad hygienic surroundings. Traumatic tetanus comes on after injuries of the fingers, hands, and lower extremities.

All these facts must previously have led to the thought that tetanus was a specific infectious disease, but we have only recently succeeded in establishing an actual foundation for this suspicion. Rosenbach cultivated from the wound of a man who died of tetanus a special form of bacillus which Nicolaier had formerly found in garden earth. These bacilli are distinguished by a little head at one end (spur). If a small amount of these bacilli be injected under the skin of mice, the most violent tetanic spasms ensue in the animals thus treated. Since the development of the bacteria remains limited to the seat of the wound or the injection, it is *a priori* probable that the spasms are not excited immediately by the bacilli, but by a chemical poison produced by them during life. In fact, Brieger has lately succeeded in producing several alkaloid-like substances from tetanus-cultures, so-called toxines, which he terms tetanine, tetanotoxine, and spasmotoxine. All these substances are violent poisons, and, like strychnine, provoke the most violent tetanic spasms in the animals experimented on.

It is still uncertain whether the tetanus bacilli enter the body only through injuries of the skin, or in other ways.

Clinical History.—In rheumatic tetanus the symptoms usually begin soon after exposure to the exciting cause. There may be, however, an interval during which the patient feels perfectly well, or at most has only certain mild and indefinite premonitory symptoms, such as languor and headache. Similar prodromata may occur in the apparently idiopathic cases.

Traumatic tetanus seldom begins immediately after the injury has been received. Several days or even weeks may intervene previous to the outbreak of the disease. Here, too, there may be mild prodromata for a brief period preceding the graver phenomena. The patient's wound presents no specific appearances. Tetanus may be associated with either slight or severe injuries, whether treated carelessly or kept aseptic.

The symptoms of the disease proper are the same in both rheumatic and traumatic tetanus. They usually begin gradually. Ordinarily, the first thing noticed is a feeling of rigidity and tension in the muscles of the face, lower jaw, and back of the neck. The stiffness spreads by degrees to the muscles of the back and abdomen. The disease is sometimes completely developed in a few hours, but sometimes not till after several days.

The persistent tension of the facial muscles gives the countenance a strange immobility. The brow is usually wrinkled, and the corners of the mouth are often drawn back in a "sardonic grin." Most prominent of all is the tonic spasm of the masseters, or trismus. The teeth are so firmly pressed together that it finally becomes impossible to open the mouth more than one or two millimetres. The eyes are staring, the pupils usually contracted. The muscles at the back of the neck draw the head somewhat backward, and it is immovable. The spinal column is bent forward, so that the trunk is convex anteriorly, permitting the hand to be passed between it and the bed—opisthotonos. The epigastrium and the anterior part of the abdomen are flat. The abdominal muscles are as hard as a board. The lower limbs may be rigidly extended, but the arms generally can be quite freely moved. Convulsive dysphagia, as seen in hydrophobia (*q. v.*), may occur, but it is rare (*vide infra*).

In many cases the continuous tonic spasm is occasionally interrupted by sudden and irregular paroxysms, during which all the affected muscles become still more tense. In severe cases this gives the whole body a violent shock, and makes the opisthotonos even more pronounced. In a very bad case the paroxysms are very frequent; in a mild case they are rare or almost indistinguishable. Sometimes they are apparently spontaneous, and sometimes they are evidently of reflex origin, being superinduced by external irritation. In severe cases the cause may be comparatively insignificant, such as a slight jar or noise.

If there are other nervous derangements, little is known about them—partly, no doubt, because it is seldom possible to make an extended examination of the patient. Sensation is said to have been impaired in some instances, but in others it is perfectly normal. The muscles affected by the spasms are usually the seat of severe pain. The cutaneous reflexes are almost always greatly exaggerated. In two cases which we saw very recently, the patellar reflex was much increased, and in one there was distinct ankle-clonus. Paralysis is extremely exceptional. There is often profuse perspiration. The intellect remains perfectly unclouded.

There is a special form of tetanus which must be briefly mentioned. It was first described by E. Rose, and is called "hydrophobic tetanus," or "cephalic tetanus." It occurs only in connection with injuries situated in the distribution of the cranial nerves—that is, in the face and head—and is characterized in most cases by violent spasm of the pharynx and œsophagus. This is in addition to the other ordinary phenomena of tetanus. The disease in many ways reminds one of hydrophobia. Another characteristic point is that in most cases there is facial paralysis on the injured side.

Tetanus seldom gives rise to disturbances referable to the internal viscera. In one case, however, in the Leipsic hospital, croupous pneumonia and acute nephritis came on a few days before the end. Often there are dyspnœa and a most harassing sense of thoracic oppression—symptoms due mainly to the convulsive rigidity of the muscles, by which the thorax is constantly maintained in the position it normally assumes during inspiration. Expectoration is impeded; and, finally, there may be such an accumulation of secretions in the mouth and air-passages as to cause a secondary diffuse bronchitis, or an inhalation pneumonia. Another occasional source of extreme dyspnœa is spasm of the glottis.

The pulse often remains normal for a long while, but it is usually accelerated, not infrequently reaching 120 or 160 beats a minute in severe cases. Such a pulse is small, and may be somewhat irregular. The temperature is at first usually normal, or slightly elevated. Later it is almost sure to rise; and, as Wunderlich pointed out, it is often very high shortly before death—for instance, 107° to 111° (42°–44° C.). It is not rare for the temperature to keep on rising for a short time after death. No explanation of this terminal elevation of temperature has yet been furnished. It can not be the result of the increased production of heat occasioned by the muscular spasm, for in earlier stages the most violent convulsions are unattended by any such change. Authorities are, therefore, inclined to assume that at the last there is a paralysis of the centers which regulate the warmth of the body, just as is seen in other severe nervous disorders, such as meningitis, injury to the cervical portion of the cord, and uræmia.

Interesting observations have been made with regard to tissue-metamorphosis during tetanus. The excretion of urea is not increased. This fact agrees well with Voit's view, that muscular activity has no connection with the breaking down of albuminoids. Senator failed to find any increase of kreatine and kreatinine in the urine. Probably the production of carbonic dioxide is abnormally large in tetanus. At least, physiological considerations would strongly indicate this, although it has not yet been actually demonstrated. Occasionally traces of albumen and sugar have been detected in the urine. There is usually obstinate constipation, probably due to the persistent rigidity of the abdominal muscles; and, indeed, micturition is not a little impeded from the same cause.

It may be said, in regard to the general course of the disease, that there are severe and mild forms of the disease. What has been said above applies mainly to the severe form. In this, all the symptoms reach their extreme violence in a few days, the paroxysms occur in quick succession, and death usually takes place within a week or two. The fatal result is brought about by the suspension of respiration and by cardiac failure. Of course, the extreme difficulty of taking an adequate amount of food has an unfavorable influence. The bad cases seldom outlast the first week. If they do, there is some slight hope of recovery; the paroxysms may gradually become less frequent and less severe, until they finally cease altogether. The severe form, however, so rarely ends favorably that the prognosis is always very grave. The mild form, on the contrary, usually runs a much more favorable course. In it, all the symptoms are from the first much less severe. Often there is only more or less trismus, accompanied by no marked spasm in the muscles of the trunk, if any at all. There is little constitutional disturbance. The temperature is normal; and the prognosis is rather favorable. The disease may sometimes drag on for some weeks, but it often ends in complete recovery. It must not be forgotten, however, that what at first seems a mild case may develop into the severe form.

The anatomical changes in the nervous system in fatal cases are almost wholly negative. Any small hæmorrhages, etc., have, if they be present, only a secondary significance.

Diagnosis.—In most cases, tetanus can be easily recognized from the peculiar convulsions and the general aspect of the disease. It might be confounded with acute meningitis, for this may cause rigidity of the neck and back; but here there are usually certain cerebral symptoms also present, such as headache and impairment of consciousness; and, on the other hand, in tetanus, trismus is an almost constant phenomenon, although exceptional in meningitis. Strychnine poisoning produces convulsions similar to those of tetanus, but they generally affect the extremities in a more marked degree. Hydrophobia is distinguished from tetanus by the ætiology, the absence of trismus, the predominance of the pharyngeal convulsions, and the greater distinctness of the individual paroxysms.

Where trismus is the only symptom, we must guard against mistaking for tetanus the symptomatic rigidity of the jaws which occurs with severe sore throat, diseases of the teeth, or inflammation of the maxillary articulation.

Treatment.—There is no specific method of treating tetanus. In accordance with the view above mentioned as to the nature of the disease, we have employed, repeatedly, large doses of salicylic acid. This seemed to work well in one case, but in others it did not. We must therefore rely mainly on symptomatic remedies, with the aim of preserving life until a spontaneous cure takes place. For this purpose, narcotics are apparently to be preferred; and among them the best are opium in large doses, and chloral, of which thirty grains (grm. 2) should be given two or three times a day, and the amount gradually increased. If deglutition be very difficult, the chloral may be given per anum. Bromide of potassium should also be mentioned (at least two and a half to four drachms, grm. 10–15, daily), and calabar-bean (a sixth of a grain of extract of physostigmine, grm. 0·01, three to five times a day). The above remedies diminish the irritability of the nervous centers. In curare we possess a means of lowering the excitability of the terminations of the motor nerves in the muscles. It has therefore been employed by many, but by few with success. It is difficult to say what the dose of curare is, inasmuch as the strength of different samples varies. The best way is to determine the strength of the solution to be employed by experimenting on some animal. Usually a one-per-cent. solution of curare in water is employed, and an amount equal to one quarter of the contents of a Pravaz's syringe is injected, the dose being gradually and cautiously increased. [Such a syringe contains about thirteen minims (0·8 grm.).]

It is very desirable to put the patient by himself in a darkened and quiet chamber. Nourishment should be liquid, and lukewarm stimulants, such as alcohol and camphor, should be given from the first. Protracted warm baths may be given cautiously. We know from personal observation that such baths are very grateful to some patients.

It need hardly be said that in traumatic tetanus the primary wound should receive careful attention. Since, according to recent investigations, the tetanus bacilli remain limited in their growth to the seat of the wound, it may be indicated in the beginning of tetanus, if possible, to amputate the wounded part or to excise the wound. Of course, from present experience we can not promise a successful result. Otherwise the treatment is the same as in rheumatic tetanus.

CHAPTER VII.

CONGENITAL MYOTONIA.

(*Thomsen's Disease.*)

In 1876, Thomsen, a Sleswick physician, described a peculiar disease which up to that time had escaped observation. He had had experience of it in himself and numerous members of his own family. Thomsen called it "tonic convulsions of the voluntary muscles," an appropriate but somewhat clumsy name, for which we suggested instead "congenital myotonia." Apparently the disease is very infrequent; but a considerable number of cases have already been reported in Germany, France, and Italy.

The disease seems to be congenital; at least, the symptoms invariably date from the earliest infancy. It is very often hereditary; and males seem to suffer oftener, and also more severely, than do females. The essential symptom of myotonia is this: whenever any voluntary muscle has been inactive for a time and is then made to contract, it falls into a state of more or less persistent contraction, a mild sort of tetanus, so that it can not be immediately relaxed. It is obvious how this would interfere with any series of motions, and make voluntary movement difficult. The patient is not paralyzed at all, but he has a feeling of great resistance to be overcome in performing any act. Quick and accurate motions are often out of the question, so that, for instance, the patient can not perform military duty. It is noteworthy that the stiffness temporarily disappears after the patient has been moving his muscles for some time. On going upstairs, the first steps are often very stiff and laborious, while succeeding ones grow easier and easier. Mental excitement invariably exerts a very unfavorable influence, exaggerating the stiffness of the muscles.

Upon physical examination, the observer is usually struck by the extraordinary development of the muscles. The size of the extremities, in particular, almost deserves the term "genuine muscular hypertrophy," although the strength is not always proportionately great. It is a remarkable fact that, upon direct electrical irritation of the muscles, the contraction outlasts, in most cases, the passage of the current. This is also true, although less marked, when the electricity is applied to the motor nerves. Erb has also observed, during the stabile application of the galvanic current, wavelike contractions starting from the cathode and passing one after another over the muscles toward the anode. The direct mechanical excitability of the muscles is sometimes normal, but sometimes increased. The idio-muscular contractions (*vide* page 546) are especially apt to be increased. The reflexes, sensation, and, indeed, all other nervous phenomena, are normal.

These facts render it very probable that the cause of the disease is to be sought in the muscle itself, and that myotonia is due to a congenital peculiarity of the muscular system. Erb reports that, upon microscopic examination of minute particles of muscular tissue which were excised, he found marked hypertrophy of individual muscular fibers, and an increase in the number of nuclei in the sarcolemma.*

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* [In a recent treatise Erb admits only twenty-three typical cases known at present. His investigations show that on voluntary movement there are protracted contractions of the muscles lasting from five to thirty seconds; these he terms myotonic disturbances of motion. Mechanical excitability of the nerves is perhaps diminished; faradic excitability of the nerves is normal, but a sudden increase of the current may excite protracted contraction; galvanic excitability is nearly normal, but a succession of shocks may produce the tonic contraction. The mechanical excitability of the muscles is increased, a touch with the finger being enough to excite tonic contraction. Faradic excitability

The disease persists through life. The patient gradually becomes accustomed to it, and learns to conceal his misfortune as much as possible. There may be no constitutional disturbance. Sometimes there is melancholia. Therapeutic efforts have not yet been attended with much success. We might try cold sponging, with friction, gentle massage, and methodical exercise of the muscles.

[Eulenburg describes a congenital family affection, which he calls congenital paramyotone, allied to myotonia, where tonic spasm was produced by cold. Gowers has found the association of tonic spasm with ataxia—ataxic paramyotone.—K.]

CHAPTER VIII.

CATALEPSY.

Formerly catalepsy was regarded as a special form of disease, but at present the opinion is almost universally held that it is merely a symptom of several different diseases. As was mentioned on page 541, "cataleptic rigidity" is a term descriptive of that peculiar condition of the muscles in which the limbs maintain involuntarily any position which the observer puts them into. If we change the position of the members which are cataleptic, the patient does not make the slightest effort to alter the posture in which we leave them, however strange and awkward, and apparently insupportable, it may be. The limbs may be moved in this way almost like wax, and have therefore been said to exhibit a "waxy flexibility."

No real explanation of the cataleptic state has been given. We have not yet advanced beyond the study of the circumstances under which it appears, and of the associated phenomena. As has been said, the tonic muscular spasm of catalepsy is never very great, being little more than suffices to overcome gravity and maintain the limb in the posture given to it. This shows that there must, in every case, be a due proportion in the vigor of the contractions of antagonistic muscles; and this relative force must vary with every change of position. How this continuous and remarkable regulation of motor nervous energy takes place is an unanswered question. Perhaps reflex action has something to do with it. A further interesting point is, that changes of position induced by electrical stimulation of the nerves or muscles are not permanent; when the stimulus ceases to act, the limb falls back into its old place.

Catalepsy occurs oftenest as a symptom of hysteria. In this case it is usually associated with other disturbances, chief among which are impairment of consciousness and anæsthesia. The anæsthesia is especially marked in the muscles. For example, the patient will stand for an hour with arms extended, and yet experience not the slightest sensation of weariness. At last, however, the arms sink slowly down. Closely allied to hysterical catalepsy is hypnotic catalepsy, a condition which can be artificially produced by certain procedures (see the next chapter) in many hysterical subjects at will. Charcot has reported cases where

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is very marked; the muscles are quickly relaxed after weak currents, but a sudden increase produces protracted contractions. Galvanic excitability is increased quantitatively, anodic and cathodic closure contractions appearing with currents of one or two milliampères; qualitatively both poles react alike. The wavelike contractions mentioned above are very characteristic. Erb has given the name of myotonic reaction (MyR) to these electrical phenomena. Hereafter, he thinks, a tap with the percussion hammer and a few cathodic closure contractions will suffice to establish the diagnosis. Besides the anatomical changes above mentioned, Erb also finds that the cross-section of the individual muscular fiber is rounded instead of polygonal, like the normal fiber, and that the interstitial tissue is increased. In one case he found a striking vacuolization of the individual fibers.—Trans.]

the lethargy of hypnotism could invariably be transformed into catalepsy by opening the previously closed eyes. These cases also presented, in addition to the catalepsy, the strange phenomenon called "suggestion." If the patient were put into any posture associated with some definite mental conception (such, for example, as the attitude of prayer, or that assumed in terror, or to express detestation), then the corresponding thoughts would come into the mind, as a hallucination, but with all the vividness of reality. Ample proof of this was visible in the expression of the face and in the whole bearing of the subject. An analogous fact has been observed by Duchenne, Lasègue, and ourselves—namely, that sometimes a hysterical person can be brought into the cataleptic state by artificial closure of the eyes (compare what is said in the next chapter about hysterical anæsthesia).

Catalepsy is also seen in many psychoses, particularly in certain grave forms of melancholia known as melancholia attonita and katatonia, and sometimes in progressive general paralysis. (For particulars see works on insanity.) The cataleptic state may also develop in connection with grave organic cerebral disease, as in meningitis and apoplectic coma. It may finally be mentioned in passing that quite well-marked catalepsy is sometimes observed in young children of one or two years when they are ill. Probably they fall into a sort of stupor; or often it seems that they are rendered hypnotic, as it were, by the presence of strangers.

Catalepsy has been regarded as a special disease in those very rare cases where otherwise healthy persons are liable to "cataleptic fits." The condition comes on suddenly, unprovoked, and passes off spontaneously after a variable length of time. It is, however, very probable that these cases are either hysterical or epileptoid, and deserve to be classed with genuine epilepsy.

Prognosis and Treatment.—Since catalepsy is a symptom of so many different diseases, it is impossible to make any general statements with regard to prognosis or therapeutics. The reader may gain some light from the remarks on the treatment of hysteria in the next chapter.

CHAPTER IX.

HYSTERIA.

Ætiology and Definition.—It is impossible to give a definition of hysteria that shall be at once brief and accurate, for the aspects of the disease are so manifold that there is no one symptom which can be called pathognomonic or even universally characteristic. Hysteria is therefore symptomatically not a well-rounded morbid unit, but the nature of all those pathological conditions which we term hysterical may very well be regarded from a single point of view. Hysteria thus has its peculiarities and its rules like any other disease. Only because its laws have been sought elsewhere than where they really are, has the claim often been made in the past that the morbid symptoms of hysteria were subject to no law.

The fundamental condition for a right understanding of hysteria seems to be that we should free ourselves from the old untenable idea that hysteria is a "general functional disease of the whole nervous system, where now this and now that portion of it may be disturbed in its functions." We regard hysteria as a disease which relates to the cerebral activity immediately associated with the psychical processes—that is, if you will, with a psychosis, but in the broader sense of the word; the disturbance affects not only the normal course of the psychical processes, but also their association with the purely corporeal processes of innervation. The starting point of the disturbance, however, is always, in its final relation, in the psychical sphere, although, as we shall see later, the occasion of the psychical change is often to be sought in material processes.

Starting from this standpoint we may note the following characteristic marks of hysterical affections:

1. All hysterical disturbances, no matter how severe the functional nervous derangement attributable to them, are without visible anatomical basis. The best proof of this is the fact that any case, however alarming, may recover completely in a very short time.

2. The hysterical affection is very often intimately associated with exciting causes of a psychical nature. Not only is its appearance and incipiency most closely linked with emotional excitement, but later on the mind is the main if not the only channel through which causes can operate to change the condition of the patient, whether favorably or unfavorably.

3. It is therefore evident that the origin of all hysterical disturbances must be sought in the most central portions of the nervous system—those regions which are most directly concerned in the mental processes. Hysterical phenomena are, however, exhibited in all parts of the nervous system, so far as these depend upon psychical processes or may be altered by them. The symptoms of hysteria are therefore more manifold than those of almost any other disease. Although so manifold, certain symptoms predominate with such frequency as to be characteristic of hysteria, and therefore to be regarded as especially valuable in diagnosis. Following Charcot, we term such symptoms "hysterical stigmata." These symptoms are most constantly present, and therefore they can easily be found at any time.

4. Besides the permanent symptoms—the "stigmata"—we see in many cases of hysteria peculiar nervous attacks. These are also highly characteristic of hysteria, since they come on in forms which are seen in no other disease; but there are also patients who suffer from hysteria who nevertheless are never visited by attacks. The possibility of the occurrence of an attack is, however, always present. Very often the attacks are provoked by easily discoverable psychical causes.

After this brief introduction, turning to the ætiology, psychical causes, as has been said, are of first importance. In numerous instances hysteria comes on as an immediate sequel to violent emotional excitement or to a "psychical trauma," if we may use such an expression. Hysterical convulsions or paralysis may be excited by great terror or violent anger or any unusual agitation. Here the efficient cause is often hidden by some attendant circumstance. If, for example, a woman falls into the water, or gets burned, or tumbles downstairs, and thereupon develops hysteria, the mistake is often made of ascribing the disease to catching cold, or to the injury received, although really it was the mental excitement which produced it. Only in severe traumatic concussion of the whole body may the partly physical symptoms of commotio cerebri and commotio spinalis be united with the psychical shock. There then develop peculiar morbid conditions which we term traumatic neuroses and which are closely related to hysteria. We will speak of them particularly in the last chapter of this section. Purely hysterical conditions, such as anæsthesia, hyperæsthesia, contracture, paralysis, etc., may also arise from slight local injuries. What is very remarkable in such cases is, that the special attendant circumstances at the time of the psychical disturbance often influence the localization of the hysterical phenomena: that part, to which attention was particularly directed at the time, not infrequently becomes, later on, the seat of the nervous disturbance. In hysterical joint-affections (page 528) the cause not infrequently proves to have been an injury to the particular joint now suffering from hysteria. A young girl, who was awakened at night by the smoke of her burning mattress, and who had a severe laryngitis from inhaling the vapors, exhibited later an indubitably hysterical paralysis of the vocal cords. In the case of another girl, who in jumping from a carriage fell upon her side, we afterward saw hemianæsthesia of the same side. Many such instances could be cited.

Certain hysterical cases, therefore, have for their obvious cause a single occasion of mental agitation; but in many others the disease is not thus abruptly excited. As, in poisoning, we can distinguish between the sudden action of a large dose and chronic cases where minute amounts of poison are absorbed daily for a long period, so hysteria may come on not only after a single violent shock, but also as the final consequence of psychical influences insignificant of themselves, but potent because of their frequent repetition or persistency. It is in cases of this sort that the causes do not become evident to the physician until he has gained the entire confidence of his patient; for the root of the trouble is often entwined about the most private affairs. Anxiety, sorrow, disappointed expectations, abandoned hopes, and, in brief, everything which can depress and overwhelm the mind—these are factors which may at last excite the functional nervous derangements of hysteria.

Finally, we must state that hysteria may often develop without any specially noticeable cause. We see this particularly in young people with a marked hereditary nervous taint. Here the ordinary daily demands upon the central nervous system sometimes suffice to disturb in a measure its normal equilibrium.

There are still other causes. The blow which brings a feeble body to the ground rebounds without effect from a massive frame. Exactly the same may be said of the "blows" to which the nervous system is subjected. Few entirely escape these influences, but there are some "strong natures" who resist the psychical assault without wavering, while others have a feebly resistant nervous organization and are overpowered. We see, therefore, that the predisposition of different individuals to diseases of the nervous system varies. This fact is a very important one in the pathogenesis of all functional nervous disorders. In what this predisposition consists we do not know, being acquainted only with its results and with some of its causes.

In many cases this predisposition is hereditary. Hysteria is prominent among those neuroses which attack different members of a family—one suffering from one disease and another from another (*vide* page 770). It is also possible to acquire such a predisposition. At least it may be developed and fostered on the one hand, or, on the other, checked and repressed. In these directions physical as well as psychical factors are of importance. Anything which weakens the constitution diminishes the resistant power of the nervous system. Among psychical influences, nothing favors the development of hysterical tendencies more efficiently than does a bad education. Hysteria is often ascribable to an irritability and feebleness of the nervous system thus engendered. The whims of the child are not controlled, its will is not strengthened, nor its energy developed; its imagination is unsuitably and excessively stimulated, or its intellectual powers are overtaxed and prematurely ripened.

It is well known that hysteria occurs more frequently in the "feebler" female sex than among men; but it is by no means a rare thing for men to exhibit hysterical convulsions, paralysis, contracture, or other well-marked disturbances. Most cases occur between puberty and the end of middle life. It is not very rare to see pronounced hysteria in children, especially such as are over eight or ten years old. The disease very frequently begins to develope a year or two previous to puberty. Nationality and race also seem to exert some influence—for instance, the severer forms of hysteria are decidedly more frequent in France than in Germany; and the Jewish race are particularly subject to the disease.

One matter remains to be considered with regard to ætiology, to which a greatly exaggerated importance was formerly ascribed. It is the influence of disease of the sexual organs. The very name "hysteria" (ὑστέρα = uterus) reveals what the old view was, namely, that hysteria invariably originated in disease of the female genitals. Not to speak of the fact that the disease occurs in men and children, an unprejudiced consideration of the matter will show that the above assumption is entirely groundless even in regard to women. A large number of hysterical women present no anomaly of their sexual organs; and even if the latter be diseased, we are not justified in at once assuming that the hysteria is secondary to the sexual disorder. In these cases, also, we usually find, on careful inquiry, that psychical causes have been at work; and these are incomparably more potent in exciting hysteria than is malposition of the uterus or constriction of the cervical canal. It is true, however, that disease of the genital organs may depress the spirits more than some other diseases would, and so indirectly promote hysterical disturbances. In the same indirect way, menstruation, pregnancy, and confinement exert an important influence upon the development and course of hysteria. Sexual excesses or total abstinence from sexual indulgence also produce their effects indirectly through the mind.

THE SYMPTOMS AND MANIFESTATIONS OF HYSTERIA.

Considering the great differences in the external morbid symptoms under which hysteria may manifest itself, it is not easy to give in a brief summary a superficial, intelligible, and yet accurate description of its clinical manifestations. We believe that we can best attain this end by speaking first of the most important and most frequent of the single symptoms of hysteria, by describing next the hysterical attacks, and finally by trying to evolve a general picture of the disease.

1. **The Hysterical Stigmata, especially the Sensory Anæsthesia and Hyperæsthesia.**—In every case where the diagnosis of hysteria has already been established or where it is to be confirmed, we must examine the patient for certain symptoms, which, as we have said, are so common in hysteria, and in part so peculiar to it, that by themselves they are often of decisive diagnostic significance. These symptoms are termed "hysterical stigmata." A knowledge of them is the more important for the physician in that we often detect them only by a special examination directed to that end. Patients only exceptionally inform us as to these symptoms. It is not at all unusual for patients to have no inkling of their existence until after an examination.

The most important, because the commonest, of these hysterical signs relate to sensibility. The more carefully we examine, the more rarely will we meet with a case of severe hysteria in which in some sensory region we do not find manifest disturbances, especially a diminution of sensibility. We must therefore test accurately not only the sensibility of the whole surface of the body, but also the functions of all the other senses (sight, hearing, smell, and taste).

The sensory disturbances of the skin must first be mentioned. Not infrequently we find a diminution of sensibility over the whole surface of the body, especially a more or less complete analgesia. In such cases we can stick a pin in deeply anywhere, or pierce a fold of skin with a pin, without the patient's complaining of pain. As is well known, it often happens that hysterical patients, in order to appear interesting or from some other reason, produce upon themselves deep injuries and wounds; this is almost always connected with the patient's analgesia. Very often, however, the anæsthesia is not general, but it is limited to a definite region. We find complete anæsthesia of one arm or leg, or we find on the trunk or the extremities single areas of anæsthesia, in which the anæsthetic parts are bounded by the normally sensitive skin; these areas may take all possible forms, and may often be very peculiar. The sensibility to pain is almost always the most disturbed when the other forms of cutaneous sensibility may be quite unaffected, but changes in the sensibility to temperature, pressure, etc., may also occur. It may be added that in the severer forms of anæsthesia the deeper parts may also be insensitive, so that the so-called muscular sense is absent, and patients with their eyes shut have not the slightest idea of the position of the anæsthetic limbs.

Besides cutaneous anæsthesia, disturbances in the sensibility of the other sensory organs (other "sensory anæsthesias") are among the commonest signs of hysteria. Sometimes the patients say, when asked, that they see indistinctly and dimly. If we examine the eyes we often find a loss of visual acuteness and a rapidly induced fatigue on using them. A limitation of the visual field is the most characteristic sign—that is, an anæsthesia of the peripheral portions of the retina. The degree of this limitation sometimes shows manifest differences for the different colors. Hysterical achromatopsia is a common symptom—that is, a partial or complete loss of the color sense. According to Charcot, in the hysterical the perception of violet usually first disappears, then green, and lastly blue and yellow; but we must not hold too strictly to all these rules; for, although the French investigators are otherwise deserving of great praise in adding to our knowledge of hysteria, in this respect they seem decidedly too schematic, and to have generalized too rashly. Diminution of the auditory acuteness in one or both ears is also not uncommon. Anæsthesia of smell and taste is still more common. Salt, quinine, vinegar, sugar, etc., either excite no sensation of taste at all, or the tongue is anæsthetic for one or another of these substances. The same holds for the sense of smell. All these symptoms are especially important in diagnosis, because in organic diseases they are relatively much rarer in such a degree than in hysteria.

The sensory anæsthesias above described may of course be combined in various ways in different cases, but none of these combinations is so characteristic and individual as hysterical hemianæsthesia, a symptom-complex which occurs almost solely in hysteria, and is therefore of distinct diagnostic value.

Hysterical hemianæsthesia is one of the most common symptoms of profound hysteria. It must often be sought for, inasmuch as the patient herself frequently has no suspicion of its existence until her attention is called to it. It seems just as if one half of the body had been entirely lost to consciousness; the patient does not know whether it is or is not capable of feeling.

In a typical and fully developed case the hemianæsthesia does actually affect just one half of the body. There are rudimentary forms; but in these complete ones, the boundary between the anæsthetic parts and the parts retaining normal sensitiveness accurately corresponds to the median line of the body. The skin on the affected side is entirely insensible to the prick of a needle or to heat. Often it seems somewhat blanched, and its blood-vessels seem to be constricted; at least the skin very often bleeds surprisingly little if wounded. The mucous membranes upon the abnormal side are all equally anæsthetic, including the conjunctiva, that half of the buccal cavity and of the tongue. The deeper parts, such as the muscles and joints, are also almost invariably anæsthetic. The patient can no longer feel in what position the limbs of the affected side are; and if they are moved passively, no sensation is communicated to her. The organs of special sense are usually involved. Hearing is impaired upon the affected side; the corresponding half of the tongue can not taste; the corresponding nostril can not smell; and sight upon that side is affected in a peculiar manner. There is no hemiopia, but a total amblyopia, or possibly amaurosis. If the amaurosis be not complete, we find at least a limitation of the visual field and the signs of the above-mentioned achromatopsia.

Apart from other hysterical symptoms with which hemianæsthesia as well as all the other sensory anæsthesias may be associated, we must mention here one symptom which is in close relation to anæsthesia, and which was first described by Duchenne under the name of "loss of muscular sense" (*perte de la conscience musculaire*). The patient, whose arm, for example, is anæsthetic, can not move it if she closes her eyes, although with open eyes she can move it as well as ever. When the eyes are shut the arm remains motionless in the position it has previously occupied. If its position be altered by passive motion, the new position is maintained with equal persistency. There is, with the eyes shut, pronounced catalepsy. Duchenne referred this peculiar symptom to the loss of a special sense which he termed "muscular sense" (*conscience musculaire*). According to our present conception of hysteria it would be more correct to regard the symptom as purely psychical.

The much-discussed and peculiar symptoms of transfer, as well as metalloscopy and allied symptoms, will be mentioned below.

Thus far we have spoken only of anæsthesia, but we very often find in the hysterical certain hyperæsthetic regions. These hyperæsthesias are so characteristic that we must reckon them among the hysterical stigmata, and must therefore look for them in every case. Sometimes the physician's attention is called to these places because they are the seat of constant slight or severe pain. In other cases the tenderness appears only upon pressure. The hyperæsthesia may be so great that the patient can scarcely bear the slightest touch. Of course, the hyperæsthesia is very closely connected with the condition of the patient's attention. If the thoughts be diverted, even firm pressure is often not at all noticed. We see clearly, then, that we usually have to do here with a "psychical hyperæsthesia."

The hyperæsthetic spots are either quite extensive or quite circumscribed, or even limited to one definite spot. They may lie near to or in the midst of anæsthetic areas. They are relatively rarest in the extremities and more common on the head and trunk, especially on the sternum, on the sides of the chest, under the mammæ, etc. Most common, and therefore especially characteristic, is the hyperæsthesia of the vertebral column, and, in women, of the lower abdominal region ("ovarian pain," "ovarie"). The latter term is convenient but by no means correct, since really we have only a tenderness of the soft parts generally, which is at once noticeable on deep pressure, and we do not have solely or chiefly a tenderness of the ovary. We usually find "ovarie" only on one side, oftener on the left than on the right. Analogous symptoms in men—tenderness in the groins or the testicles—are less common. Hyperæsthesia of the vertebral column, hysterical "spinal irritation," is even more important for diagnosis than ovarie. The hyperæsthesia affects either the whole spine or merely single portions of it, often only single vertebræ. It may attain so high a degree that the slightest touch of the skin over the vertebræ, or somewhat deep pressure, may cause the liveliest outcries and shrinking. This symptom is almost pathognomonic of hysteria.

The relations which these hyperæsthetic regions have to the hysterical attacks are very important, and they have therefore procured for them the name of "hysterogenous zones." We will speak further on this point in describing the hysterical attacks.

Hyperæsthesia in other sensory regions—abnormal sensitiveness of the eye, ear, etc.—also occurs. Thus certain cases of hysterical blepharospasm seem to be due to an immoderate sensitiveness to light. Unusually sharp hearing is noted, especially during the hysterical attacks. Here, too, belongs the aversion of many patients to certain smells and tastes, and the like.

2. **Hysterical Paralysis.**—Hysterical paralysis is frequently an immediate sequel to some violent mental excitement (for example, "paralysis from fright"); but it may come on gradually. Hysterical paralysis is indubitably of central origin. It is a paralysis of the will. The patient has lost the power to will a contraction of the affected muscles. One always has the feeling that the patient could move the paralyzed limb perfectly well if she only desired to; but she can not bring the will to bear on it, and this inability is the real trouble. It also seems a remarkable circumstance that hysterical paralysis often, but of course not always, affects only certain combined movements. Many patients, for instance, can move their legs very well in bed, but they can not walk a step.* We have seen a hysterical writing paralysis; the right arm was not paralyzed, but it utterly refused to make any attempt to write.

The extremities are most frequently paralyzed, particularly the lower limbs; but hemiplegia is not especially rare. A very common manifestation is loss of the power to walk. The patient lies in bed or on a sofa, and sometimes while thus reclining she can flex and extend the legs very well; but as soon as she is urged to stand up or walk, the knees double up, the patient begins to tremble, the respiration grows rapid and convulsive, and there is not the slightest effort made to move the legs. If only one leg be paralyzed, the gait is often very peculiar and characteristic. The sound limb makes long strides, while the paralyzed one is held perfectly rigid, and often is dragged along with a loud shuffling sound. The arms are much less often affected. The facial muscles are hardly ever paralyzed. We once saw hysterical diplopia.

In regard to the more precise form of the paralysis, both flaccid and spastic paralyses occur in hysteria. In many cases of hysterical paralysis the tendon reflexes may be so exaggerated—nay, there may even be, as we have repeatedly seen, such a lively foot phenomenon—that one is at first disposed to think of actual spinal paralysis. In other cases, as in paralysis of an arm, the paralyzed limb hangs down completely flaccid. The paralyzed parts are often at the same time completely anæsthetic (a circumstance which may often be distinctive in diagnosis), but in other cases they are normally sensitive or even hyperæsthetic.

Hysterical paralysis of the vocal cords is seen very often. The voice is generally lost suddenly, and the patient can talk only in a whisper—hysterical aphonia. On laryngoscopic examination, we are often struck, at the outset, by the anæsthesia of the pharynx and its lack of reflex excitability. We find no trace of any anatomical lesion of the cords, but merely that they are paretic. The glottis can not be completely closed, and sometimes the vocal cords actually grow wider apart upon every effort at phonation. The patient then speaks exclusively in a whisper. Hysterical mutism quite rarely follows hysterical aphonia. The patients entirely lose voluntary control of their speech apparatus, and finally become completely dumb.

Hysterical paralysis of the pharynx and œsophagus is much less frequent. If there be dysphagia, it is often not an easy matter to determine whether it is due to paralysis or to spasm.

3. **Hysterical Contractures.**—Contractures may occur as isolated phenomena, or in combination with other symptoms, such as anæsthesia or paralysis. They

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[* This inability to stand or walk, with the preservation of good strength and co-ordination in the legs when sitting or standing, and the persistence of power to execute certain less usual movements, such as hopping on one foot, has been termed by Blocq *astasia-abasia*. It is often associated with hysteria, but it may exist independently of it.—K.]

are caused by unusually strong tonic muscular contractions. The starting point of the irritation is certainly in the central nervous system. Sometimes the contractures are temporary, but often they are characterized by their intensity and their great persistency. The extremities suffer most frequently, although the trunk or the back of the neck may be affected. In the hands and feet, especially in the toes, flexor contractures predominate, but in the larger joints extensor contractures are the rule. Although many varieties occur, single forms of contracture are especially characteristic. As an example, we will refer to the accompanying illustration (Fig. 109).

The relations of hysterical contractures to the articular neuralgias have already been mentioned (p. 529). They may occur in a hemiplegic, paraplegic, or monoplegic form. They often follow a hysterical convulsive attack (*vide infra*). All hysterical contractures disappear completely in chloroform narcosis and in deep sleep.

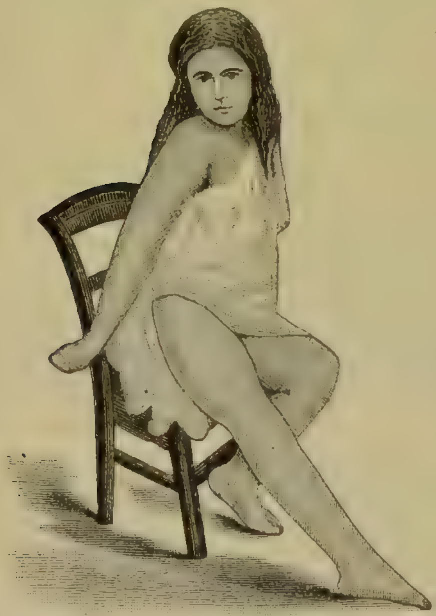

FIG. 109. — Hysterical contracture. From Bourneville and Regnard's Iconographie de la Salpêtrière).

4. **Vasomotor Disturbances. Secretory Disturbances. Symptoms in the Internal Organs.** —Besides the sensory and motor symptoms just described, other symptoms occur in hysteria which fall under the domain of the vasomotor and secretory nerves. The significance of many of these symptoms is, of course, not yet clear.

In the first place abnormal anæmia or abnormal fullness of the vessels (a cool, pale skin in the one, a hot, red skin in the other) is not infrequent, and points to vasomotor influences. Since we know that the vascular nerves are influenced in a high degree by the emotions, we can probably assume a central origin for these symptoms. Hæmorrhages from internal organs are harder to explain; they are apparently not uncommon in hysteria, and they have often been explained by "nervous vasomotor influences," but in our opinion we must be extremely guarded in such an assumption. We most frequently see hysterical hæmatemesis or hysterical hæmoptysis—that is, the evacuation of blood from the mouth, either with cough or with more of a strangulation. Inexperienced physicians have thus often been led to a false belief in dangerous pulmonary disease or gastric ulcer; but if we look more closely we shall find that the blood evacuated is of a clear raspberry-red color, is mixed with a good deal of mucus and saliva, and is of a thin fluid consistency. The whole amount is seldom more than two or three ounces (grm. 50-80). If we inquire more carefully as to its origin, we can usually find a source for the blood in the gums or in the oral or pharyngeal mucous membrane. There are often spasmodic chokings or hiccoughs, by which a hæmorrhage of the mucous membrane is mechanically produced. It is also certain that in many cases intentional deception on the part of the patients may lie at the bottom of the hæmorrhage. We must be especially cautious in hæmorrhages from the genitals, from the skin, from the palms in the "stigmatized," etc.

Finally, among the vasomotor disturbances we must mention one symptom whose significance, in our opinion, has not yet been satisfactorily explained—we mean hysterical fever. In severe hysteria, especially at the time of severe attacks and psychical disturbances, other observers, and we ourselves, have repeatedly seen high febrile temperatures, 106° (41° C.) and more; and these temperatures come on in a very irregular manner. In none of the cases under our own observation, however, could we wholly exclude the possibility of simulation—that is, of producing a rise of the mercury by rubbing and pressing on the thermometer. In all the measurements we made ourselves in the rectum the temperature was normal, while the high fever was always said to occur in our absence. We therefore recommend great caution with reference to this point.

Anomalies of the secretory and excretory organs have also been met with in hysteria. Many patients have a remarkably dry skin, while others sometimes perspire very freely. The secretion of saliva is subject to similar modifications. Very remarkable observations have been made in a few cases with regard to hysterical ischuria; for days only a very small amount of urine has been passed, although there has been no retention. In one case of this sort, observed by Charcot, there was violent vomiting at the same time, and the vomitus contained a comparatively large amount of urea (vicarious excretion). Hysterical polyuria occurs more frequently than ischuria. A large amount of very light-colored urine of low specific gravity is excreted. This polyuria is in many instances merely the result of excessive ingestion of liquids. Polydipsia (excessive thirst) is a very frequent symptom in hysteria, particularly at the close of a fit.

The digestive disturbances which many patients have are mainly such as have already been discussed on page 400, under the head of "nervous affections of the stomach." Colicky pains, obstinate constipation, occasional diarrhœa, and similar symptoms, are by no means rare. Hysterical tympanites also deserves mention. It is due to the accumulation of a large amount of air and gas in the primæ viæ. This may be in part the result of a sort of paresis of the muscular coat of the stomach and intestines, but another frequent cause is the swallowing of large amounts of air. Perhaps the patient does this on purpose. The prominence and tension of the abdominal walls may be so considerable as to simulate grave diseases, such as peritonitis, or a tumor, or pregnancy. Doubts of this sort can always be dispelled by inducing anæsthesia with chloroform. It is possible to remove the gas completely in a short time by pressing upon the abdomen, or by introducing a long tube through the rectum.

Sometimes there are symptoms referable to the genital organs. It has been already pointed out that too much prominence was formerly given to the influence of sexual diseases in exciting hysteria. It is also true that nervous derangements of the genital organs may be among the symptoms of hysteria. In this way pain and hyperæsthesia may be occasioned, and possibly many instances of dysmenorrhœa and leucorrhœa have a similar origin. We can also readily understand that sexual relations often influence very excitable, hysterical individuals to no slight extent, as indeed is very frequently betrayed by the character of their hallucinations and their utterances when delirious.

5. **General Mental and Bodily Constitution of the Hysterical.**—If we regard hysteria as a disease which is largely psychical, which is the only correct view, it will not seem strange if the whole mental constitution and nature of the patient show special peculiarities. In many cases, therefore, the psychical condition of the hysterical is so characteristic that the physician can, from the patient's nature and demeanor, form his conclusions as to the form of her disease.

Hysterical persons are irritable and emotional, easily depressed, sensitive, whimsical, and subject to violent extremes of feeling. They are inclined to exaggerate their sufferings, exact a great deal of attention, and are anxious to excite sympathy. They have little energy or force of will, but they are sly and obstinate in carrying out any pet desire. Again, they can be very amiable and attractive if they take the fancy. They are almost invariably clever. It is comparatively exceptional to see hysteria in dull and stupid persons.

This brief sketch represents many cases, as we have said, but not all. Such patients very frequently present no very great disturbances, but complain merely of all sorts of general derangements, sometimes of one kind and sometimes of another, and yet are able to perform their daily duties tolerably well. A case of paralysis, contracture, or other important localized hysterical trouble may not present any marked mental peculiarities. Either there are none, or, if they exist, the patient conceals them from the physician.

With regard to the general physical constitution of hysterical subjects, it has already been mentioned that any bodily weakness favors the development of the disease; and yet hysteria is by no means confined to the ill-nourished, weakly, and anæmic. On the contrary, many patients seem to be in blooming health and well nourished. In a severe case of hysteria, however, the effects of the disease on general nutrition may be very distinct. Little food is ingested, sleep is disturbed, digestion is affected (*vide infra*), and the bodily health is gradually undermined.

HYSTERICAL ATTACKS, CONDITIONS OF SPASM, ETC.

While the symptoms thus far described are largely of a permanent character, paroxysmal nervous symptoms are also very common in hysteria. The diagnosis of the entire morbid condition is often made certain by the onset of such hysterical attacks, which are often very characteristic, and their significance is readily perceived by the experienced physician. There are some cases of hysteria which run their course entirely free from these attacks, where there are only certain permanent nervous symptoms, such as paralysis, contractures, anæsthesia, etc.; but there are also other patients in whom the hysterical attacks dominate the whole scene, and are sometimes the only manifestations of the disease. A hysterical attack is not infrequently the beginning of the trouble, especially in those cases where the disease is excited by fright, etc., and where the attack follows immediately upon the psychical excitement.

In regard to the severity and the variety of the hysterical attacks, they are so manifold that an exhaustive account of all the possibilities can not here be given; but certain features and details are so common in them, and recur so often, that a consideration of them is often in itself sufficient for a correct diagnosis.

The mildest form of the hysterical attack consists of the development of a feeling of distress, anxiety, vertigo, and especially of a loss of voluntary control of the body. The patient sinks on a bed or chair, closes the eyes, and becomes incapable of action or speech. Mild symptoms of motor irritation usually ensue, most commonly an acceleration of respiration, general tremor, winking of the eyes, etc. Mild spastic symptoms in the pharyngeal muscles and the diaphragm are not uncommon. There is very often a lively palpitation during the attack. The face is sometimes quite red, but in other cases pale.

In such mild attacks any impartial observer has distinctly the impression that the patient is humoring her inclinations. If, therefore, she be encouraged, or if cold water be thrown in the face or on the back without too great regard for her feelings, she usually soon regains her will-power and rapidly recovers.

In an unbroken series these mildest forms of attack pass over into the severer types, where the clouding of consciousness is greater and the symptoms of motor irritation are more severe. Complete loss of consciousness, so common in epilepsy, is scarcely seen in hysterical seizures, but there is very often a marked clouding of consciousness, and in the severe forms morbid alterations of consciousness (*vide infra*). If hysterical attacks occur which resemble those of epilepsy ("hystero-epilepsy"), we must think of the possibility of a combination of the two diseases; but usually the form of the hysterical convulsion is essentially different from that of the epileptic. In severe hysterical convulsions the convulsive movements are more varied, more extensive, and more complicated than in epilepsy. The arms make thrashing and thrusting movements, and often apparently quite well-coordinated movements. Patients strike the bed, and sometimes their own bodies, with the clenched fist; they seize objects, such as the bedclothes or furniture, and cling fast to them. We also see clonic or tonic flexor and extensor spasms in the legs. The eyes almost always converge or are turned to one side, and they often roll. The lids are lightly closed or occasionally open. There is almost always trismus, and there is often grinding of the teeth. The trunk usually twists and turns the most. The patient often thrusts her head hard against the wall or the bed. The whole body may assume positions which a healthy person can scarcely imitate without special practice. The best known, and in fact one of the commonest and most characteristic, is the arched position (*arc de cercle*), of which the accompanying illustration (Fig. 110) gives an idea.

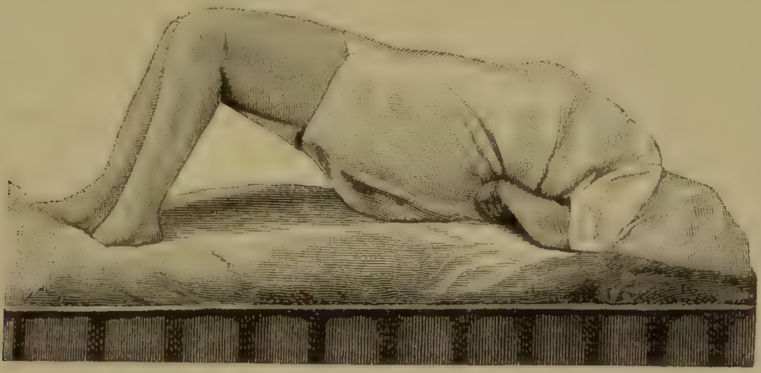

FIG. 110.—Hysterical *arc de cercle* (Bourneville and Regnard).

Many patients rest for a time on the floor supported only by the head and toes. At intervals they sling or roll their bodies to and fro, drum on the floor with their legs, throw themselves into the air, etc. Any one who has once seen such a "grand hysterical convulsion" will never forget the picture.

A different but still common form of hysterical convulsion is characterized by a pronounced implication of the respiratory muscles. The attack begins with a spasmodic acceleration of respiration, and the breathing becomes shorter and more rapid. We have ourselves counted two hundred respirations a minute. Other peculiar spasms of the respiratory muscles, which occur in this way only in hysteria, are also common; hiccough, loud sobbing, grunting, etc. The muscles of the pharynx are usually implicated in producing these noises. Of course, all these spasmodic conditions may be joined in the most varied ways with spasms of the trunk and the extremities.

One of the most essential factors which often gives the most characteristic stamp to the grand hysterical attack, is the relation which the spasms often have to certain co-existing abnormal conditions of consciousness. During the attack the patient is usually not unconscious, but she is under the dominion of inner morbid delusions, and these often mirror themselves in the outward movements. The patient is wholly dominated by a definite circle of ideas; she has hallucinations, and passes through some frightful or exciting event. All this is most strikingly manifested in her movements and her expression. Hence, in an attack the face often expresses terror, rage, threatening, concupiscence, serenity, etc. Very often the internal excitement breaks forth in words, and there is actual hysterical delirium, the most marked emotional utterances, etc. The patients often talk continually to themselves, usually very rapidly, with frequent repetitions of the same phrase or word. We can often succeed in giving the delirium a definite direction by questioning the patient, or converse regularly with the patient during an attack, but usually the pure motor spasms again set in, or persist for some time in a tonic form.

We can not go further into the numerous details of the symptoms of the grand attack ("*grande hysterie*"), for an accurate knowledge of which we must thank the observations of Charcot and the school of La Salpêtrière in Paris (Bourneville and Regnard, P. Richer). The descriptions of the French authors apply to the grand hysteria not infrequently seen in Germany. In regard to the whole picture of the attack the French neurologists distinguish several periods which correspond in general with the above-described conditions. The first period consists of severe epileptiform convulsions apparently associated with loss of consciousness. Then comes the period of "contortions and grand movements" (clownismus), and finally the period of plastic positions and passionate attitudes (*attitudes passionelles*). The short and very significant expressions of this scheme are of practical value, but in our experience we can only rarely expect sharply defined "periods" in the individual case; the grand hysterical attack is rather composed in the most varied ways of the different symptoms above mentioned.

There remains one essential relation in order to complete the picture of the hysterical attack, and that is the "suggestibility" of the patient, which is so peculiarly characteristic of hysteria. By "suggestion" we mean the artificial production of a definite psychical state, or a physical state dependent on the mind, by arousing the appropriate ideas. In the general characteristics of the mental constitution of many hysterical persons we have already had to point out how much and how often such patients let themselves be dominated by their imaginations. Suggestion is merely the artificial fostering of this psychical peculiarity and its special application. The more we try to foster and preserve this peculiarity, the more we leave uncorrected the false ideas which the patients have, the better we succeed at last in making the patients merely a shuttlecock for their ideas. Hence the physician's daily experience teaches that, as suggestion experiments are repeated, the patients more easily become susceptible to them, so that finally we can actually "do everything with them." There can scarcely be any difference of opinion as to whether such an experiment without restraint is medically and morally permissible; and even the fostering of suggestion for therapeutic purposes is a two-edged sword whose action is not always rigidly within the physician's control.

Suggestion is most easy during the hysterical attack itself, especially in those forms where the patients speak, hear, and answer. As soon as we give the substance of the patient's ideas a definite direction, and tell them in a convincing tone that they are in a garden or a wood, picking flowers or fruit, that they are attacked, bound, lying on the edge of a precipice or the water, etc., we see in their bearing and speech that they think they are actually experiencing all these conditions in their delirium and hallucinations. The emotional utterances of fear, terror, joy, or aversion are then often expressed with astonishing art. In the same way we can suggest paralyses, contractures, or anæsthesias. The most interesting feature of it all is, that when the attack is over every trace of recollection of what happened during it is lost. The same patients who just now were so much excited by some definite idea, a few seconds later, when the attack has ceased of itself or has artificially been brought to an end (*vide infra*), know nothing more about it. They have not even a vanishing recollection like that of a dream about what they have just said and done, even if it be expressly described; but it is still more remarkable that during the following attack they often remember very well what they have experienced actually or in their imaginations in the past attack. In such cases we can actually speak of "double consciousness." Processes of the conscious waking life, however, often remain in the consciousness during the attack. We often see that the actual event (scene of terror, etc.) which has given rise to the first onset of the attacks, is often gone through with anew in the delirium of subsequent attacks.

Hypnotic symptoms are closely allied to the processes in suggestion. We can not give here a complete review of this field, which, as is well known, has been so much studied of late. The tendency of human nature to mysticism and the influence of suggestions, which may be of value not only with the patients but even with the investigating physicians, are the reasons why the false and the true have often been mixed in the study of hypnosis. We can say only that one great advance is now generally recognized, and that is, that most persons of scientific training have abandoned the hypothesis, once frequently accepted, of a special "magnetic power" (animal magnetism), by which the "magnetizers" could put their "mediums" into the "magnetic sleep" or other abnormal conditions.

Hypnosis is nothing more than the intentional artificial production of a hysterical attack, or a hysterical psychosis by suggestion—that is, by the action of definite ideas on the person to be hypnotized. Therefore, only those persons can be hypnotized in whom these ideas have a strong enough influence. No man can be hypnotized to whom the nature of hypnosis is clear. The essential feature of all hypnotic procedures is merely to produce in the most lively way possible the idea, "It will happen as the hypnotizer says." All other things—the fixation of the eyes on bright objects, the vibrations of a tuning-fork, etc.—are side issues, and serve merely to support the suggestion. In all easily hypnotizable persons the mere closure of the eyes and the remark, "Now go to sleep," are enough to produce the hypnotic sleep. Patients (and here we may actually speak of "patients") reach this suggestibility, of course, only after they have often been hypnotized; for the oftener the same action of an idea is produced the more easily it occurs—a law which follows also from many other experiments in the psychical domain. The different forms of hypnosis are not distinguishable from the various hysterical conditions. Hypnosis is artificial hysteria, and from this alone we note the danger of all hypnotic experiments as soon as they are practiced by the ignorant. In this, too, it has often been shown that we can not become free again from the spirits which we call up. The French physicians (Richer) distinguish four chief forms of the hypnotic state, which show, however, many transitions: 1. The cataleptic state, in which the limbs retain all the positions artificially given them (see the previous chapter). 2. The state of "suggestion," of artificially produced hallucinations. If we put the body passively into certain positions corresponding to definite acts, we can produce in the patient all the appropriate ideas with the distinctness of an hallucination. To this category belong the well-known hypnotic exhibitions where hypnotized adult men toss babies, eat raw potatoes with an expression of enjoyment, etc. 3. The lethargic state, that is, a state of apparent unconsciousness, with the eyes closed, the muscles completely relaxed, and a marked increase of excitability in the muscles and nerves. A light pressure or a slight blow on a nerve, like the facial, suffices to put all the muscles supplied by it into a tetanic contraction which outlasts the irritation. 4. By certain manipulations (for instance, rubbing over the parietal) we can change the lethargic state into one of hysterical somnambulism. The patients remain half unconscious, but answer automatically questions which are put to them, obey orders given to them, and sometimes show certain sensory hyperæsthesias. We see that all these forms are precisely identical with the different forms of the hysterical attack. Only the increased mechanical excitability of muscles and nerves is not yet fully explained.

May not suggestions—that is, ideas which lead to unconscious voluntary muscular contractions—also play a part here?

We have thus far omitted to dwell upon an important point in the description of the hysterical attack—namely, the relation to it of "hysterogenous zones." We have mentioned above how often in hysterical patients certain parts of the body (the ovarian region, the sides of the chest, etc.) are extremely sensitive to pressure. It is by no means uncommon that, in spite of the patient's resistance, somewhat longer pressure on such a spot may excite a hysterical attack. On the other hand, we sometimes succeed in causing an existing attack to cease by pressure on the same zone. We are of the opinion that in these manipulations also ideas become potent as connecting links.

Finally, we must add that there are also milder forms of hysterical spasm which are limited to a definite muscular region, and are not associated with any marked clouding of consciousness. There are, for example, isolated spasms of the muscles of the neck, isolated respiratory spasms (spasmodic cough, etc.), and isolated spasms in the arms and legs; the laryngeal muscles may also be affected (hysterical spasms of the glottis). Spasms of the diaphragm and other muscles of inspiration are quite common under the form of hysterical hiccough, which may sometimes last in the severest way for days or weeks. To spasmodic conditions in the muscles of the pharynx and œsophagus we refer the well-known symptom of so-called *globus hystericus:* the patients feel as if a ball were moving up and down in the throat.

We sometimes see spasmodic conditions which come on in a clonic fashion, or in single twitchings in this or that muscular territory; sometimes affect symmetrical groups of muscles; are not associated with disturbances of consciousness; cease during sleep, and are often easily cured by appropriate mental treatment. Such conditions have been described under special names as special diseases—electrical chorea, paramyoclonus multiplex, or myoclonia. In our opinion, most of these cases, if not all, are undoubtedly hysterical (see pages 573 and 576).

General Course of the Disease.—Our description of the symptomatology of hysteria has been confined to the most important and frequent phenomena; and yet even this meager outline shows what an infinite variety of shapes the disease assumes. 1. In one class of cases there are no severe symptoms whatever. The patient merely displays the general mental condition characteristic of hysteria: she is easily excited, prone to make much of her ills, has all sorts of symptoms, such as pain, palpitation, dyspepsia, and dyspnœa, and these are aggravated by mental excitement, while at other times they may so nearly vanish that the patient does not appear to be ill. 2. A second class of cases has more severe disturbance, coming on after some unfavorable psychical influence. The patient may have displayed a general hysterical tendency previously, or may have seemed perfectly well. Here we may observe all the symptoms above enumerated and described. There may be paralysis, spasm, contracture, anæsthesia, or paræsthesia. The individual symptoms may persist obstinately for weeks and months; but again they may vanish on a sudden or give place to other disturbances. Psychical influences are unmistakably potent, not merely in the incipient stage but also in the further course of the disease. Any aggravation of the symptoms is usually referable to emotional excitement. This is particularly true of the hysterical convulsions. In many cases, almost every fresh paroxysm is due to anger, terror, or some similar cause. 3. The third class comprises the most severe forms of hysteria, with those nervous disturbances briefly outlined above. They are as complicated as they are puzzling, and form manifold combinations with all the other hysterical phenomena, including anæsthesia, contracture, and paralysis.

The entire duration of the disease varies greatly. The true root of all evil is the abnormal excitability of the nervous system, which always remains in unstable equilibrium; and often it is not possible to cure this. If not, the trouble lasts almost indefinitely. New manifestations of the disease succeed to periods of apparently perfect health. Usually the symptoms do not abate till quite late in life. There are, however, many instances of complete and permanent relief. This favorable termination is more especially to be hoped for where the patient comes into suitable and appropriate conditions of life, having some regular occupation which is not exposed to all sorts of unfavorable psychical influences. Many cases of hysterical disturbance, in previously healthy children or young adults, and due to some distinct cause, terminate comparatively soon, and never recur. It is never possible, however, to be sure that there will be no relapse, inasmuch as a single appearance of hysteria shows unmistakably that the nervous system is abnormally vulnerable to external impressions and the mental emotions excited by them.

Diagnosis.—An experienced physician is seldom greatly puzzled by hysterical affections. Although the disease may at first simulate some grave organic disorder, a careful physical examination and continued observation will almost invariably disclose the true character of the case. In the first place, there are never any such symptoms as would absolutely prove the existence of some organic lesion. For example, we never find atrophy or loss of electrical reaction in connection with hysterical paralysis. Secondly, many symptoms are characteristic of hysteria and are never seen in any other disease. Such are numerous forms of convulsions, and hemianæsthesia with amblyopia of one eye. Above all, we should regard the whole psychical behavior of the patient, the influence exerted upon her by emotional disturbances, and the ætiology of the illness—for instance, if caused by some mental excitement or emotion. The discovery of specific hysterical symptoms, the so-called hysterical stigmata, sensory anæsthesias, hysterogenous zones, etc., are especially important. Many symptoms of hysteria, especially certain forms of spasm and hemianæsthesia, are in themselves so characteristic that from them alone a correct diagnosis can be made.

Treatment.—What has been said about the ætiology of hysteria at once suggests a possible method of prophylaxis. A watchful eye will often detect, even in childhood, the signs of abnormal nervous excitability, and in such a case the parent will make it his duty to impose a suitable physical and mental regimen, that graver disturbances may be averted.

If hysteria be already developed, the first and most important treatment is mental. There could be no greater mistake than to deride the patient or treat her as a malingerer; for hysteria is a disease, and its symptoms are just as independent of any conscious volition on the part of the patient as those of any other disease. It is, however, absolutely essential to carry out the moral training, which the physician must institute with all the proper strictness and energy, because in this way alone can any good be accomplished. Sometimes this most important indication can be fulfilled only after the patient has been withdrawn from the over-anxious and over-assiduous parents or relatives, and like unfavorable influences. In such cases, treatment in some institution will often be vastly better than the best care at home; and our own experience leads us to recommend most urgently that the eventual necessity of removal to an asylum should be constantly borne in mind with regard to aggravated cases. Often the mere dread of removal to such a place has a favorable mental influence.

Proper moral treatment achieves comparatively the best results where there is hysterical paralysis. When we are once certain that the paralysis is due to hysteria, the patient must be instructed how to regain by practice the lost power of the will over the paralyzed muscles. If the paralysis affect the lower extremities, as it usually does, the patient must be set on her feet, regardless of all her opposition and complaints, and kindly but most firmly required to try to walk. Of course, at first she must be well supported. This exercise must be methodically gone through with several times a day. Gradually the patient's gait becomes more and more secure. She regains confidence in her own ability, and, having once begun to improve, usually makes rapid progress toward complete recovery. Every experienced physician can recall numerous instances where hysterical paralysis which had lasted weeks and months was cured in a few days by this mode of treatment. Faradization of the muscles, cold sponging, with friction and bathing, are excellent adjuvants; and the disagreeable element in these procedures of itself stimulates the patient to make every possible exertion to regain the use of her limbs.

When there is hysterical paralysis of the vocal cords, a similar training will be found both practicable and efficient. Electricity is also of great value. It may be applied externally or within the larynx. Often the patient, terrified by the sudden pain it causes, recovers her voice at once.

The treatment of hysterical contractures consists, first, in an effort to loosen up the contracture by massage and energetic passive motion. Faradism will be found of assistance here also. In order to maintain the ground thus gained, the patient must be induced to exercise the muscles regularly by making voluntary movements.

The treatment of hysterical spasmodic conditions often causes greater difficulties. In many cases a sharp sensory irritation, douching with cold water, or a cold bath with cold shower-bath, suffices to restore the energy of the patient's will, which is necessary to regain control over the muscles and thus check the spasms. The dread of the repetition of the bath does its part in warning the patients from giving themselves up unresistingly to a repetition of the attack. The electric current (strong faradism during the attack) may also act favorably in the same way. The action of such measures, however, very often gradually weakens, the patients become accustomed to the cold baths, and they remain without effect.

The milder varieties of hysterical convulsion, such as hysterical hiccough or cough, are often controlled by a stern reproof. It is precisely in these cases that the moral effect of transfer to some institution frequently causes the abrupt disappearance of symptoms which have lasted for months. The severe hysterical attacks are often, of course, peculiarly obstinate, and they may resist for months, and even years, the most intelligent treatment.

Hysterical anæsthesia is best treated with the faradic wire-brush. This vigorous irritation restores the anæsthetic parts to the domain of consciousness. It should be said, however, that these cases may prove obstinate or relapse.

The most difficult of all hysterical cases to treat are those where the symptoms are not strongly pronounced, but where there is a general hysterical condition. expressing itself in a multitude of nervous derangements, such as palpitation, dyspepsia, and general debility, or in purely subjective symptoms, or in emotional tendencies. Such patients are often advanced in years, so that little is to be hoped for from moral training; and their circumstances may be unfavorable without our being able to remedy the situation. Even here, however, the physician may greatly benefit the patient by means of psychical influences, if he once gains her complete confidence. It will also be found advantageous to employ such remedies as invigorate the nervous system (see the next chapter); electricity should be given, either in the form of general faradization, or the faradic brush applied to the back and shoulders, or the galvanic current applied to the spinal column and the sympathetic nerve; and of still greater importance is a methodical cold-water treatment, either by sponging, or bathing, or douches. Such patients are often vastly improved by sea-bathing in summer, or by going to the mountains.

The numerous internal remedies for hysteria are also of more use in these general conditions than where there are marked nervous disturbances in special parts of the body. In the latter, internal remedies do good only indirectly and subjectively, and about in proportion to the confidence of the patient in the virtues of the medicine. This is the explanation of the frequent cases of rapid recovery after taking homœopathic and "electro-homœopathic" remedies, and those still more marvelous cures effected by means of holy water and relics.

Among the "antihysterical" agents contained in our medical thesaurus, asafœtida, valerian, and castoreum are the most famous: but probably few would at the present day claim that they possess any specific virtues. Perhaps the preparations of valerian are the most useful where there is hysterical excitement, as evinced by convulsions or palpitation. Bromide of potassium, arsenic, and other medicines which ordinarily exert a favorable influence upon the nerves, seldom accomplish any permanent good in hysteria, although often prescribed. Narcotics do little good, and may do much harm. It is easy to develop the morphine habit in such patients.

If hysteria be complicated by some actual organic disease, the latter, of course, demands special treatment. Great benefit is hoped for by many from the cure of any uterine complaint which may be present. Cases are known where grave hysterical disturbance has vanished upon dilatation of a constricted cervical canal or rectification of a displacement; but there are numerous other instances on record where gynæcological treatment has proved entirely unavailing. It may also be questioned whether, in the successful cases, the main benefit was not due to subjective influences. Hegar has removed the ovaries in a few severe cases, but the operation is not yet fully established. At any rate, it is justifiable only when the ovaries are known to be in an abnormal condition. Friedreich claims to have had excellent results from energetic cauterization of the clitoris. We do not believe that in this he will have many imitators.

In general, we may certainly maintain that all methods of treatment of hysteria are efficient only when associated with the necessary psychical factor. Hence any prescription, no matter how senseless it seems, may have the greatest effect as soon as the patient "believes in it"—that is, as soon as the psychical action of this belief is manifest. Hence we may say that in hysteria, as a rule, treatment either produces rapidly a brilliant success or it has no effect at all.

We must here mention two methods of treatment which have lately been much discussed—metallotherapy and treatment by hypnosis.

In regard to metallotherapy a French physician, named Burq, discovered years ago that by laying plates of metal upon a cutaneous surface affected by hysterical anæsthesia a remarkable result is sometimes produced. Almost at once sensation is restored to the immediate region, and often to a much larger area. Most of the cases have been those of hysterical hemianæsthesia. It is not every kind of metal which will prove effective, nor will the same kind affect all patients. It is said that iron is most frequently efficient; but sometimes copper, zinc, or gold is required. The process of determining the metal essential to each individual case Burq called "metalloscopy"; and he stated that this metal would also have the same effect if given internally! In 1876 a committee appointed by the Parisian Société de Biologie tested these statements, at least with regard to the external application of metals—the idea of their internal administration having been pretty much abandoned—and confirmed them. Charcot also discovered many remarkable facts of a similar nature, which likewise soon received universal substantiation. The most remarkable of these phenomena is known as transfer. The return of sensation to the anæsthetic area, as a result of applying a metal plate, is accompanied by a simultaneous development of anæsthesia upon the opposite, previously normal side, in an exactly corresponding place. Sometimes sensation oscillates from one side to the other and back again, so that now one half of the body and now the other is alternately sensitive or anæsthetic. If the metal be placed at the start upon the normal skin, that part becomes anæsthetic, while the corresponding part upon the opposite side of the body regains its former normal condition.

It has also been discovered that other hysterical symptoms exhibit analogous phenomena. Transfer can sometimes be observed in hysterical amblyopia, achromatopsia, deafness, loss of smell and taste, contractures, and paralysis. Such transfers may be induced by various means other than metal plates. These are classed as æsthesiogenous remedies, and include large magnets, feeble galvanic currents, and static electricity. Vibrating tuning forks and sinapisms have also produced similar results. Hence it seems to us to be an undoubted conclusion that the whole of the group of symptoms just described has no peculiar place. They are simply the results of suggestion, produced once more by ideas. The similarity of the test as performed by the physician produces the similarity of the symptoms that ensue.

Of late the treatment of hysteria by hypnosis has acquired a far greater practical significance than metallotherapy; it is a mode of treatment which has been practiced most extensively by the "school of Nancy" (Bernheim). If, during hypnosis, morbid states can be produced by suggestion, it naturally follows that morbid states can also be cured by suggestion. If by many well-known successes the hypnotizing physician has at the outset acquired the patient's confidence, as relics have the confidence of believers, the most beautiful results can of course be attained in this way. There is no special peculiar principle in the hypnotic treatment. Any other efficient mode of treating hysteria rests on the same conditions and presumptions. Hypnosis has only the one great consequence, that it is artificially produced as a severe abnormal mental state in a patient who previously had not spontaneously fallen into this state. In that lies the sequel, which of course need not always be permanent, but which often enough has been most severe: the attempt to hypnotize a patient who is suffering from a slight hysterical affection has not infrequently been followed by the onset of a severe hysterical attack. The mischance will seldom happen to the physicians and magnetizers who practice hypnotism as a specialty, because their psychical influence on their patients is usually greater from the outset; but we should consider it a misfortune if hypnosis should come into too general use. That in this way apparently the most wonderful cures can often be obtained, is perfectly well established and not at all surprising; but the same cures can also be obtained in other ways, without running the risk of producing first that of which you would cure your patient, for to hypnotize means to make hysterical. Furthermore, it is not hard to prophesy that, as the knowledge of the peculiar nature of hypnosis becomes general, it will lose its halo with the patient, and with that it will lose its healing power.

CHAPTER X.

NEURASTHENIA.

(Nervous Debility.)

When studying the disorders of the spinal cord, it will be remembered that we found one group of symptoms which did not rest upon any discoverable anatomical basis, but which were merely functional (*vide* page 605). In some part due to abnormal excitability of the nervous system, but mainly, however, the result of impaired vigor, this condition was denominated "nervous weakness." Perfectly analogous phenomena may originate in the brain, and are named cerebral neurasthenia, in contrast with spinal neurasthenia. In most instances we meet with both cerebral and spinal symptoms, and must therefore call the disease cerebro-spinal, or general, neurasthenia.

The American neurologist Beard was the first to recognize the importance of this disease and to give it its present name. Beard was at first inclined to believe that neurasthenia was mainly an "American disease"; but this is by no means the case, inasmuch as sufferers from neurasthenia form a very important contingent among the patients of German specialists. Neurasthenia is certainly one of the most frequent and important nervous diseases from a practical standpoint, nor is its study by any means devoid of scientific interest.

Causation.—A full list of the causes of neurasthenia would include almost all those influences which in any way act unfavorably upon the nervous system. Most of these have been enumerated on page 606. Where the disease is mainly of the cerebral form, excessive brain-work contributes very largely to its production, particularly when combined with certain kinds of excitement. The merchant is liable to it, whose bold ventures subject him to deep anxiety and eager hope; and the politician, who is incessantly agitated by party strifes; and likewise the artist or scholar, whose ambition gives him no rest. In all these cases the nervous system finally becomes exhausted—that is, neurasthenia is established. Even here, however, neuropathic tendencies come into play, for some are crushed by a burden which others can bear. In very many instances this liability to the disease is inherited; in others it is acquired (*vide* page 606).

As was mentioned under spinal neurasthenia, hypochondria frequently assumes an important *rôle* in these cases. It not only exaggerates the existing symptoms, but it contributes others of its own. In this we find one essential difference between neurasthenia and genuine hysteria. In the latter, however much complaint there may be, a genuine hypochondriacal tendency is extremely rare. Hypochondria becomes even the essential factor in those melancholy forms of neurasthenia which so often result from onanism or other sexual abuses. This same element is also probably the main one in the strikingly frequent cases of neurasthenia in physicians.

The symptoms of spinal neurasthenia have been already briefly described, so that we may here confine our remarks chiefly to the still more important cerebral symptoms. Most frequent among these is a subjective sensation of pressure in the head. Patients give a very various description of this sensation. Essentially, however, it is a feeling of pressure and numbness, and makes the patient doubt whether he is in the full possession of his intellectual powers. The pressure is sometimes chiefly frontal and sometimes occipital; it may rise to the height of actual pain, this being frequently associated with marked hyperæsthesia of the scalp.

Associated with this pressure there is, as we have just intimated, in many cases a sense of incapacity for any methodical intellectual effort, an intellectual debility often rendering the patient entirely incapable of performing the duties of his vocation. He can no longer write or read for any length of time, these pursuits being further interfered with, in some cases, by a feeling of weakness and pressure in the eyes themselves (neurasthenic asthenopia). A very important symptom is loss of sleep. This symptom is in many cases the most annoying of any, and makes the patient importunate for relief. There is almost sure to be depression of spirits; the patient does not believe that he will ever recover, and gives voice to the most melancholy predictions. Beard has called attention to peculiar conditions of anxiety sometimes observed in neurasthenic patients. The sufferer dreads to go into society, or to mingle with a crowd, or to be subjected to any physical jar. Vertigo is also frequent, but is rarely very severe.

The lack of intellectual energy is, in most cases of any severity, accompanied by decided bodily weakness. This, too, would often seem to be of cerebral origin, and consequent upon deficient innervation of the muscles by the nervous centers. The patient can not walk far without becoming weary, is incapable of any great manual effort, and in some cases feels so weak that he does not like to leave his chamber, and passes most of his time in bed or on the sofa. The various bodily functions may be interfered with. The appetite is diminished, the bowels constipated, the skin is dry, and the circulation feeble in the extremities, so that there is in many cases constant complaint of cold hands and feet. Sometimes the secretions are increased in amount rather than diminished. There is salivation or profuse perspiration. There may also be palpitation of nervous origin. [In most cases of neurasthenia there is defective metabolism. The urine is scanty, the uric acid increased relatively to the urea, and in sexual neurasthenia there may be an excess of indican. Many neurasthenics, however, drink very little.—K.]

There are numerous other nervous phenomena occasioned by neurasthenia, which we need not here describe with great minuteness. Some of them are most marked in the "spinal form" of the disease; such are pain in the back, spinal irritation, paræsthesia and pain in the extremities, and sexual derangements. Sometimes, however, these symptoms appear to be rather of psychical—that is, of cerebral—origin. Nervous dyspepsia is frequently conjoined with neurasthenia; it has already been described on page 400.

The general course of the disease is almost always chronic. In the milder cases there is little outward evidence of derangement; the patient endeavors to hide his troubles, as his indefinite symptoms seldom gain much sympathy, and are apparently contradicted by his well-nourished and healthy appearance. In the severe cases, however, the patient's vigor is so much impaired that the disease acquires a grave aspect even for others than the patient, and fills them, as well as him, with infinite anxiety. The course of the disease is apt to be varied by alternate improvement and relapse.

Prognosis.—It is difficult to make a general statement as to the termination of cases of neurasthenia. The disease is never actually dangerous, nor does its existence often prepare the way for more severe secondary nervous disease. And yet the nervous constitution of many neurasthenic patients is such that complete recovery can not be attained. There are, however, numerous cases, especially such as have resulted from a special exciting cause, which can be removed, where permanent and complete recovery ensues. In other cases the symptoms can be so far abated that the patient is practically well, although not entirely free from discomfort.

Diagnosis.—Neurasthenia can usually be detected without difficulty, but the establishment of the diagnosis requires the exclusion of organic lesions of the nervous system. Every case, therefore, must be submitted to a thorough and careful examination. Grave cerebral diseases, such as incipient tumors or general paralysis, have been repeatedly mistaken for neurasthenia. One important point in diagnosis is the ætiology, including both the outward circumstances and the presence or absence of a constitutional predisposition to nervous diseases. Hysteria has certainly many points in common with neurasthenia, but it is essentially an entirely different disease. In neurasthenia we find none of those innumerable localized nervous disturbances which we saw in the preceding chapter to be so well marked in hysteria; nor do we ever observe in neurasthenia that rapid onset and sudden disappearance of the symptoms, nor their abrupt development as a consequence of some violent emotional excitement. A severe case of neurasthenia is decidedly the graver disease of the two, at least in this sense, that it represents a far more profound functional disturbance of the nervous system than does hysteria. On the other hand, certain special symptoms, such as convulsions or paralysis, may be more severe in hysteria than in neurasthenia.

Treatment.—As in hysteria, so also in neurasthenia, moral treatment is of prime importance; but here it must be of a different kind than in hysterical cases. The neurasthenic requires sympathy. He must be repeatedly examined by the physician. Every fresh examination, at the end of which the physician is able to assure him of the absence of any serious objective change, has a most quieting and beneficial effect upon the patient. In so far as hypochondriasis is a prominent symptom, this moral influence may alone restore the patient to health.

Where the neurasthenia rests on some other basis than mere hypochondriasis we must, in addition to moral treatment, employ remedies which have a tendency to invigorate the entire nervous system. In order to bring about any permanent improvement, the treatment must be methodical and long continued, so that the patient may remain under the personal influence of the physician for a considerable length of time. Moral training is always an important and indeed an essential part of the treatment of nervous debility.

In any methodical course of treatment, regimen is of great importance. The rules laid down must be carefully adapted to the special circumstances of each individual. Severe mental labor must be forbidden, and mental excitement avoided. The diet depends upon the individual case. For a corpulent patient, treatment calculated to diminish obesity will sometimes be followed by decided improvement in the general condition and in bodily vigor. In those frequent instances where the patient is pale and thin, and very likely oppressed by nervous dyspepsia (see page 400), we should, on the other hand, make vigorous efforts to improve nutrition.* Definite instructions must be given in order that the patient may ingest a proper amount of food. Milk, butter, fresh meat, eggs, and simple puddings are appropriate articles of diet. Often the weight and strength both improve rapidly. Any large amount of alcohol or of tobacco should be forbidden. Tea and coffee may be taken in moderation, if the patient is accustomed to their use. In regard to bodily exercise, we must again be guided by the condition of the individual. We would most earnestly warn the physician from the error, frequently committed, of driving weakly and debilitated persons to take long walks. For such, bodily rest is much more desirable; and fresh air may be enjoyed at the same time, if the patient sits out of doors or drives. The sluggish and corpulent, on the other hand, often require an increased amount of exercise.

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* Playfair, Weir Mitchell, and certain other neurologists, have built up a special "method" of treating neurasthenia and allied conditions of nervous exhaustion; this consists in "overfeeding" the patient—that is, in introducing as large an amount of nourishment as possible into the system at the same time that complete bodily and mental rest is secured. Faradic electricity and massage are also daily employed. This mode of procedure is certainly excellent in many cases, but it must not be regarded as universally applicable. There are cases of neurasthenia for which it is not suitable.

It is a good plan in many instances to employ the Swedish movement cure, or similar gymnastic exercises.

Less general remedies are electricity and hydropathic treatment. Electricity is warmly praised by many patients. The galvanic current is generally employed, and is applied either over the sympathetic nerve or along the spinal cord. Its use demands great caution. The current should not be too strong, and there should be no abrupt changes in it. Galvanism applied to the head is seldom well borne. Another very valuable mode of treatment was first practiced by Beard and Rockwell, and consists in general faradization. The patient is almost completely stripped, and places both feet upon a large, flat electrode, while the various parts of the body are stroked with another large sponge electrode; in place of this second electrode the "electrical hand" of the physician may be employed. The physician takes the second electrode in his left hand and allows the current to pass through his own body. Various institutions have lately begun to employ electrical baths; these also often seem to produce good results. In addition to peripheral galvanization and faradization of the nerves and muscles, it is also advantageous to employ the faradic wire-brush, particularly on the back of the neck, along the spinal column, and over the shoulders and thighs.

The hydro-therapeutic treatment may be quite well carried out at the patient's home, but a severe case will be better off in some well-conducted institution. Cold sponging, douches, hip-baths, lukewarm baths (or swimming), are all employed. Douches must not be applied to the head. If there is sexual disturbance, hip-baths of cold water are advisable. They should not be taken at night. Douching of the genitals and loins is also excellent. Subsequently sea-bathing will prove extremely beneficial for many patients. We would recommend the seashore especially for emaciated and anæmic subjects, who are frequently greatly benefited by the improved appetite and rest thus obtained. If the patient be well nourished, on the other hand, a journey on foot through the mountains, if made cautiously, may be very valuable.

In neurasthenia, internal remedies should be given only as indicated by the symptoms. If there is anæmia, iron, quinine, or Fowler's solution is prescribed; if there is dyspepsia, some stomachics, such as dilute hydrochloric acid, pepsin, or some bitter. The constipation should be overcome mainly by diet. A valuable adjuvant is massage of the abdomen; and, indeed, massage is coming to be regarded as a valuable tonic for the whole system where there is nervous disturbance. It is especially appropriate where there are painful sensations in the nerves and muscles, and may here be combined with electricity. When there are vasomotor symptoms (a feeling of heat, congestion, palpitation), we often prescribe ergotine, four to six one-grain (grm. 0·05) pills a day. In all states of nervous irritation bromine preparations are much used, a powder of potassic and sodic bromides, or bromine water. In nervous headaches, and also in other nervous states, antipyrine often has a good effect. Antifebrine and phenacetine act in the same way.

The treatment of the wakefulness which results from neurasthenia deserves a brief mention. In the first place, we would warn the physician against the abuse of chloral and morphine. The attempt should always first be made to secure sleep by a rational general treatment, or by some less injurious remedies. Often a warm bath for half an hour at bedtime soothes the patient and brings him sleep; and in other cases a wet cloth laid upon the head or back of the neck produces the same favorable result. Patients often report that general faradization at bedtime is an excellent soporific. Sometimes a moderate dose of alcohol is efficient—for instance, a glass of beer or good wine taken before going to bed. If none of these means avail, our next resort should be the bromide of potassium. Very likely small doses of this have only a subjective effect, but there can be no doubt that a large dose, say about a drachm (grm. 3–5) in a glass of water, does have a direct tendency to produce sleep. We may also mention extract of cannabis indica; the preparation known as cannabinum tannicum, five to ten grains (grm. 0·2–0·5); paraldehyde, about a drachm (grm. 3–5) at night; and urethane, twenty to forty-five grains (grm. 1·5–3) in water at bedtime. Paraldehyde has a very disagreeable taste. Kast has lately recommended sulphonal, twenty to thirty grains (grm. 1·5–2) in a large amount of water, in soup or tea, two or three hours before bedtime. These various remedies seldom give great satisfaction, and we must therefore rely mainly on general treatment.

CHAPTER XI.

THE TRAUMATIC NEUROSES.

As an appendix to the last two chapters on hysteria and neurasthenia, we must now speak of a series of morbid states which are at any rate closely related to the above-mentioned affections, but which yet show certain peculiarities. We have to do here with nervous symptom-complexes which come on as a result of a severe concussion of the whole body, or sometimes after a more circumscribed injury of a definite part of the body, and hence are termed "traumatic neuroses." By the term "neurosis" we would imply that the nervous symptoms produced by the trauma do not depend upon coarse material injuries of the nervous system, but upon finer changes not yet made out anatomically.

Considering first those conditions which arise from severe general concussion of the body, they were first observed, especially after railway accidents, by English and American physicians, and given the name of "railway spine" or "railway brain." It was soon proven, of course, that precisely the same morbid symptoms may arise after other injuries.

The type of disease is very characteristic. Usually the original trauma (overturn, collision, etc.) is so great that immediately after it the well-known symptoms of commotio cerebri or commotio spinalis ensue with more or less severity—loss of consciousness, general paralysis of the extremities, collapse, small and slow pulse, cool skin, pallor of the face, dyspnœa, vomiting, retention of urine, etc. Such cases may terminate fatally in a few hours with no material visible change in the brain or cord to be found at the autopsy. In other cases the first severe shock passes off and a series of objective and subjective disturbances remain, which may persist for a long time, and which often do not disappear even after years have passed. It is these subsequent states which deserve the name of general traumatic neurosis. It is worthy of note that these appear not only after severe injuries, but also after mild ones in which the initial symptoms of commotion were not especially marked. We will return to this point in speaking of the nature of the traumatic neurosis.

The severe initial symptoms of commotion cease in a few days, or even sooner. The patient recovers, tries to get up, and the improvement goes on to a certain degree; but a number of symptoms remain which do not disappear, and which diminish or wholly take away the victim's capacity for work. If we examine such patients we find no signs of material injury either in the nervous system or in any other internal organ. What first strikes us is a peculiar psychical change. The patients are dull, depressed, disinclined to any occupation, and have no more pleasure in intercourse with their families and friends. They brood constantly over their accident and its results. The sleep is disturbed and often rendered uneasy by dreams. Of other subjective symptoms we must mention especially pains in the part which was hurt; these are commonest in the back, the sacral region, the sides of the chest, the hips, etc. They also complain of headache, vertigo, dullness, dim vision, tinnitus, spots before the eyes, loss of appetite, etc. Objective examination often shows a general motor weakness. Many patients can walk quite well alone, but they are soon fatigued; others walk only slowly and stiffly with support, complaining of pain in the back on walking, and therefore holding the hand on the back, etc. The nutrition of the muscles is usually good. The electrical excitability of nerves and muscles is completely normal. Examination of the sensibility may reveal very important changes, which are of value in diagnosis. This examination must always extend to all the sensory organs. The skin over almost the entire body is often very insensitive to painful irritations, pin-pricks, the electric current, etc. There is analgesia. In some places we find complete anæsthesia, which may affect an entire extremity or only circumscribed parts of the extremities and the trunk. The boundary between the anæsthetic and the normally sensitive parts is usually quite sharply defined, and the arrangement is often very peculiar. Sometimes the skin is peculiarly insensitive to certain irritants (heat, cold, etc.), or, on the other hand, it is peculiarly sensitive. Examination of the eyes sometimes shows loss of visual acuteness, limitation of the visual field, and imperfect color perception. Very often the hearing is poor on one or both sides. The smell is often much blunted, and the taste completely lost, so that even quinine, vinegar, and similar strongly tasting substances no longer excite any sensation of taste.

Besides the symptoms of impairment of sensation and motion just described, we often find also symptoms of sensory and motor irritation. Pain in the head, spots before the eyes, and tinnitus, have been mentioned above. Hyperæsthesia of the skin is more characteristic—tenderness of the vertebral column, and particularly great tenderness of such parts of the body as were most affected by the original injury. Among the symptoms of motor irritation we may mention muscular contraction and muscular rigidity, again most common in the limbs most affected by the injury. A marked tremor is also very common. The reflexes differ in different cases; they are often much increased. Among the trophic changes we may mention that the hair may turn gray or fall out. Marked muscular atrophy occurs only in the more local traumatic neuroses (*vide infra*).

If we ask after the cause and the special nature of this peculiar affection, characterized chiefly by psychical disturbances, sensory anæsthesia, and motor weakness, two chief factors are to be considered. First, a purely physical factor, the concussion of the nervous system. That such a cause can produce, without coarse anatomical injury, the severest functional injury to the nervous system, is beyond a doubt; and if the most threatening symptoms can come on immediately after the concussion, it is certainly not to be regarded as impossible that permanent results may be left after the concussion which form the basis of the later symptoms of the traumatic neurosis. Furthermore, a purely psychical factor is undoubtedly to be considered. A great fright is associated with the injury. The accident readily excites a fear of an incurable illness, with permanent incapacity for work and for earning a living. The perplexity and frequent contentions about sick funds, insurance societies, and claims for damages, do their part in keeping the patient disturbed. These factors also bring about a state which has the closest points of contact with general nervousness, neurasthenia, and hypochondria. In the individual case it is often scarcely possible sharply to separate the results of the material injury from this mental activity.

In many patients—and then the causes are purely psychical—the morbid symptoms that follow the injury follow closely the type of hysteria. Then we should speak not of a traumatic neurosis but of traumatic hysteria. We have already said, in the chapter on hysteria (*vide supra*), that this is very common. We must regard most of the cases of "local traumatic neurosis" as hysterical. We see not very rarely, after injuries which affect one limb, nervous disturbances in the injured arm or leg which can not possibly be due to a local injury of the nerve—flaccid paralysis, anæsthesia, contracture, hyperæsthesia, etc. We have already learned to recognize these conditions either in the chapter on articular neuralgia (*vide* page 528), or in the previous chapter. Here, too, the psychical factor involved in the injury certainly plays the most important part in the ætiology.

We should, however, at present still distinguish the special traumatic neurosis from all these states, although we must admit that we can not draw a sharp boundary between the traumatic neurosis and hysteria, that both states are certainly closely allied, and that the two may also be combined. Theoretically, however, we can make this distinction, that in the genuine traumatic neurosis the purely physical factor of material concussion is of ætiological significance.

In regard to the course of the true general traumatic neurosis almost all observers agree that it is protracted. In fact it is practically important to know that most of the severe cases of this sort are not capable of complete recovery. The patient's complaints and troubles persist for years without becoming much worse, but also without any marked improvement; but the course is often still more unfavorable, since the patient's mental powers may show a progressive decline, and complete dementia may finally develop.

The treatment of the traumatic neurosis is based on the same principles that we have studied in the two previous chapters, in speaking of the treatment of hysteria and neurasthenia. Great stress is to be laid on exerting a favorable mental influence on the patient. The most useful remedies besides are the electric current (galvanization of the spine, galvanization and faradization of the muscles and nerves, and the faradic brush), and also baths and rubbing, and internally quinine, iron, and strychnine.

[It hardly seems proper to regard the "traumatic neurosis" as a single morbid state. In some cases the symptoms point definitely to an affection of the cord, and the mental symptoms are absent. Here the patient complains of pain, weakness of the legs, and disturbances of the sphincters; the sensibility may be diminished, and the reflexes are exaggerated. The most marked changes in the cord have been found in the lateral columns. In other cases the symptoms are clearly those of grand hysteria, but such cases are rare in America. In many cases the symptoms are chiefly neurasthenic. In yet a fourth class there are more pronounced symptoms of mental and physical failure; anæsthesia is marked, the muscular strength is slight, and the symptoms mentioned above are pronounced. These cases may terminate fatally after a few years, and are probably cases of diffuse sclerosis of the brain and cord. Traumatic lumbago is a common complication.—K.]

DISEASES OF THE KIDNEYS, THE PELVIS OF THE KIDNEY, AND THE BLADDER.

SECTION I.

DISEASES OF THE KIDNEYS.

CHAPTER I.

GENERAL PRELIMINARY REMARKS UPON THE PATHOLOGY OF RENAL DISEASE.

ALTHOUGH some knowledge of the occurrence and significance of renal affections had been acquired even by the older physicians, still the service of having pointed out the frequency of these diseases, and of having clearly recognized their most important anatomical forms and their chief clinical symptoms, belongs undoubtedly to the English physician Richard Bright, who was born in 1788 and died in 1858, as physician in ordinary to Queen Victoria. Bright's first work on this subject appeared in the year 1827. In this he brought forward the special discovery that, in many cases of general dropsy, which are associated with the secretion of an albuminous urine, a primary affection of the kidneys must be regarded as the special cause of the disease. Since then, the disease described by him has been almost universally called "Bright's disease" ("*Morbus Brightii*"), a name still much employed, but in whose stead the anatomical terms would be more proper, since many forms were previously classed under it which, according to our more accurate present knowledge, must be separated.

Bright's statements were either confirmed or expanded in subsequent times by many other observers. Christison, Osborne, and R. Willis in England, and Rayer and M. Solon in France, were the chief students of renal diseases. Frerichs published the first great work in Germany in the year 1851. His division of Bright's disease into three different "stages," based on Reinhardt's histological investigations, was for a long time quite generally accepted, until gradually further clinical experience showed that it was untenable. A more accurate division of renal diseases was first opposed to it in England (Johnson, S. Wilks, and others), and then in Germany (Traube, Bartels). Under the incentive of these labors, especially the work of Bartels in 1871, renal pathology thus fell into a doubtful classification, with which the facts of experience could be harmonized only by force. Only of late years has a natural theory of renal diseases, derived from general pathological observations, at last become accepted—a theory which is based chiefly upon the anatomical work of Weigert, but which may also be brought into complete harmony with the data of clinical observation.

The chief reason why the kidneys are so often diseased, either alone or in conjunction with other organs, is to be found in the fact that the body must eliminate all forms of injurious matter, which circulate in the blood, in great part by the kidneys. Consequently the action of any injurious substance is often manifested chiefly in the kidneys, since they must, in a certain measure, be repaid for the service which they do for the rest of the body by their own disease. According to their nature and character, the injurious substances, which are here to be considered, are divided chiefly into two great groups—the chemico-toxic and the organized infectious substances. In this way the kidneys may be involved sympathetically after the ingestion of many poisons, and also in the great majority of all the infectious diseases. In these cases, of course, as we shall see later, certain chemical and infectious poisons exert their action in a particularly frequent and in a particularly severe or definitely characterized fashion. Beside these forms of origin for many renal diseases, which are the chief ones to be considered, we must consider other causes of disease which are much rarer. One way in which the morbific agents may also enter is especially important—namely, from the lower urinary passages, the bladder, and pelvis of the kidney upward into the kidney. In this way those renal diseases arise which come on secondarily to cystitis, pyelitis, etc. Finally, of course, disturbances of circulation and mechanical traumatic injuries may also make themselves manifest in the kidneys.

The clinical symptoms which are caused by the different forms of renal disease, and which serve for its recognition, are referable only in very small part directly to the diseased organ itself. In renal diseases characteristic subjective local symptoms—like local pain—are but rare, and the anatomical position and the physiological conditions of the kidney make it almost impossible to discover any changes in their size, their physical consistency, etc., by a direct objective examination. In the diagnosis of renal diseases we are therefore confined chiefly to the investigation of two groups of symptoms: in the first place, to the examination of the secretion from the kidneys, the urine, whose character, as we know by experience, may be materially altered when there is renal disease; and, in the second place, to the discovery of certain phenomena in other portions of the body, which are immediately dependent upon the renal affection. Since both the pathological changes in the urine, and the symptoms in other organs occurring in renal affections, have much in common in almost all the forms of renal disease, it is advisable first to describe the main features, at least, of the general symptomatology of renal diseases. We shall then be obliged, in the following special chapters, to mention only the precise circumstances of the occurrence and onset of each symptom; the general significance of the symptoms being already known.

1. ALBUMINURIA.

The most constant symptom, and one which in many cases first of all, and often even alone, renders the diagnosis of a renal affection possible with complete certainty, is albuminuria—that is, the appearance of albumen, and especially of serum albumen and serum globuline (paraglobuline), in the urine. From recent investigations (Leube, Fürbringer, and others) we know that in some cases the urine may contain a very slight amount of albumen even in healthy persons, especially after physical exertion, in emotional disturbances, etc. These rare exceptions, however, do not invalidate the correctness of the assertion that when a definite amount of albumen is persistently eliminated by the urine it must be regarded as something pathological.

The detection of albumen in the urine for clinical purposes, wherein no regard need be paid to the separation of serum albumen and serum globuline, is performed almost exclusively by means of the so-called heat test. If the urine is cloudy, it must be filtered before heating. The reaction of the urine must always be tested first. If it is acid, as it usually is, the urine is heated in a test-tube without any further addition.* If the reaction of the urine is neutral or alkaline, we acidify it, before heating, with a few drops of acetic acid. If the urine contains albumen, a decided flocculent precipitate of coagulated albumen appears on heating it. We can make a mistake only where there is an alkaline reaction in the urine, which sometimes happens in neutral or very faintly acid urine, owing to the escape of carbonic acid during the heating, and the consequent precipitate of phosphates, especially of calcic phosphate. In order not to mistake such a precipitate of phosphates for a precipitate of albumen, it is necessary, after the urine has been heated for a short time, to add to the precipitate, if present, nitric acid (an excess does no harm). A precipitate of phosphates is dissolved at once, but a precipitate of albumen usually becomes thicker and more compact. We can measure the amount of albumen contained in the urine approximately by the height of the settled precipitate on the bottom of the test-tube. We often speak of "one half or one fourth of the volume being albumen," but we can not state any definite relation between this estimate of the volume and the precise amount of albumen. Of other tests for albumen we will mention here the very accurate one with ferrocyanide of potassium and acetic acid. If we add acetic acid to a urine containing albumen, and then add drop by drop a solution of ferrocyanide of potassium, a very distinct precipitate of albumen is at once formed.

If we have found out that the urine certainly contains albumen, we must then decide whether we have really a true renal albuminuria—that is, whether a urine already albuminous is secreted in the kidneys, or whether the albumen is not mixed with a perfectly normal or at least non-albuminous urine later, in the kidneys themselves or in the urinary passages, the pelvis of the kidney, or the bladder (spurious, accidental albuminuria). Such a spurious albuminuria occurs when the urine is contaminated with blood (as in hæmorrhages from the kidneys, the pelvis of the kidney, the bladder, or the urethra), or with pus (in pyelitis, cystitis, etc.). In these cases, of course, the albumen contained in the serum of the blood or pus is found in the urine. Spurious albuminuria is usually easily recognized, since the presence of pus or blood in the urine, which is shown by the appearance of the urine or upon microscopic examination (red blood-corpuscles, pus-corpuscles), points with immediate certainty to the origin of the albuminuria. Beside that, the amount of albumen in these cases is usually but slight, and corresponds to the amount of pus or blood in the urine. A disproportion in this respect must excite the suspicion whether, beside the spurious albuminuria, there is not perhaps at the same time an affection of the kidneys causing a true renal albuminuria. The determination of this point is not always perfectly easy, but we can usually come to a decision by finding abnormal morphological constituents of the urine, the so-called urinary casts (*vide infra*), which give indubitable evidence of the existence of a disease of the kidneys.

What general pathological significance has the true renal albuminuria, and what are the causes of its origin? According to our present theories, the answer to these questions is simply this: Almost every pure albuminuria is a direct sign of an abnormal perviousness of the walls of the glomeruli; and the pathological changes, which the glomeruli undergo in the different diseases of the kidney, have as their immediate result this abnormal perviousness, and the consequent transudation of albumen into the urine. The fact that the easily filtrated serum albumen of the blood, as well as the water, does not pass through the vascular loops of the glomeruli, even under normal conditions, is due entirely to the circumstance that the capillaries of the Malpighian bodies are not inserted bare into the beginning of the uriniferous tubules, but that they are covered with epithelium. This epithelium of the glomeruli has the task and the power of providing for the retention of the albumen in the blood. If it suffer a pathological change in any way, it loses this power, and then the albumen passes into the urine (Heidenhain). The simplest experimental proof of this theory is furnished by the albuminuria which appears whenever the supply of arterial blood to the kidney is checked by a temporary constriction of the renal artery. The epithelium of the glomeruli thereby suffers a visible microscopic change, as its nuclei are found considerably swollen. If the kidneys in this condition are removed as rapidly as possible and boiled, according to Posner's suggestion, we can discover under the microscope in the capsules of the glomeruli the albumen that is thus coagulated (Ribbert)—the most certain sign that the passage of the albumen from the blood-vessels into the urinary passages has in fact taken place in the glomeruli.

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* The heat-test becomes still more certain, but it is rather more elaborate, if we first add to the urine a few drops of acetic acid and about one sixth of its volume of a concentrated solution of common salt or Glauber's salt, and then heat it.

Almost all cases of albuminuria may readily be referred to analogous disturbances of nutrition in the epithelium of the glomeruli, whether they be excited by anomalies of the circulation, like arterial anæmia or venous stasis, by toxic or infectious influences which have reached the glomeruli, or by any other circumstances. In these cases the changes in the glomeruli need not always be of a very severe or irreparable nature; for we often see a slight albuminuria appear under the most different conditions, and rapidly pass off again. This is the so-called "transitory albuminuria," which is seen, for example, in various febrile affections, after slight intoxications, after epileptic attacks, or in other severe nervous conditions, in lead-colic, etc. We will show later, in the separate chapters, how the anatomical changes which are found in severe renal diseases explain the occurrence of albuminuria.

The other factors, which have also been made answerable for the origin of albuminuria, are without doubt quite subordinate to the changes in the epithelium of the glomeruli, and at most they can affect the amount of albumen eliminated. The changes in the composition of the blood, on which formerly great stress was laid, especially the hydræmia and hypalbuminosis (the diminished amount of albumen) of the blood, have probably only an indirect significance, since the nutrition of the walls of the glomeruli suffers in such a faulty consistency of the blood, and this circumstance again is the special cause of the elimination of the albumen.

The significance of the blood-pressure for the occurrence of albuminuria was also formerly very much overrated. According to the older hypothesis, it was believed that, in an increase of the blood-pressure, the molecules of albumen in the blood could be pressed through the filter formed by the membrane of the glomeruli. This hypothesis, which was not based upon experiments, has been disproved, especially by the experiments of Runeberg; these experiments showed that, in the filtration of solutions of albumen through animal membranes, a rise in the filtration pressure was followed by a decrease, and a fall in the pressure by an increase of the per cent. of albumen in the filtrate. Runeberg attempted to refer the origin of albuminuria in many cases directly to a diminution of the blood-pressure in the renal vessels; but the attempt, on the ground of these results, is not sufficiently justified. A diminution of the blood-pressure hardly ever is itself followed by albuminuria, and the clinical facts which may be brought to support the above hypothesis may all be explained by the change in the character of the walls of the glomeruli, which is always present at the same time with the decreased pressure.

Although in the preceding paragraphs only the Malpighian bodies have been regarded as the spot where the transudation of the albumen of the blood into the urine takes place, we must also note that, under some circumstances, we may admit the possibility of a passage of albumen directly into the tubules from the capillaries that encircle the uriniferous tubules; but we must also necessarily assume in such cases that there is a disturbance of nutrition in the membranæ propriæ, or at least in the epithelium of the uriniferous tubules. Such an assumption seems to explain the albuminuria, according to Senator's experiments, in venous stasis in the kidneys, although in these cases the epithelium of the glomeruli also suffers soon, and then becomes pervious to albumen.

Finally, we may briefly mention that in some cases other soluble albuminous substances are also found in the urine in renal diseases as well as serum albumen and globuline, especially paralbumen, hemialbumose, etc., but the presence of these substances has not yet attained any practical diagnostic significance.

2. Casts and other Abnormal Morphological Constituents of the Urine in Renal Disease.

Beside albuminuria, certain peculiar morphological constituents of the urine, visible under the microscope, are of especial importance for the diagnosis of renal affections—the urinary casts, whose significance was first correctly recognized by Henle in 1842. These are cylindrical bodies, whose breadth corresponds to the width of the uriniferous tubule, and whose length only exceptionally reaches a millimetre, and which must be regarded in their chemical nature as consisting mainly of a coagulated albuminous substance. To the latter circumstance we owe their old name of "fibrine casts," or "fibrous casts," a name which can no longer be used with propriety, since the coagulated albuminous substance of most casts is, at any rate, not identical with fibrine.

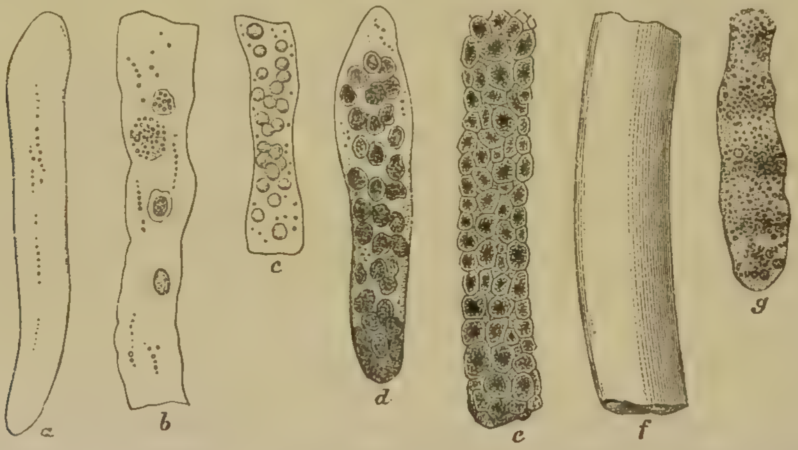

Fig. 111.—Different forms of casts. *a.* Hyaline cast with occasional granules. *b.* Hyaline cast with fat-drops and granular cells. *c.* Hyaline cast with red blood-corpuscles attached. *d.* Hyaline cast with white blood-corpuscles attached. *e.* Epithelial cast. *f.* Waxy cast. *g.* Cast with a large number of fat-drops.

Since the precise conditions of the occurrence, and the character of the renal casts in the different diseases of the kidneys, will be spoken of later, we need discuss here only the general properties, the origin, and the significance of casts (see Fig. 111).

1. *Hyaline Casts.*—The hyaline casts are the commonest and most important form of casts, and, to a certain extent, are the ground-form for different varieties. They are perfectly homogeneous, clear as glass, colorless, soft, and flexible. We find them either wide or narrow, sometimes broken off short, sometimes quite long, usually straight, but in many cases partly curved. They are easily stained with carmine or gentian-violet. On heating the urine, they are dissolved, but they are quite resistant to acids.

The hyaline casts are very often covered to a greater or less extent with all sorts of deposits, which are usually affixed to the soft substance of the cast in the kidney itself, but which may often be attached to it later. These deposits may consist, first, of red blood-corpuscles. This condition is important, because it points with certainty to the existence of hæmorrhages in the kidneys themselves. Second, of white blood-corpuscles. These are often considerably swollen, so that we must guard against mistaking them for epithelium. Third, of renal epithelium, which may be recognized by its size, its more angular shape, and its nuclei. Of course, we often find the epithelium cloudy and granular, or shriveled and atrophied. Fourth, of fatty granular globules—that is, both fatty degenerated epithelium and also white blood-corpuscles which are filled with fat-drops from the fatty degenerated cells. Fifth, of little granular masses whose nature can not always be easily recognized. They are either coagulated granules of albumen, or fat-drops,* or urates, or bacteria, or, finally, granules of hæmatoidine, which have come from the destruction of red blood-corpuscles, and are usually easily recognized by their dark, brownish-yellow color. Sixth, we rarely find in the casts drops like myeline, as to whose precise significance nothing is known.

2. The so-called granular casts are in most cases nothing but hyaline casts which are completely covered with the above-mentioned granular masses, but sometimes the coagulated masses of albumen or the granules of hæmatoidin may themselves assume cylindrical forms.

3. The pure blood-casts are not very common. They consist of coagulated blood, and form casts of the uriniferous tubules into which the hæmorrhage has taken place.

4. The epithelial casts are composed exclusively of renal epithelium, although probably a hyaline cast often affords a basis for the epithelium to cling to. The epithelial casts are usually easily recognized, and always point to a marked desquamation of epithelium in the diseased kidney. We must guard against mistaking renal epithelium for swollen white blood-corpuscles, as we have already said. On the epithelial casts the single epithelial cells may also present different changes, such as granular opacity, fatty degeneration, or atrophy.

5. The so-called waxy casts are almost always quite broad, uniformly yellowish, opaque casts, which proceed, perhaps, from a metamorphosis of the albumen of the hyaline casts. Their special diagnostic significance is unknown. At all events, they are not by any means found chiefly in the amyloid kidney, but they are found most commonly, comparatively, in acute and subacute nephritis.

Nothing definite can at present be stated as to the mode of origin of hyaline casts; the origin of the blood and epithelial casts is self-evident. Hyaline casts are most probably formed from the coagulated albumen eliminated from the kidneys, since the formation of casts is almost always coincident with albuminuria. We can not state definitely how far destroyed white corpuscles or degenerated renal epithelium participate in the formation of casts.

The clinical diagnostic significance of renal casts is very great. They are, in the first place, always a sure sign of the existence of some renal disease, since in normal urine casts are not found at all, or, at most, they are exceptional and are present in small numbers. The consideration of the special forms of casts, and of the deposit upon them, is also of great diagnostic importance, although from it we can never decide immediately upon the general form of the renal disease, but we can recognize with certainty the type of special pathological processes in the kidneys. The blood-casts and the red blood-corpuscles sticking to the cylinders point to the occurrence of renal hæmorrhages; the epithelial casts to a desquamation of the epithelium in the kidneys; the white blood-corpuscles to an emigration of the colorless cells from the vessels; the fatty granular cells and the fat-drops to the presence of processes of fatty degeneration in the kidneys.

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* It is doubtful whether the hyaline casts themselves may in part undergo fatty degeneration.

We have already learned to recognize in a great measure in the preceding, as occasional deposits on the casts, the other morphological constituents found, beside the casts, in the sediment of the urine in renal disease. Briefly recapitulated, they are as follows:

1. Red blood-corpuscles. A large amount of blood in the urine (hæmaturia) is almost always to be recognized by its blood-red color. The presence of blood may be made out with certainty by the microscope, or by Heller's blood-test. The latter is performed by heating the urine in a test-tube with sodic or potassic hydrate. The blood-corpuscles are thus dissolved, and the hæmatine is precipitated with the phosphates, giving to the precipitate of the latter a very characteristic blood-red color. Finally, of course, the spectroscope may serve for the detection of hæmaturia. Hæmoglobinuria will be described in a special chapter later.
2. White blood-corpuscles. Only when they are also attached to the casts can we assume with certainty that these come from the kidneys, and not from the lower portions of the urinary tract.
3. Renal epithelium.
4. Fat-drops and fatty granular cells.
5. Uric-acid crystals, urates and calcic oxalate, bacteria, etc.

3. THE DROPSY OF RENAL DISEASE.

Although the changes in the urine must be alone decisive in the diagnosis of any renal disease, there are yet certain other symptoms which are also due immediately to the renal affection, and which may first direct our suspicions to the existence of a disease of the kidneys, and consequently lead to a careful examination of the urine. Among these symptoms the dropsy of renal disease is one of the commonest and most important. This may, indeed, quite frequently be entirely absent, both in acute and chronic nephritis, and in other diseases of the kidneys; but in many cases it is decidedly prominent in the whole clinical picture.

If we ask what is the reason of the frequent occurrence of dropsy in renal disease, the answer at first does not seem difficult. Since the main function of the kidneys is to excrete water from the body, and since, as we shall see later, in many cases the diseased kidney can no longer fulfill this task, or it can fulfill it only to a slight degree, we are not, in fact, very much out of the way in considering the retention of water in the body as the main cause of the consequent œdema. Clinical observation seems in general to agree completely with this assumption. The œdema in renal disease seldom appears until the daily amount of urine has been below the normal for some time, while, on the other hand, in those cases where the amount of urine passed is normal, or even abnormally great, in spite of the existing renal disease, the œdema is usually wholly absent. In individual cases, too, we very often see a decrease of the œdema associated with an increase in the amount of urine, and an increase of the œdema associated with a corresponding diminution in the excretion of urine. The pathological process accordingly seems to consist of an accumulation in the body of the water which can not be excreted from it, and which transudes from the vessels and thus gives rise to the development of œdema.

On more careful consideration, however, there are some objections to this theory, which is apparently so simple. In the first place, it might be supposed that, when there is a retention of water, the body must get rid of the surplus water by employing to a greater degree the other channels of elimination which are at its service—the skin and the intestines. Since we can never determine accurately the time when the water first begins to be retained in the body, the clinical experience just mentioned may also be thus interpreted, that the lessened excretion of urine is not the cause of the œdema, but that, on the contrary, the appearance of œdema is rather the cause of the diminished elimination of water by the kidneys. For many cases this objection seems somewhat artificial, because the anatomical changes in the kidneys must often, without doubt, have a direct influence upon the secretion of urine ; but, still, this can not be entirely and conclusively demonstrated. The results of Cohnheim's and Lichtheim's experiments also are not in harmony with the above theory of the origin of œdema. By injecting a large amount of a half-a-per-cent. solution of common salt into the vascular system of an animal we can so greatly overload its blood with water as to produce an artificial "hydræmic plethora," and nevertheless there is not the slightest œdema, not even when the animal's renal arteries are tied. In conclusion, we must also state that cases have been seen repeatedly where a complete anuria has existed for several days, as a result of the plugging or compression of the ureters, and where there was nevertheless not a trace of œdema.

There seems to be, then, another factor beside the retention of water in the body which plays a part in the origin of œdema; but it is not easy to decide what it is. Cohnheim lays the greatest stress upon a change in the walls of the vessels, by which they become abnormally pervious and permit the water accumulated in the blood to pass out into the tissues. This hypothesis seems plausible, especially with reference to the dropsy in scarlatinal nephritis, and in the cases that arise after the skin has been thoroughly chilled; but we must also admit that in many cases such a vascular change has not been certainly discovered.

The discussion so far relates mainly to the origin of the dropsy in the acute and subacute forms of nephritis. In chronic nephritis the œdema, without doubt, often arises in quite another fashion, especially as a result of the disturbance of compensation in the final paralysis of the hypertrophied left ventricle (*vide infra*). This œdema is then a true general œdema of stasis, and may be regarded as analogous to the œdema in uncompensated heart disease.

The special peculiarities in regard to the appearance of œdema in the different diseases of the kidney will be described later. The first signs of the development of dropsy are generally noticed in the skin, usually in the face, and especially in the eyelids. The ankles and legs also swell, then the scrotum, the dependent parts of the trunk, etc. In all severe cases the whole subcutaneous cellular tissue finally takes part in the dropsy, so that the whole body is swollen to the utmost degree. We almost always find at the same time an effusion into the cavities of the body, hydrothorax, ascites, and finally even hydropericardium. In some cases the dropsy of the serous cavities may attain even a high degree without there being very much anasarca—that is, general œdema of the skin. More rarely we see œdematous swelling in the mucous membranes, especially in the conjunctivæ, the soft palate, and the arytæno-epiglottic ligaments (œdema of the glottis). Among the œdemas of internal organs, œdema of the lungs has a great practical significance. The questions as to the occurrence and significance of œdema of the brain will be spoken of below (see uræmia).

In its chemical composition, the dropsical fluid corresponds to a very thin blood-serum. The amount of water is usually 97 to 98 per cent., the amount of salts one to one and a half per cent. The amount of albumen is usually very slight. Urea has been repeatedly found in it.

4. URÆMIA.

If the diseased kidneys can no longer perform their secretory functions in a satisfactory way, not only does the elimination of water from the body thereby suffer, but the soluble constituents of the urine, the salts, the urea, and the other final products of tissue metamorphosis may also be retained in the blood and accumulate there. Hence we often find the blood, in patients with renal disease, not only more watery than under normal conditions, so that the specific gravity of the serum may fall from 1030 to 1020, or even lower, but, in almost all cases where there is a diminished excretion of urine, it is also richer in urea, as many experiments have shown, and under corresponding conditions it is probably also frequently richer in the other constituents of the urine.

This accumulation of the urinary constituents in the blood, and further, perhaps, in the tissues themselves, is the cause of a class of symptoms which are often seen in diseases of the kidneys, and which are termed uræmic symptoms or uræmia. It is probable that the retention of urea plays the main part here, but it is also probable that the retention of other constituents of the urine, perhaps the potassium salts chiefly, is likewise not without significance. Many experiments have shown that, by extirpating the kidneys or by tying the ureters in animals, we can provoke a symptom-complex, characterized by vomiting, convulsions, and coma, which corresponds almost perfectly to the uræmia of renal disease. We almost always see at the same time a very pronounced anæmia of the brain and cord, so that the accumulation of the products of tissue metamorphosis seems to excite a vascular spasm (Fleischer). We must also, perhaps, consider this secondary cerebral anæmia as well as the direct toxic action, in studying the origin of the severe nervous uræmic symptoms. That large amounts of urea injected into the blood of healthy animals usually have no injurious results, is explained simply by the fact that in this case the urea is very rapidly and completely eliminated again by the kidneys. If, when feeding an animal with large amounts of urea, we hinder its elimination, as Voit has shown, by withholding water at the same time, uræmic symptoms also appear.

Clinical experience in most cases also agrees perfectly with the theory that uræmia is caused by a retention of urinary constituents in the body. In most cases the uræmic symptoms appear only when the daily amount of urine has previously fallen to a very low figure, or when the secretion of urine has wholly ceased for several days. That in these cases not only the elimination of water, but also the elimination of an amount of urea corresponding to the food taken, and also the elimination of the other urinary constituents, is very much diminished, is shown by the experiments in regard to this point made by Fleischer and others. Furthermore, a great increase of the amount of urea in the blood in uræmic patients has been found in many if not in all cases.

Of course there can be no question that some clinical facts can not be brought into exact harmony with what has been previously said. If cases are repeatedly reported in which no uræmic symptoms have appeared in spite of anuria for several days, it does not prove too much, since we can never make an exact estimate of the matter accumulated in the blood which ought to have been eliminated; for the organism can certainly get rid of the final products of tissue metamorphosis in other ways than through the kidneys—for instance, through the skin or the intestines—and we must also bear in mind that different individuals show a great variation in tolerating the action of any poison in the body. It is harder, however, to explain those cases which are sometimes seen, where uræmic symptoms suddenly appear in patients with renal disease without being preceded by any noticeable diminution of the secretion of urine. In these cases we must assume that, in spite of the abundant elimination of water—that is, in spite of a normal amount of urine—there is a retention of the solid constituents. Such cases, however, should always make us consider whether, in renal disease, other circumstances than the retention of urinary constituents may not give rise to the development of severe nervous symptoms. In some such cases the appearance of uræmia coincides with the disappearance of the previously existing œdema. It has therefore been conjectured that, in such cases, the blood all at once becomes rich in urea from the rapid absorption of the œdema fluid which contains urea, and that therefore uræmic symptoms now arise, in spite of the abundant elimination of urine which at once sets in. This hypothesis does not seem to us very probable, since, as was said above, very large amounts of urea may be injected into the blood of animals with healthy kidneys, without the appearance of uræmia. In the cases above mentioned we must therefore still further suppose that nothing but the water is rapidly passed off again by the kidneys, while the solid constituents remain.

Among the other theories of uræmia which have therefore been advanced to explain the apparent contradictions in the clinical observations mentioned, Traube's theory must be mentioned especially; according to this, an acute œdema of the brain is the cause of the uræmic symptoms. There is no doubt that this theory does not apply to many cases of uræmia; but, on the other hand, we can not claim that it never finds an application. It seems to us that, in general, the possibility of actual anatomical changes in the brain in renal disease has not yet been sufficiently considered as a cause of severe nervous symptoms, since the frequent occurrence of special changes in the retina, which also consists of nervous elements, is closely connected with such a theory. In most cases of uræmia we can, however, hold to the original explanation, according to which these symptoms owe their origin to the retention of the urinary constituents in the blood; but we can not exclude the possibility that, under some circumstances, severe nervous symptoms may arise from other causes in patients with renal disease, which of course do not deserve the name of "uræmia," although clinically they may greatly resemble it.

Finally, we may mention here the theory advanced by Frerichs in the year 1851, which at first found much favor, but which at present is almost universally abandoned. According to this, the urea retained in the blood was not in itself the cause of the uræmic symptoms, but it was changed into carbonate of ammonia by the action of a ferment in the blood, and from this the severe nervous symptoms arose. This theory is untenable, because carbonate of ammonia is scarcely ever found in the blood of uræmic patients. It is much more probably formed first in the stomach and intestinal canal of uræmic patients from the urea there excreted (*vide infra*), as Claude Bernard, Treitz, Voit, and others have shown.

In regard to the clinical symptoms of uræmia in the individual case, they show all possible transitions from the mildest symptoms, which are only intimated, up to the severest nervous symptoms, which may be the immediate cause of death. The severe forms of uræmia may sometimes come on quite suddenly, while in other cases they may be preceded for a long time by milder uræmic symptoms, which are then termed prodromata. The severest symptoms may not appear at all, and the milder symptoms may exist alone for a longer or shorter time. This latter condition is called chronic uræmia.

The milder uræmic symptoms, which are observed either alone or as precursors of severe uræmia, consist of headache, somnolence, and mental stupor, of a peculiar uneasiness, or of a feeling of anxiety and constraint (sometimes associated with hurried respiration), and very often of nausea, spasmodic eructations, and repeated vomiting; and, finally, not infrequently, of various symptoms of motor irritation, of slight twitchings or temporary tonic rigidity of the face or the extremities, etc.

The most characteristic symptom of severe uræmia is the uræmic convulsion, or the so-called uræmic eclampsia. It corresponds almost exactly in its details to the pure epileptiform attack; it usually begins with a short tonic stage, in which the whole body is generally in a position of extension in opisthotonos, and then follow vigorous clonic contractions in the face and extremities. The face becomes cyanotic, a bloody froth comes from the mouth, the pupils are usually dilated and almost without reaction, the respiration is accelerated (but at times it is intermitting from spasm of the respiratory muscles), the pulse is small and accelerated, and it can scarcely be felt in the radial artery, and the temperature is sometimes raised. In other cases the spasm begins with short jerky contractions in one extremity, as in the arm, and then invades the trunk muscles, the face, and the legs. One half the body is often more affected in the attacks than the other. The spasms usually cease in a few minutes, and are followed by deep coma and stertor, which last for several hours or more. There is only rarely a single attack. The attacks are much oftener repeated after longer or shorter intervals, so that there may even be twenty or more in the twenty-four hours, during the whole of which time a complete loss of consciousness persists. Severe and fully developed epileptiform attacks often alternate with slighter convulsions.

Some other uræmic symptoms besides the convulsions have already been briefly mentioned, but they merit a somewhat fuller description.

The uræmic amaurosis occasionally seen is especially interesting. It is usually left after recovery from the convulsions. Only rarely does it precede them or appear without them. It always develops quite rapidly, so that the first disturbance of vision soon passes into complete blindness. The reaction of the pupils to light is almost always retained, and the ophthalmoscope shows a perfectly normal retinal image. From this we can scarcely doubt that genuine uræmic amaurosis is of purely central origin; it is probably due to a disturbance in the cortex of the occipital lobe. Its prognosis is on the whole favorable, since the disturbance of vision usually disappears completely in a day or two, though sometimes not until after a longer time. Anomalies are only rarely seen in the region of the other nerves of special sense, the most frequent, comparatively, being a difficulty in hearing, or even complete deafness.

Other motor disturbances, except twitchings and convulsions, are rare. Only in a few cases have hemiplegic or monoplegic paralyses, contractures, etc., been observed. Mental symptoms are more common. Delirium, and maniacal or sometimes melancholic states, occasionally follow uræmic coma.

Those uræmic symptoms have also a great interest which are to be regarded as a sort of self-help on the part of the organism, since they often lead to a vicarious elimination of urea. The first of these is uræmic vomiting, which is a frequent and often an extremely obstinate symptom both in acute and chronic uræmia. In many cases it is of central origin, and is to be regarded as analogous to the vomiting so frequent in different forms of cerebral disease; but it is often produced by the irritation which the gastric mucous membrane suffers from the urea eliminated, or rather from the carbonate of ammonia arising from it. The latter is always first formed from the urea in the stomach itself, and we find in the vomitus of uræmic patients either the still undecomposed urea or the carbonate of ammonia in considerable quantities. Sometimes there is quite a violent hiccough besides the vomiting.

Uræmic diarrhœa has the same significance as uræmic vomiting. It is usually provoked by the carbonate of ammonia arising from the urea in the intestines. The latter often causes quite a severe catarrhal, and even at times a diphtheritic, inflammation of the intestinal mucous membrane.

Another way in which the organism sometimes tries to get rid of the amount of urea accumulated in it is by the sweat-glands. Schottin first described the remarkable discovery of a coating of urea on the skin in the uræmia of cholera, an observation which since then has been repeatedly confirmed in other cases of uræmia. This coating is most frequently seen on the face, especially on the sides of the nose, to which little faintly lustrous scales are seen sticking after the evaporation of a clammy sweat. Chemical examination shows that these scales are urea. The excretion of urea is much more rare in other parts of the skin, but perhaps the occasional severe uræmic itching of the skin is due to an irritation of the cutaneous nerves by some of the constituents of the urine that are excreted.

Other organs besides the skin and the digestive tract are but rarely to be considered as a means of the vicarious elimination of urea, but Fleischer was once able to discover considerable amounts of urea in the sputum of a uræmic patient.

In conclusion, we must describe the condition of the pulse, of the temperature, and of the respiration in uræmia. The pulse is often very slow before the appearance of severe symptoms, sometimes 48 or 40, but it is almost always tense and hard. In chronic uræmia, also, a moderate slowness of the pulse is not infrequent. When uræmic convulsions appear, however, the pulse usually becomes small and very frequent, especially in cases that terminate unfavorably. The temperature but rarely remains unchanged in severe uræmia. If there are convulsions, it usually rises several degrees, in severe cases even to 106° or 108° (41°–42° C.). We have seen these high temperatures, especially as a terminal rise with an unfavorable issue, although there may sometimes be an improvement even in such cases. On the other hand, there are also great declines in temperature, down to 93° or 91° (34°–33° C.), most frequently again as a terminal temperature of collapse, in cases which end in deep coma without marked symptoms of motor irritation. We might also mention the "uræmic chills" which we have seen several times—that is, a chill coming on suddenly along with other uræmic symptoms, with a great increase of temperature, and followed by a rapid fall in the temperature. The respiration in uræmic patients is sometimes very much accelerated, and is especially deep—a symptom which recalls the peculiar breathing in diabetic coma (*vide infra*). Certain severe attacks of dyspnœa in patients with renal disease have been described as "uræmic dyspnœa" or "uræmic asthma"; but it is not always easy to decide whether this is really a uræmic nervous symptom in these cases, since similar conditions of sudden dyspnœa may depend upon insufficiency of the left ventricle or upon inflammatory affections of the lungs.

In regard to the general course of uræmia, we have already spoken of the different ways in which it occurs, either coming on quite suddenly or announcing itself by different prodromata. In most cases the special exciting cause of uræmia is to be found in a failure of the renal activity caused by the anatomical lesion of the kidneys, either because the glomeruli are unable to perform their functions owing to the disease, or because the uriniferous tubules are considerably plugged by casts, or from similar reasons. In the more chronic forms of nephritis with cardiac hypertrophy (*vide infra*) the activity of the heart sometimes plays a very considerable part in the occurrence of uræmia, since, when insufficiency of the left ventricle sets in, the insufficiency of course leads to a fall in the arterial pressure and to a consequent diminution in the excretion of urine.

In regard to the duration of uræmic symptoms and to the different forms and ways in which the various uræmic symptoms may be combined in the clinical picture, we can give only a few general statements. The division of uræmia into an acute and a chronic form, already mentioned, is generally very useful practically. In the acute form we usually have the severe uræmic symptoms, especially uræmic convulsions and uræmic coma. This condition usually lasts some days, while chronic uræmia, in which the milder cerebral symptoms—uræmic vomiting, difficulty in breathing, etc.—are most prominent, may last as many weeks.

The termination of uræmia is always doubtful in every severe case, but it is by no means always unfavorable. Even after coma lasting for several days, with very severe and often-repeated convulsions, the uræmic symptoms may wholly disappear, while on the other hand, of course, uræmia is by no means a rare cause of death in the most diverse forms of acute and chronic renal disease. In judging of the individual case, the most stress is to be laid on the condition of the pulse, the respiration, and the temperature; we must also consider, of course, the character of the urinary secretion, and especially the other morbid symptoms dependent upon the primary disease.

5. THE CHANGES IN THE CIRCULATORY APPARATUS IN RENAL DISEASE.

Although it had not escaped Bright's observation that changes in the heart are also present in diseases of the kidney, this condition was first generally known when Traube, in 1856, in a treatise which has become famous, discovered that a change in the heart was very common in certain renal affections, and thus gave the chief impulse to the numerous clinical and experimental investigations that have been made since then as to the connection between cardiac and renal disease.

This connection, generally considered, may be accounted for in three ways:

First, the heart disease may without doubt be the primary disease, and only secondarily lead to a disease of the kidneys, as a result of disturbances of circulation. In this way develop the kidney of passive congestion (*vide infra*, and page 300) and the embolic processes in the kidney (*vide infra*).

Secondly, heart disease and renal affections may also develop independently of each other, as a result of an injurious influence that affects both organs at the same time. Thus, for example, a general arterio-sclerosis leads to cardiac hypertrophy or to myocarditis, and also to a granular kidney (*vide infra*), as a result of an implication of the renal vessels. Certain other injurious influences, such as toxic and constitutional influences, alcohol, syphilis, or improper living, may also cause a disease of the heart and the kidneys at the same time. Later on, if both affections have developed, their influence upon each other is often, of course, considerable—a circumstance which may render our judgment as to the condition decidedly difficult.

In the third place, finally—and this is the point with which we are here chiefly concerned—the renal affection may be the primary disease, and is itself the cause of a change in the heart, and especially of a secondary hypertrophy of the left ventricle. At present there can no longer be any doubt of the fact of this dependence. We also know now that the secondary development of cardiac hypertrophy is not confined to one form of chronic nephritis, the so-called contracted kidney, as was at first believed, but that it is almost as constant in many other forms of nephritis. Opinions are at present still much divided as to the precise nature of this connection, and as to the causal factors which act here, as the following account will show.

The theory which Traube himself advanced for the explanation of the cardiac hypertrophy in nephritis was that, in the first place, less water is withdrawn from the blood in nephritis for the formation of the renal secretion, and that, in the second place, the flow of arterial blood into the venous system is hindered by the changes in the kidneys. Both circumstances must raise the pressure in the arterial system, and therefore gradually lead to cardiac hypertrophy. Thus formulated, Traube's theory can not be maintained. The first statement especially is untenable, because in many cases of chronic contraction of the kidney with co-existing cardiac hypertrophy there is never a diminution of the elimination of water by the kidneys, and, besides, this can never of itself cause an increase of the arterial pressure. The greatest stress, however, has lately been laid by Cohnheim on the second factor in Traube's theory—on the disturbance of circulation in the kidney—although he modifies the form of Traube's statement. Cohnheim shows that the hindrance to the circulation in the kidneys, which develops chiefly from disease of the glomeruli, must be followed by an increase of arterial pressure, because the flow of arterial blood to the kidneys is not lessened in nephritis. Abnormal resistance to the circulation forms behind the little renal arteries into which an abundance of blood pours, and this may cause an increase of the general arterial pressure.

This theory, however, is opposed by the fact that even complete ligature of both renal arteries does not raise the arterial pressure, because the blood at once escapes into other vascular regions which dilate. The place where the contraction of the caliber of the renal artery occurs, whether in the main trunk or in the terminal branches, can make no difference, because by it only the length of the impeded, or rather, to a certain degree, of the stagnant blood-column, is altered, which is without influence on the general blood-pressure.

Besides Traube's and Cohnheim's "mechanical theory," the "chemical theory" of cardiac hypertrophy, which was advanced in a partial form by Bright, has lately found many advocates (Senator and others). According to this, the retention of urinary constituents, especially urea, in the blood causes the increase of arterial pressure. Of course the quantitative changes to be considered here are comparatively very slight, even if we admit the retention of urinary constituents in all forms of renal disease which lead to cardiac hypertrophy, but the possibility of the final effect of such slight but permanent influences can not be questioned.

We can not at present give a definite decision as to the cause of cardiac hypertrophy in diseases of the kidneys. Experiments to provoke hypertrophy of the left ventricle in animals by artificial disturbances of the circulation in the kidneys, by feeding with urea, etc., have given scarcely any absolute and positive results, so that it seems needless to go into them more fully in this place. It may, however, be assumed as certain that the cause of the increased arterial pressure is to be found in the renal affection itself, and that the hypertrophy of the left ventricle appears only as a result of the permanent increase of pressure. Corresponding to this increase of pressure, the increased tension of the arterial system is often found clinically very early, while the signs of the consecutive hypertrophy of the left ventricle gradually develop later. In the following chapters we will discuss the great compensatory significance belonging to cardiac hypertrophy in renal disease, and how the condition of the heart finally occupies almost entirely the center of the whole morbid picture.

The relation between certain diseases of the vessels and diseases of the kidneys will be spoken of in the chapter on contracted kidney.

CHAPTER II.

ACUTE NEPHRITIS.

(*Acute Bright's Disease.*)

Ætiology.—Acute nephritis, like most of the other forms of nephritis, is not a disease whose ætiology is uniform. The same anatomical change, which we term "nephritis," and which is attended by about the same morbid phenomena, may be excited by influences of very different kinds. Almost all these influences have one thing in common, namely, that, as we have stated in the preceding chapter, they reach the kidneys by way of the circulation and are here in part eliminated, and thus exert their specific injurious action upon the parenchyma of the kidneys; but they differ considerably from one another in their precise chemical or biological nature. Since the pathological change in the kidneys depends upon the amount of the noxious material, upon the intensity of its action and the duration of its influence, we see that the cases of nephritis that arise in this way must present a perfectly continuous series from the mildest to the severest, from those that pass off rapidly to those that last perhaps for years and years. The history of renal pathology teaches us in the plainest way that all attempts to divide the forms of nephritis into different clinical and pathological "varieties" can not be strictly carried out. The more scientific experience increases, the more numerous must be the forms established, and still we only too frequently have to assume all sorts of "transitional forms" merely to bring the reality into harmony with the scheme. It therefore corresponds merely to our practical needs if we take certain types from this whole list and divide nephritis into various groups; for, from the nature of the case, there can be no question of a sharp separation of the various forms.

We accordingly call those inflammatory renal affections acute nephritis which arise comparatively rapidly from any of the injurious influences soon to be enumerated, and which terminate, after a few days or a few weeks, either fatally or with recovery. Acute nephritis, on the one hand, follows immediately, without any fixed boundary, the mildest morbid changes in the kidney, which are usually not termed actual nephritis, but simple "parenchymatous degeneration"; while on the other hand it shows a continuous transition to those forms which last for several weeks or months, or longer, and hence are called subacute or subchronic nephritis.

Among the causes of acute nephritis we must mention first a large group which consist of infectious influences (acute infectious nephritis). In these cases the nephritis usually develops as a secondary complicating disease upon an already existing primary infectious disease, and may be regarded in most cases as a special "localization" of the specific morbid poison present in the body. In other cases there may be chemical substances which act injuriously; these may develop in the body under the influence of infection, be excreted by the kidneys, and thus exert a morbific action on the renal tissue. There is scarcely a single infectious disease in which there may not be occasionally a renal affection as a complication. We observe these affections in our experience much more frequently in certain infectious diseases than in others, so that we may rightly assume that certain infectious substances have a special injurious relation to the kidneys. Since we have already dwelt upon the occurrence, the frequency, and certain peculiarities of secondary nephritis in the description of the different infectious diseases, a brief recapitulation of the facts, which have already been for the most part described, will suffice here.

The infectious disease which most frequently gives rise to an acute nephritis is scarlet fever. As has been shown previously (see page 41), the renal affection appears but rarely at the beginning of the disease, and then in a very mild form, while the special severe scarlatinous nephritis usually attains its development only toward the end of the third week of the disease. In measles, secondary nephritis is very much rarer than in scarlet fever; in rötheln it is only of very exceptional occurrence. It is commoner again in smallpox, especially in the severe hæmorrhagic forms. In varicella, renal affections are very rare, but they have been occasionally observed. They are always of but slight intensity. In typhoid fever a slight albuminuria is very common, but genuine acute nephritis is quite rare. There are some cases, however, where a nephritis appears very early, and where the other typhoid symptoms are so crowded into the background by it that it is decidedly more difficult to make the diagnosis of typhoid; this is called the "renal form of typhoid fever." In typhus and recurrent fevers severe cases of nephritis are not especially common, but they are seen more frequently than in typhoid fever.

The nephritis that often comes on in cholera is of great practical importance. This is seen in the earlier stages, and is especially one of the most frequent causes of the so-called cholera typhoid (see page 86). Of course it may appear questionable whether the renal affection here is always of an inflammatory infectious nature, or develops only in consequence of the disturbance of circulation.

Nephritis develops quite frequently in the course of diphtheria, especially in severe cases of this disease; but the renal affection only rarely reaches a high degree. We sometimes see, however, very severe forms of nephritis in the so-called septic diseases (septic nephritis, see page 112), in acute ulcerative endocarditis and endocarditis verrucosa, and allied affections, such as puerperal fever, septic wounds, etc. Among other acute diseases which, in comparatively rare cases, may be accompanied by nephritis, we may mention croupous pneumonia,

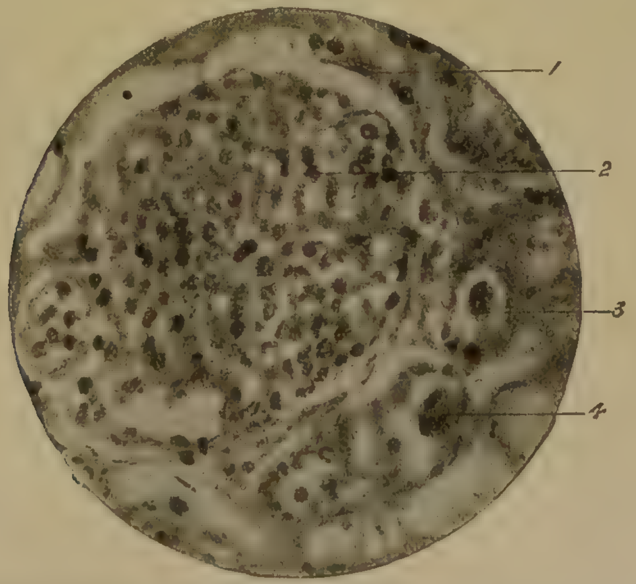

FIG. 112.—Acute nephritis (scarlatinal, early stage). 1. Bowman's capsule: 2. Glomerulus, increase of nuclei and swelling of ground substance; 3, 4. Hyaline casts in renal tubes, the appearance of the nuclei in places is such as to suggest nuclear figures. Obj. $\frac{1}{8}$, without eyepiece. (Eosinated Logwood.)

epidemic meningitis, erysipelas, certain forms of sore throat, certain acute intestinal affections, acute articular rheumatism, and tetanus.

Finally, acute nephritis may also develop in the course of chronic infectious diseases, especially tuberculosis and syphilis. We have ourselves repeatedly seen a mild or even a severe acute nephritis come on in the secondary stage of the latter disease. A genuine acute nephritis may also arise in the course of pulmonary tuberculosis, but for the present we can not decide whether this is directly connected with the tuberculosis, or whether, as is more probable, it arises in consequence of the absorption of septic substances from the contents of the cavities.

We ought also to speak of the acute nephritis sometimes seen in patients with pustular eruptions, such as impetigo, pustular eczema, severe scabies, etc. It may, however, appear doubtful whether the cause of the nephritis is to be sought in the absorption of some injurious substance from the diseased skin, or whether the disturbance of the cutaneous activity may not in itself exert a deleterious influence on the kidneys.

Besides the infectious forms of nephritis just described, there is a second great group, which may be classed under the general heading of toxic nephritis. In these cases we are dealing with the deleterious action of chemical substances which enter the body from without and are excreted from it by the kidneys. It is wholly impossible to enumerate all the substances which have this injurious effect; we will therefore confine ourselves to mentioning those of the greatest practical importance. Among the poisons proper we may mention the mineral acids, sulphuric, hydrochloric, and nitric acids, oxalic acid, phosphorus, arsenic, lead, and chromate of potassium. Among remedies used internally, which may excite nephritis when given in too great doses, we may mention cantharides, squills, balsam of copaiba, turpentine, salicylic acid, and chlorate of potassium. It is also very important to know that many remedies applied to the external skin are absorbed by the skin, and in this way may reach the kidneys and excite severe changes there. Among these are cantharidal plaster, preparations of tar, petroleum, styrax, naphthol, and pyrogallic acid. We must mention, in addition to these, the renal affection which may arise from the too abundant use of carbolic acid or iodoform to the surface of open wounds. Under some circumstances renal affections may even arise in individual cases from taking excessive amounts of certain foods and drinks, such as spices, alcohol, or very acid foods.

In the forms of acute nephritis which can not be immediately referred to infectious or toxic influences, and whose number is comparatively small, we can sometimes find no definite cause at all. We then speak of a primary idiopathic acute nephritis. We are indeed justified in thinking of causes of origin here similar to those in the cases previously described, but causes which at present escape direct detection. In other cases an acute nephritis immediately follows exposure to severe cold or wetting of the skin. We can not doubt the possibility of such a connection after quite numerous and indubitable clinical experiences, although the exact conditions to be considered here are still almost wholly unknown. We usually take refuge in a rather noncommittal reference to the "well-known connection between the activity of the skin and of the kidneys." The experiments on this point have also confirmed the fact itself, but have furnished no precise explanation of its cause.

We have still to mention the nephritis of pregnancy (*nephritis gravidarum*) as a special form of acute nephritis. This usually comes on in the last months of pregnancy in women previously perfectly healthy, and is decidedly more frequent in primiparæ than in the course of later pregnancies. The precise causes of the nephritis of pregnancy are still very obscure. Some authors lay the blame on the pressure of the pregnant uterus on the renal vessels, others on its pressure upon the ureters, etc. (compare the text-books on obstetrics).

Finally, we must mention that an acute nephritis may ensue on a chronic nephritis that has existed for a long time, perhaps without symptoms (acute recurrent nephritis of Wagner).

Pathological Anatomy.—The anatomical changes, which are excited in the kidneys by the causes mentioned in what has gone before, show a continuous series from the mildest to the severest degrees, according to the intensity of the injurious action. The mildest changes, which, as we have said, are not called actual "inflammation," but usually simple parenchymatous degeneration, affect exclusively the parenchyma of the kidney—that is, the epithelium—while the interstitial tissue, the connective tissue, and the vessels remain perfectly normal. This fact is especially important since it implies that, in almost all the injurious influences acting on the kidneys, the specific renal parenchyma itself is diseased first and before any other. On macroscopic examination, the kidneys may show scarcely any plainly perceptible changes, but it sometimes strikes the practiced eye that the kidneys are a little enlarged, that the cortex on section shows either a more reddish-gray, dimmed coloring (cloudy swelling), or a more grayish-white, yellowish hue (fatty degeneration). The microscopic examination gives more accurate information as to the degree and the extent of the disease. We distinguish different conditions according to the form of change in the epithelium, of which the three following are most important: 1. *Cloudy Swelling:* It is most easily made out in the epithelium of the cortical tubules, but it may also be seen in the epithelium of the glomeruli. The cells swell, their contents become uniformly granular and cloudy, the nuclei swell, and finally disappear. Such changes are often found in acute infectious diseases, like typhoid, small-pox, and diphtheria. 2. *Fatty Degeneration:* This may proceed from the cloudy swelling, or may develop independently. Many fat-drops appear both in the cells of the uriniferous tubules and also in the epithelium of the glomeruli, and they may finally lead to the disintegration of the cells. Simple fatty degeneration of the kidneys is sometimes found in acute infectious diseases, after certain poisons, like phosphorus, and finally in anæmic conditions. 3. *Necrosis of the Renal Epithelium:* The nuclei of the cells disappear, and the cells are changed to clear homogeneous flakes, while in some cases they are greatly swollen ("dropsical degeneration" of Nauwerck-Ziegler). Genuine epithelial necrosis is found in the kidneys, chiefly after the action of toxic substances—like cantharides, the chromic and chloric salts, etc.—but sometimes also in infectious diseases. Combinations of simple necrosis with granular cloudiness and fatty degeneration are not infrequent. Both the last-named states may undergo resolution if they have not reached a high degree. Otherwise all the degenerations mentioned lead to the destruction and disintegration of the cells; nevertheless, a complete restoration is possible, from the regeneration of new epithelial cells from epithelium that is still present.

We term those changes in the kidneys genuine acute nephritis, in which not only the renal parenchyma proper, the epithelium, but also the interstitial tissue, especially the vessels, is affected; so that we can make out the exudative changes characteristic of all inflammatory processes—the escape of fluid and cells from the vessels. In these cases the different histological processes may be combined in the most varied ways, so that the anatomical picture presents quite great variations, although it is almost always principally concerned with the same processes.

If we begin with the histological lesion in acute nephritis, in order to learn to recognize at once the essential changes, we have first precisely the same processes of degeneration in the epithelium which have been already described, but they are usually present here in a more marked degree. In some cases the simple necrotic processes predominate; in others, the fatty degeneration. We often find degenerated cells, and not infrequently a more or less marked desquamation of epithelium. We see besides the special inflammatory changes. We find a fluid inflammatory exudation, rich in fibrine, and therefore soon coagulating in the interstitial connective tissue, which is dilated and swollen by it—inflammatory œdema. The same exudation is also found in the uriniferous tubules, and, by the proper methods, by alcohol or by boiling the fresh kidney, the albuminous effusion can be made out both in the capsules of the glomeruli and in the uriniferous tubules. The interpretation of the exudation is, of course, made very difficult, or often wholly impossible, by the presence of albuminous urine in the uriniferous tubules. The second characteristic of inflammation, the "cellular exudation"—that is, the emigration of white blood-corpuscles—is also present. In the interstitial tissue we find accumulations of round cells, usually distributed in foci, and white blood-corpuscles in greater or less numbers also enter the interior of the uriniferous tubules. We often find many hyaline casts in the lumen of the straight tubules or of Henle's loops, whose origin is, in all probability, connected with the albuminous exudation and the emigrated white blood-corpuscles (see page 776). The vessels themselves are often hyperæmic and dilated, but in some cases they are compressed by the interstitial inflammatory œdema. It is of special significance that in very many cases there are hæmorrhages, either into the interstitial tissue or into the interior of the uriniferous tubules, or even into Malpighi's capsules.

All the changes described are not always found uniformly distributed over the whole kidney. Some parts are often much diseased, others but slightly, while others still are nearly, if not quite, intact. We may accordingly speak in individual cases of a diffuse or of a localized nephritis, although there are no strict limits here.

If the histological processes have been made clear, the understanding of the macroscopic appearance of the inflamed kidney is very simple. We can understand that either this or that "form" of acute nephritis must be present according to the predominance of this or that histological process. If an abundant interstitial exudation is present, the kidney is much enlarged; if this exudation is slight, the kidney varies but little, or not at all, from its normal size, notwithstanding any other severe changes. In the first case it usually feels soft, from inflammatory œdema; in the second case, it is comparatively firm. If there is a marked hyperæmia of the kidney, it appears much reddened; if the kidney is anæmic, it is paler; and if an extensive fatty degeneration is also present, it is yellowish-white or yellow. If hæmorrhages are present, they can easily be recognized with the naked eye on the outer surface beneath the capsule as dark-red points that can not be wiped away. We speak then of an "acute hæmorrhagic nephritis." On section, the medullary substance is more or less dilated, its normal striated appearance is almost always obliterated, and its color shows the same variations as the outer surface of the kidney. Since, as we have said, the nephritic changes often show not a uniform, but a nodular arrangement, we can understand that the kidneys sometimes have quite a mottled appearance, since hyperæmic or hæmorrhagic red spots alternate with the lighter anæmic and the yellow fatty-degenerated parts.

There are, accordingly, cases of nephritis which show almost nothing abnormal to the naked eye, while, on the other hand, there are hæmorrhagic and non-hæmorrhagic forms, appearing pale, yellow, red, or variegated, none of which can in the essential features be separated from one another, but which are combined with one another in all conceivable ways. The forms of nephritis that differ in ætiology have, to a certain degree, definite and characteristic anatomical types, but strict rules can not be laid down in regard to this.

In addition, quite a characteristic anatomical form of acute nephritis deserves a brief mention, in which the changes are almost exclusively limited to the glomeruli, and which are therefore called *glomerulo-nephritis* (Klebs, Friedländer, Ribbert). In the purest cases of this form, as it is seen especially in scarlet fever and also in other infectious diseases, we find degeneration and abundant desquamation of the epithelium only in the glomeruli of the kidneys; and we usually find, besides, a marked disease of the vessel-walls, which swell and acquire a homogeneous hyaline appearance. The glomerulo-nephritis can not in its essentials be perfectly definitely separated from the other forms of acute nephritis, since in these, under some circumstances, the glomeruli may first be chiefly affected; but, as it seems, this need not happen in all cases by any means.

Clinical History.—The most essential symptom of acute nephritis is the abnormal character of the urine. In most of the milder, and even in many of the severer cases of nephritis, the change in the urine is the sole objective clinical symptom which renders the diagnosis possible. The physician must, therefore, make it his practice to submit the urine to repeated examinations in every case of disease where there is any possibility of the presence of a nephritis.

The simple parenchymatous degenerations of the kidneys, cloudy swelling, fatty degeneration, etc., which we will first briefly touch upon, may probably sometimes exist without being followed by any discoverable change in the urine; but they often lead to a slight albuminuria, which is easily explained from the change in the epithelium of the glomeruli. If, then, the urine contains a slight amount of albumen (which usually soon passes off) in the course of any febrile infectious disease or other affection (the so-called febrile albuminuria, etc.), we are justified in assuming some of these mild conditions of degeneration in the kidneys. Usually the urine shows no other peculiarities, but sometimes we find in the sediment a few hyaline casts, a few white blood-corpuscles, etc. As we have repeatedly stated, these conditions pass into nephritis proper without any sharp limitations.

Character of the Urine in Acute Nephritis.—In almost every severe nephritis the amount of urine for the twenty-four hours is more or less diminished. This is either caused directly by the lessened elimination of water by the kidneys, or by the plugging of many uriniferous tubules by casts, desquamated epithelium, etc. The amount evacuated daily is often only fifteen or twenty ounces (400–700 c. c.), but it sometimes falls to a much lower figure, two or three ounces (100–50 c. c.), and there may finally be even complete anuria. In general, though not without exceptions, the diminution of the amount of urine runs parallel to the severity of the anatomical changes in the kidney. Improvement in the disease is very often first seen in an increase of the amount of urine. If there was a previous œdema, and this is absorbed, the daily amount of urine often rises during convalescence to a very considerable quantity, eighty to a hundred ounces (2500–3000 c. c.).

The specific gravity of the urine is at first usually increased, since the urine is poor in water, but comparatively rich in solid constituents, especially in albumen (*vide infra*). Of course there are great differences here, and a urine secreted in an abnormally small amount may show a specific gravity of only 1010 or 1015, while, on the other hand, urines with a specific gravity of 1020 to 1030, or even more, have been observed. If during convalescence a very abundant, watery urine is passed, it of course usually has a low specific gravity, 1005 to 1008.

In many cases, but of course not in all, we may suspect the abnormal character of the urine from its appearance. This depends chiefly upon an admixture of abnormal morphological constituents. If these are present in large numbers, as is usually the case, the freshly passed urine is cloudy, and deposits a more or less abundant floccular sediment. The appearance of the urine is most altered if blood be mixed with it (hæmorrhagic urine). According to the amount of blood, the urine is a light or dark red, or even a dark black-red, and often has a greenish reflection when the light falls on it.

The microscopic examination of the sediment gives more accurate information upon the different morphological constituents. We can not, of course, enumerate all the possibilities that may exist in these cases (see page 826 *et seq.*). It is generally the case that in most of the severe forms of acute nephritis the urine contains many casts of all sorts, usually hyaline, but sometimes partly fatty or waxy, and very often covered with red or white blood-corpuscles, epithelium, detritus, etc. The different cases are often characterized by a striking predominance of some one constituent—epithelium, white blood-corpuscles, or red blood-corpuscles—but no special rules in regard to this can be given. We have spoken previously (page 827) of the special conclusions we can draw from the different objects found in the sediment. We can accordingly distinguish an acute hæmorrhagic, or non-hæmorrhagic, an acute desquamative, and a fatty degenerative nephritis, but we must always bear in mind that all these forms pass into one another without sharp boundaries.

The chemical examination of the urine gives, as the most important and constant result, usually a considerable amount of albumen. Since the reaction of the urine is almost invariably acid, the albumen is immediately precipitated on heating the urine, and sinks to the bottom of the test-tube, where it usually takes up about one half or three fourths of the volume of urine used for the heat-test. More accurate quantitative determinations of albumen give most frequently in acute nephritis an amount of albumen of from three tenths to one per cent.; higher percentages are rare. The daily total amount of albumen eliminated amounts to about one or two drachms (5–8 grammes), or sometimes more, but the daily loss of albumen from the body hardly ever exceeds the amount of five drachms (20 grammes). The variation in the amount of elimination of albumen in different cases is quite noticeable.

The examination of the other solid constituents, which is not generally employed in practice, usually gives a diminished secretion of urea, phosphoric acid, etc., corresponding to the diminution in the whole amount of urine.

THE OTHER SYMPTOMS OF ACUTE NEPHRITIS.—Local symptoms in the kidneys themselves are only rarely present. There is, of course, a certain tenderness in the region of the kidneys, which, however, is too ambiguous to have a great symptomatic importance. It is more frequently the case that the abnormally concentrated urine causes the patient to micturate more frequently than usual, and that micturition itself is associated with a disagreeable burning—a sort of vesical tenesmus.

The subsequent symptoms of acute nephritis, which appear in the rest of the body, and among which dropsy takes the first place, are far more important than the local symptoms. Although œdema may be entirely absent in acute nephritis, it is present in most severe cases, and often is predominant in the clinical picture. We must always be prepared for its appearance, especially when the amount of urine shows a considerable diminution.

The œdema is usually discovered first in the face, which has a bloated, and often a pale and somewhat shiny appearance. The eyelids are usually most swollen at first. Beside the face, the ankles, the legs, the scrotum, and the dependent parts of the trunk may be the chief seat of the œdema, the severity and extent of which may of course vary greatly in different cases. If a high degree of general dropsy develops, this is a source of great distress to the patient. The movements of the body are much restrained, and all changes of position are difficult, associated with great exertion, and painful. In the severest degrees of dropsy small fissures may form here and there in the excessively tense skin, from which the dropsical fluid oozes. Such little wounds are sometimes the starting-point for disagreeable erysipelatous inflammations, etc.

If great œdema of the skin is present, we usually find at the same time a more or less marked dropsy of the serous cavities. It is often hard, however, to make out ascites or hydrothorax on physical examination, owing to the œdema of the skin that is present. The symptoms mentioned acquire their chief clinical significance from the difficulty of respiration necessarily associated with them, since the diaphragm is pressed upward by ascites, and the lungs are compressed by hydrothorax. If a hydrothorax is more marked on the left, or especially if hydropericardium sets in, the activity of the heart is materially impaired.

A marked œdema of the mucous membranes develops but rarely; in a few cases we have seen œdema of the conjunctivæ, œdema of the soft palate, and œdema of the glottis. Of the œdemas of internal organs, œdema of the brain has already been mentioned as a possible cause of severe nervous uræmic symptoms. Œdema of the lungs, which often comes on toward the end of the disease, when it terminates unfavorably, is usually not to be regarded as a part of the general œdema, but as a result of the final cardiac weakness.

In regard to the other symptoms in the different organs, the symptoms on the part of the circulatory apparatus must first be mentioned. The pulse is often abnormally tense, hard, and full (see page 835). In the beginning of the disease it is often somewhat slow; later it is usually accelerated. A beginning cardiac hypertrophy can often be made out post mortem, and sometimes clinically, in cases which have lasted a somewhat longer time, two to four weeks. It seems to develop most rapidly in children who were previously well and strong. We pay especial regard to the condition of the apex-beat, and to the accentuation of the aortic second sound. The occasional nose-bleeds are probably connected with the increased arterial tension. Pericarditis is seen as a very rare complication—a complication which is connected with the general fact that in all forms of nephritis the different internal organs, especially the serous membranes, have a tendency to inflammation. Whether this circumstance is connected with the retention of urinary constituents, as has been repeatedly imagined, can not at present be decided with certainty.

Of the symptoms in the respiratory apparatus, we have mentioned above the dyspnœa consequent upon the dropsical symptoms. In severe cases the lungs themselves are often drawn into sympathy, since a diffuse bronchitis or a peculiar form of pneumonia develops in them, which latter stands midway between a catarrhal and a croupous inflammation. It exhibits, to a certain degree, a form of stiff inflammatory œdema, and occurs in just the same way in the chronic forms of nephritis as in acute nephritis. When it involves both lungs to a great extent, it may be the immediate cause of death. The development of a pure general pulmonary œdema is almost always a sign of beginning weakness of the left ventricle, as we have said above.

Vomiting is the most important symptom in the digestive apparatus. If it appears in a marked degree, it may almost always be considered as a uræmic symptom, and then is often the precursor of severe nervous symptoms. The appetite is almost always diminished in acute nephritis; the bowels are usually constipated, but there may be quite severe diarrhœa (see page 832). We may mention peritonitis, which is sometimes purulent, as a very rare complication (*vide supra*).

The temperature is markedly influenced by acute nephritis only in those cases where the disease develops in previously healthy persons, or at least in those free from fever. Then we see quite frequently a moderate fever, with an irregular rise of temperature of about 100° to 102° (38°–39° C.). It is quite rare that an apparently primary acute nephritis begins suddenly with a chill and high fever, 104° (40° C.). The condition of the temperature on the onset of uræmic symptoms has already been described (page 833).

The state of the general nutrition suffers in quite a noticeable degree in most of the severe cases of acute nephritis. The emaciation is often concealed by the œdema; but the anæmia is the more prominent, and often lends to the bloated face a peculiar pallid aspect.

Uræmic symptoms may come on at any time in the course of acute nephritis. We are often prepared for the onset of uræmia by a previous marked decrease in the secretion of urine, or by the well-known prodromal symptoms, but in other cases it begins very suddenly with severe symptoms of eclampsia. In regard to all further details we may refer to what was said on page 829 *et seq.*

THE COURSE AND DIFFERENT FORMS OF ACUTE NEPHRITIS.—The whole clinical picture of acute nephritis depends very materially upon the form of its development. If an acute nephritis comes on in the course of a severe infectious general disease, as in the course of a septic affection, of ulcerative endocarditis, or of severe typhoid, the changes in the urine are often the sole factor pointing to the occurrence of the complication. The type of the severe febrile general disease is in no way materially modified by the added renal affection; œdema and uræmic symptoms do not usually appear, often because the primary disease soon ends in death.

Also when nephritis comes on in previously healthy persons or in chronic invalids, the tuberculous, etc., in many cases the changes in the urine are the chief symptom, while the other general and secondary symptoms are scarcely evident at all, or at least only in a very slight degree. Such mild cases are associated only with more or less general dullness and loss of appetite. Œdema is entirely absent, or present only to a very slight degree. Of course such cases demand great caution, since even in them we may have a sudden outbreak of severe uræmic symptoms.

The fully developed type of severe acute nephritis is seen especially in scarlatinous nephritis (*q. v.*), which comes on in children who are fully convalescent or apparently wholly well ; it is also seen in many cases of apparently idiopathic nephritis, or nephritis coming on after exposure to cold, etc. In these cases there is often the development of a general dropsy, secondary pulmonary affections, uræmic symptoms, the symptoms mentioned in the circulatory apparatus, etc. In these cases, too, the examination of the urine affords the only certain means of judging accurately of the condition, but the other morbid symptoms which appear early—œdema, anæmia, and vomiting—may direct our suspicions to the developing renal affection.

Scarcely any more general statements can be made as to the whole course and the duration of acute nephritis, since the variations in this respect are too great. To describe here in particular all the different forms of nephritis according to the ætiological conditions in question would lead us too far. We will therefore refer to the description of the different primary diseases in which the characteristic marks of any renal complication are always stated. The primary nephritis from exposure to cold and the nephritis of pregnancy still demand a few remarks.

The NEPHRITIS FROM EXPOSURE TO COLD—primary idiopathic nephritis—usually comes on quite speedily after the exciting cause. The first symptoms of the disease are sometimes insignificant, but at other times they are quite severe—chills, fever, renal pain, etc. Sometimes other "rheumatic symptoms," like angina or articular pains, are also present. The further course may be mild or severe. In the former case the œdema that has appeared is but slight, the changes in the urine (albuminuria, hæmaturia, etc.), do not attain a very high degree, and after a few weeks complete recovery ensues. In other cases, however, the type of a severe, acute, and very often hæmorrhagic nephritis develops, with great general dropsy, uræmia, etc., which in three or four weeks, or sooner, may lead to death; but improvement may follow in spite of the severest symptoms. Then the amount of urine gradually increases, and the abnormal constituents of the urine, the œdema, and the other morbid symptoms, gradually disappear. Of course, it is often a long time before complete recovery ensues, since, even when the patient feels perfectly well subjectively, the urine may still sometimes contain some albumen, a few casts, or a few red blood-corpuscles. We must also bear in mind the possibility of a transition from acute to chronic nephritis.

The NEPHRITIS OF PREGNANCY usually begins gradually. Frequent micturition and œdema of the lower extremities make their appearance, and beside these there are often nausea and even vomiting. If we examine the urine, we usually find it quite rich in albumen, but comparatively poor in corporeal elements. The slight sediment consists of hyaline casts, a few white blood-corpuscles, and some epithelium. Only rarely does the urine assume a hæmorrhagic character.

The condition described almost always lasts to the end of pregnancy. In the cases that proceed favorably a very rapid recovery often follows after the birth of the child; but the onset of *eclampsia gravidarum* is to be dreaded as a not infrequent and a dangerous complication. This is to be regarded as entirely analogous to uræmia. It begins after mild prodromal symptoms, or even quite suddenly, with violent general convulsions, during which the child is usually born. A more or less persistent coma follows the convulsions. The convulsions may be very frequently repeated. Death ensues in about one third of the cases; the other cases usually recover, only rarely passing into chronic nephritis. The prognosis is still more unfavorable for the child than for the mother, inasmuch as the child dies in nearly one half of the cases.

The anatomical changes in the nephritis of pregnancy are hardly ever very striking to the eye. The kidneys are usually pale and but little enlarged. Under the microscope we usually find a slight interstitial œdema and degenerative changes in the epithelium. Only rarely are more marked nephritic appearances present.

Diagnosis.—Acute nephritis can be overlooked only when the examination of the urine is neglected or can not be carried out. The latter sometimes happens, for example, when the patient does not come under observation until after the onset of severe uræmic symptoms. Otherwise, however, the changes in the urine always furnish evidence enough to recognize the existence of the affection of the kidneys. We can, of course, decide that the nephritis is acute only by consideration of the history, the ætiological conditions, and the whole course of the disease. We must also bear in mind the possibility that there may be an acute exacerbation in a chronic nephritis that has already existed for a long time, and has been perhaps without symptoms—acute recurrent hæmorrhagic nephritis.

Prognosis.—The prognosis of acute nephritis depends in many cases not only upon the renal affection, but also upon the underlying primary disease. We can not here describe in detail the numerous conditions that must be considered, but they are to be found in the appropriate chapters.

Many cases of primary nephritis from toxic action, or exposure to cold, and also many cases of secondary nephritis after scarlet fever, in pneumonia, typhoid fever, or syphilis, during pregnancy, etc., recover perfectly in a short time or after several weeks, according to the severity of the individual case. On the other hand, however, it must be said that every nephritis must be judged with great caution, partly because it may be the starting-point of a subsequent chronic renal disease, and partly because dangerous sequelæ may sometimes develop in cases that at first are apparently mild. The dangers of acute nephritis are chiefly, first, the appearance of severe general dropsy, especially in the internal cavities of the body. Of the forms of dropsy hydrothorax is the most dangerous, as it may produce suffocation by compression of the lungs. Second, uræmia, especially in its severe convulsive forms, with high temperature and finally cardiac paralysis. Third, the inflammation of internal organs, among which secondary pneumonia, in particular, is a frequent cause of death, while secondary pericarditis and peritonitis, as we have said, are seen in but very few cases. We must bear in mind, however, that in individuals otherwise healthy the severe sequelæ just mentioned may also be recovered from. The most extreme dropsy may be re-absorbed, and we sometimes see recovery, especially in children, after the severest uræmic symptoms.

Treatment.—Since we may omit the description of the treatment of any primary disease, we have here to speak only of those remedies which the physician has at his command against the nephritis itself and its sequelæ.

Although it seems alluring to try to exert a favorable influence upon the nephritic process by drugs which, like the injurious substances, also reach the kidneys directly, we can not report any definite practical results from such treatment. The remedies employed with this object in view—tannin in one- to five-grain powders (grm. 0·05–0·2) several times a day, and the drugs containing tannin, like uva ursi, in a decoction of 10 to 150, and also nitric acid, tartar emetic, etc.—prove, on sober observation, to be almost wholly useless. We may, therefore, try them only when there are no more pressing indications to be fulfilled. Fuchsine, which has of late been often praised, is also not to be recommended.

We expect as little result at present from "external antiphlogosis" as from the internal remedies mentioned—that is, from local blood-letting, applications of ice to the region of the kidneys, etc. Only in the rare cases where severe pain in the region of the kidneys comes on at the beginning of nephritis, in an otherwise robust individual, are we at present justified in trying leeches or a few dry cups. The warm baths, to be described more fully below, have perhaps an immediately favorable action on the process in the kidneys, since they produce a hyperæmia of the skin, and thus lessen the flow of blood to the kidneys.

Although we must accordingly admit that there is scarcely any remedy at our service which has a direct therapeutic influence upon the diseased kidneys, the treatment of nephritis may nevertheless produce very significant results, since both a number of hygienic measures and the fulfillment of certain symptomatic indications are of the greatest importance.

Among the general hygienic measures we must mention first strict confinement to bed. In the severe cases its necessity is self-evident; but, even in the milder cases, which run their course without any severe subjective symptoms, constant rest in bed is necessary throughout. In this way we not only avoid the unfavorable action of cold upon the external skin, but the activity of the skin, which must act vicariously for the kidneys, is also excited by the uniform warmth of the bed, while any useless muscular exertion, which would tax the heart's capacity for work, is also avoided by staying in bed. In general it is advisable to cover the patient quite warmly, so as to keep him in a constant slight perspiration.

The regulation of the diet is very important. All those foods and drinks which may irritate the kidneys are to be strictly avoided, especially spices, very sour substances, strong tea and coffee, or alcoholic drinks. Milk has for a long time proved itself to be by far the most suitable and best food. This has won for itself the reputation of a remedy in renal disease, and the best results have often been seen from a methodical "milk-cure"—that is, from feeding the patient almost exclusively with milk. The great aversion of some patients toward milk, however, is sometimes an obstacle to its use. We may often be aided, then, by making the milk more acceptable to the patient by the addition of a little coffee, salt, a little cognac, or soda-water. Among other foods to be recommended are buttermilk, milk-gruel with rice or groats, and flour-gruel. We should be very cautious about giving meat as long as there are severe symptoms. We may allow meat-broths, with eggs, sooner. For drinks, beside milk, we may give water and lemonade, which latter is especially suitable. Of alcoholic beverages, a little weak red wine is usually the only thing to be permitted. Stronger wines are given only when there is cardiac weakness, and then they are of doubtful benefit.

The chief object in the symptomatic treatment consists in preventing the injurious results of the defective elimination of the water and the solid constituents of the urine by the kidneys, or in removing these results if they have already occurred. This purpose can be attained only by exciting, as far as possible, the activity of other organs which in this respect may act vicariously for the kidneys. The skin deserves the first attention here, through which, by means of the sweat-glands, large amounts of water, and also, to a certain extent, the solid constituents of the urine, which have been retained, may be eliminated. The diaphoretic treatment of renal diseases has, therefore, been generally in vogue for a long time. If the patient's general condition permits, we always begin with it as early as possible, even before there have been any signs of œdema, uræmic symptoms, etc. Hot baths from 95° to 105° (36°–40° C.) are best. The patient stays about half an hour or an hour in the bath, is then rapidly dried somewhat, wrapped up in bed in a previously warmed sheet, and then is well covered up to the neck with blankets. In order to make the procedure somewhat easier for the patient, it is a good plan to cover the forehead with a cold compress, always to wipe the sweat carefully from the face, and frequently to give him a little swallow of fresh cold water. The production of sweat is, of course, better excited during the pack if the patient takes some hot drink, hot milk with soda-water, or hot elder-tea. It sometimes seems to aid diaphoresis if an internal diaphoretic be given at the same time, the best being five to ten grains of Dover's powder (grm. 0·3–0·5), or three or four drachms (grm. 10–15) of liquor ammonii acetatis (*spiritus Mindereri*) in a cup of elder-tea. We have also found a good rubbing of the whole body with dilute warm French brandy of service before the pack. The pack may last two or three hours.

In this way we succeed in many cases in causing a considerable production of sweat, so that the patient loses several pounds in weight at each pack, and an existing dropsy may sometimes be made to disappear completely in a comparatively short time. On the other hand, however, we can not deny that it is very hard sometimes to make patients sweat, even when there is œdema of the skin, and also that many patients do not bear hot baths and packs at all. The latter is especially true if the patient has dyspnœa, and if signs of cardiac weakness have already set in. Then we have to be very cautious about using sweating as a remedy. Sometimes we can bathe the patient, but we have to omit the pack, while in other cases he can take the hot pack in bed ; but we must avoid carrying him to the bath and back. We also have to get along with hot wet packs, if baths can not be used for extrinsic reasons.

Beside hot baths and packs, one diaphoretic remedy is to be especially considered in renal disease, and that is the hydrochlorate of pilocarpine, derived from the jaborandi-leaves. We use it best in the form of a subcutaneous injection, of one sixth to a third of a grain in one dose (grm. 0·01–0·02) ; but it may also be given internally in the form of pills, in like doses. Its action consists in the production of a copious sweat, and also usually of a very considerable flow of saliva, which is often very disagreeable to the patient. In general, we prefer the baths to pilocarpine, and we try the latter only when the baths are contra-indicated or do not exert any satisfactory action. The diaphoretic action of pilocarpine, moreover, is often decidedly less in dropsical patients with kidney disease than in other cases.

Next to the skin, the intestinal mucous membrane is the organ from which we may soonest expect to produce a vicarious elimination of water, and also of urea, for the kidneys. It is sometimes, therefore, of distinct service to prescribe drastic cathartics in nephritis with a diminished secretion of urine, especially if there is a tendency to constipation beside the dropsy, dyspnœa, etc. The drastic cathartics chiefly used are infusion of senna, decoction of colocynth, 3 or 6 to 150, gamboge in two-grain powders (grm. 0·1), etc.

Finally, it may be asked whether we should not excite the secretory function of the kidneys themselves by the exhibition of diuretics. The objection is, however, that all diuretic remedies irritate the kidneys, increase the flow of blood to them, and therefore can act only injuriously upon the nephritis. We must, therefore, be very cautious in using diuretics. Only the milder remedies, especially acetate of sodium, may sometimes be used with advantage, especially in the less acute cases or during the period of convalescence. The diuretic action of digitalis, which is very important under some circumstances, will be mentioned later.

The therapeutic measures so far spoken of correspond to the task of preventing as far as possible the retention of urinary constituents in the body. They are also very much employed when the signs of this retention have already appeared. The dropsy especially can be successfully treated only by methodical sweatings, with the aid, eventually, of drastic and diuretic remedies. When uræmia is threatening, and often even when it has broken out, we may try to produce an elimination of the injurious products of tissue metamorphosis from the body in the well-known ways above described, by sweating or drastic purgatives. Beside this, the uræmic symptoms, however, often demand a special symptomatic treatment. If very violent and frequent uræmic convulsions appear, we consider it advisable to try to suppress the attacks by chloroforming the patient. At any rate, it seems to us to be better to use chloroform in uræmia than to give narcotics internally, because with this we can watch the action of the remedy better, especially the condition of the pulse and respiration. Chloroform is also generally used by the obstetricians as the main remedy in the eclampsia of pregnancy. If the attacks are not very frequent, but if there is marked somnolence or coma, tepid baths with cold shower-baths are often employed with distinct advantage. Cool baths are also serviceable where there is a great increase of the temperature. If we are treating a robust individual with a full pulse, and during severe uræmia there is a decided redness or cyanosis of the face, venesection may be indicated. This sometimes has a striking and instant effect, as has lately been confirmed by various observers. Great attention is to be paid to the condition of the heart. As soon as the pulse becomes small and weak, energetic stimulants, like subcutaneous injections of camphor, must be used. If the signs of cardiac weakness appear before the beginning of severe uræmic symptoms, digitalis must be used in infusion or powder. Through its action in raising the blood-pressure—it being advisable under some circumstances to combine with it acetate of potassium—a greater diuresis sometimes comes on, and with it a disappearance of the danger from uræmia. The tinctura nervina Bestuscheffii [nearly equivalent to the tincture of the chloride of iron, U. S. P.] may also be sometimes used to advantage in uræmia. We are not apt to interfere with uræmic vomiting or uræmic diarrhœa, because these symptoms, as we have said, are to be regarded as a form of self-help by the organism. Only when these symptoms are very distressing do we give cracked ice, morphine, opium, etc. If the vomitus contains ammonia, it is a good plan to give ten or fifteen drops of dilute hydrochloric acid in water several times a day.

[A method of treatment of uræmic convulsions, whether post partum or connected in no way with parturition, which gives excellent and prompt results, is the administration of pilocarpine hypodermically—grain ¼, and repeat in twenty minutes—followed by the hot-air bath, to maintain the action of the skin after it has once been started.

Pilocarpine sometimes acts as a decided cardiac depressant, so one must be ready to administer stimulants—brandy or ether—under the skin if, as is apt to be the case, there is doubt as to the readiness with which they will be absorbed from the stomach or rectum.]

In severe cases, the patient's dyspnœa often demands urgent relief. If this be caused, or at least increased, by hydrothorax, and we do not succeed in removing the hydrothorax in any other way, it is necessary to evacuate it by puncture. In acute nephritis, indeed, we may hope by this means sometimes to preserve the patient's life until improvement sets in. Great ascites must also sometimes be punctured. Against "renal pneumonia" our remedies are powerless. Tepid baths and shower-baths sometimes procure relief. In "uræmic asthma," morphine injections may act beneficially. If pulmonary œdema ensues, the heart again is chiefly to be considered. We may try, besides, large mustard plasters, baths, and acetate of lead.

We accordingly see that many remedies are at our service in the treatment of nephritis, the choice of which in the individual case must be committed to the personal judgment of the physician. In the main, we should always begin with the necessary hygienic measures, and, if possible, with a methodical diaphoretic treatment, and govern ourselves otherwise by any symptomatic indications. After recovery has set in, great caution is still necessary for a long time. The patient must guard against physical over-exertion, errors in diet, and exposure to cold. Preparations of iron are to be prescribed when there is a subsequent anæmia.

In regard to the influence of the onset of an acute nephritis on the treatment of the primary disease, we may yet mention that cold baths are in general not to be freely used, as in typhoid fever with nephritis, but they are not absolutely contra-indicated if they are otherwise urgently desirable. We may mention besides that certain internal remedies, especially salicylic acid, must be used only with great caution when there is nephritis. In the eclampsia of lying-in women the induction of premature labor is only rarely indicated, since the child is usually born during the paroxysms without interference.

CHAPTER III.

THE SUBCHRONIC AND CHRONIC FORMS OF NEPHRITIS, WITH THE EXCEPTION OF THE GENUINE CONTRACTED KIDNEY.

("*Second Stage of Bright's Disease.*" *Chronic Parenchymatous Nephritis, Chronic Hæmorrhagic Nephritis, Large White Kidney, Secondary Contracted Kidney.*)

Ætiology.—While the acute nephritis described in the preceding chapter runs its course in several days or weeks, and only rarely extends over some months, we will now speak of inflammatory degenerative affections of the kidneys which last at least several months, and often go on for a year or two. The term "subacute" or "subchronic" is chosen for the cases that last a comparatively short time. As we must once more repeat, there is no sharp limit in this respect.

In regard to the ætiology of these forms of nephritis, they do arise from an acute nephritis, but this is quite rare. Formerly such an origin was erroneously regarded as the rule, and this is the reason why the changes in the kidney in these cases were described as the "second stage of Bright's disease" (Frerichs). The English clinical observers Wilks and Johnson, whom Bartels followed in Germany, first pointed out the fact that in most cases the disease shows a chronic character from the start, and that we can only exceptionally, as after scarlet fever, recognize an acute "first stage." The name "chronic parenchymatous nephritis," since frequently used, is chosen entirely from practical reasons, inasmuch as it briefly states the distinction from the genuine contracted kidney; but it is incorrect in principle, as will be shown from the description of the anatomical conditions later.

If we look for the ætiological conditions in cases that have a chronic course from the beginning, we can often discover nothing definite at all. The disease seems to have developed "of itself" in previously healthy persons. Most probably we have here some toxic or infectious agency that acts on the kidneys, whose detection, however, is at present impossible. In malarial regions the malarial poison may often lead to chronic nephritis. It is also attributed to syphilis and tuberculosis, but the cases met with are usually combinations of these diseases with amyloid kidney (*vide infra*). Frequent exposures to wet and cold, damp dwellings, etc., seem sometimes to be of more material significance, but it is, of course, hard to form a definite opinion on this point.

Persons in middle life are most frequently affected by the disease, and men more often than women. In children and old people the disease is quite rare.

Pathological Anatomy.—There is no essential distinction between the anatomical lesions of the kidney in acute and in chronic nephritis. The changes that are seen in both are essentially the same, only they develop and extend more slowly in the chronic forms; and they also, during their longer duration, lead to certain sequelæ in the kidney, which can not develop at all in acute nephritis, owing in part to the lack of time. Even in chronic nephritis the individual cases differ from one another in many respects. First this and then that histological process is especially prominent, and thus lends certain peculiarities to the macroscopic appearance of the kidneys. Certain sequelæ—like contractions—have also developed but little in many cases that soon end fatally, but they develop far more in other cases of longer duration. Hence it happens that we can quite well regard certain anatomical forms that are more frequently observed as types, although we must never lose from sight the principle, to be firmly held, of the pathological unity of all these forms and types. Then we shall not lose the clew to the understanding of the morbid process if the individual case does not always harmonize with the scheme of the text-books.

We distinguish the three following chief anatomical types of subchronic and chronic nephritis:

1. CHRONIC HÆMORRHAGIC NEPHRITIS in the form of the LARGE RED OR VARIEGATED KIDNEY.—The kidney is at least of normal size, and often a little or a good deal enlarged. It feels firmer than normal; its capsule is often adherent to the surface in some places. The surface looks either uniformly a more gray red or more mottled, while dark-red spots alternate with lighter gray or even yellow spots. The red spots on the surface can not be wholly wiped off, and thus prove to be hæmorrhages. The gray or yellow parts correspond to the anæmic and fatty degenerated portions. On section, the cortical substance is usually broader, its

normal boundary is obliterated, and its color is a uniform gray red, or also mottled and striated.

Under the microscope we find in part the same changes as in acute nephritis—parenchymatous and fatty degeneration of the epithelium, casts or hæmorrhages in the uriniferous tubules, inflammatory œdema or granular infiltration of the interstitial tissue, the capsules of the glomeruli sometimes thickened, the epithelium of the glomeruli sometimes proliferated or desquamated, etc. The special characteristic of this chronic form, in contrast with acute nephritis, is that in many places a complete destruction of the uriniferous tubules has occurred, and that a genuine interstitial connective tissue, richer or already poorer in cells, has taken their place. In this lies the anatomical evidence of the longer duration of the disease, since the two processes—both the complete atrophy of the epithelium, and especially the secondary proliferation of connective tissue—of course need a certain time for their development. The atrophy and the proliferation of connective tissue usually predominate in some parts, while in others nothing but fresher inflammatory and degenerative changes are perceived.

2. THE INFLAMMATORY FATTY KIDNEY, or the LARGE WHITE KIDNEY (yellow would be more proper).—In this form of chronic nephritis the kidney is usually enlarged, or at least of normal size. Its outer surface is smooth and of a yellow or an alternating yellow and gray-yellow color throughout. The broader cortical substance shows a yellow and usually somewhat mottled appearance, while the pyramids almost always appear considerably reddened. Hæmorrhages are also almost always present in this form, usually, of course, in smaller numbers than in the variegated kidney, but they are sometimes quite abundant, as in the hæmorrhagic fatty kidney.

The microscope shows the great affinity between this form of nephritis and the preceding. We have almost precisely the same changes, and we always have especially a partial destruction of renal tissue with a subsequent increase of interstitial connective tissue. The macroscopic appearance of the kidney is due to the fact that it is anæmic, and that the fatty degeneration preponderates in the epithelium. It is worthy of note that in these kidneys marked changes in the glomeruli are usually present.

3. THE SECONDARY CONTRACTED KIDNEY.—While in the two forms of nephritis thus far described the outer surface of the kidney is still smooth, and the kidney, on the whole, is somewhat enlarged, we have to do here with kidneys of about normal size, on whose surface there are granulations, which as yet are slight, but which still are already plain. This granulation signifies nothing more than that the destruction of the renal tissue has here advanced farther, and that the newly formed connective tissue has in part undergone cicatricial contraction. These kidneys, therefore, represent a later stage of the two forms first-named. They usually come under observation when the nephritis has lasted about a year and a half or two years, or even somewhat longer. The first beginnings of granulation may, of course, show themselves earlier, while, on the other hand, when the process lasts a longer time, a completely contracted kidney may develop.

The color of these kidneys is usually reddish or mottled, the red spots corresponding to the sunken atrophic parts, and the gray or yellowish spots to the elevated parts. Yellow kidneys, however, may also show at times decided granulations. Microscopically, we find already marked atrophy of the renal parenchyma, with a corresponding increase of the interstitial connective tissue.

Formerly these kidneys were called the "transition between the second and third stages of Bright's disease." As follows from the above, they are to be regarded only as a more advanced form of chronic nephritis. Since the kidneys, in spite of their granulation, have on the whole a normal size, we can decide from this, and from the clinical course, that they were usually, though of course not always, previously enlarged. Therefore the name of "secondary contracted kidney" is quite suitable, in opposition to the genuine contracted kidney, which represents a much more chronic form of renal atrophy.

Of other pathological lesions, apart from the changes in the kidney, we will mention here only the hypertrophy of the left ventricle, which is found with few exceptions (*vide infra*) in all the above-mentioned forms of nephritis. The chronic parenchymatous nephritis without cardiac hypertrophy, alleged by Bartels in his time, does not exist. Such cases were probably confused with amyloid kidneys.

Clinical History.—Only in the comparatively rare cases when the renal affection begins acutely, do the symptoms of chronic nephritis follow immediately on the first acute stage. In most cases, however, the disease develops slowly and gradually from the start, as we have said, like most of the other chronic organic diseases, so that it is usually impossible to determine accurately the moment when the disease begins.

The first signs of the disease consist of certain general symptoms, pallor, dullness, loss of appetite, nausea and headache, and later of œdema. The latter is often the first symptom which sends the patient to the physician, since in the beginning he often pays little attention to the symptoms first named. The œdema usually appears first in the ankles and legs, more rarely at an early period in the face. It often disappears at first after a night's rest, but always develops afresh during the day, gradually increasing in intensity. The patient himself now sometimes notices a change in the urine, either an abnormal color or cloudiness or a diminished amount. The accurate examination of the urine by the physician first establishes the diagnosis with certainty.

In regard to the more special symptomatology of chronic nephritis, we meet exactly the same symptoms as have been described in the preceding chapter on acute nephritis. The characteristic distinction is based merely upon the whole course of the affection and the order of development of the different symptoms, and not by the symptoms themselves.

The urine almost always is diminished. Of course the figures vary quite considerably both in different cases and at different times in the same case. The small amount of urine, ten to twenty-five ounces (300–700 c.c.) a day, is almost always an unfavorable sign, while a free diuresis signifies an improvement of the condition, an absorption of the dropsy, and finally a passage of the renal affection into a still more chronic condition, the secondary contracted kidney (*vide infra*). Under such circumstances the amount of urine may even be increased above the normal, to fifty or sixty ounces (1500 or 2000 c. c.) or more.

The specific gravity of the urine is often increased to about 1015–1025, corresponding to the amount of albumen and of other solid constituents. It is of course correspondingly lower when there is a more abundant elimination of water by the kidneys.

The amount of albumen in the urine is quite marked in all severe cases, being one third to three fourths of its volume. It amounts to about 1·5–3 per cent. by weight, so that the patient's daily loss of albumen may reach half an ounce to an ounce (15–30 grammes).

The examination of the sediment, which is usually abundant, is of the greatest importance for the accurate determination of the form of the anatomical changes in the kidneys. Above all, the question arises as to the presence or absence of blood in the urine. Every abundant hæmaturia may be recognized by the naked eye from the color of the urine. The detection of smaller amounts of blood can be made only by the aid of the microscope. It goes without saying that the amount of blood in the urine varies quite considerably in the different cases, and in the same case the urine often contains much more blood during certain periods in the course of the disease than at other times. The portions of urine passed at different times taken separately often show quite marked variation in this respect; the day's urine especially usually contains more blood than the night's. From the detection of renal hæmorrhages, of course in connection with other symptoms, we can always make with certainty the diagnosis of a "chronic hæmorrhagic" nephritis.

In most cases casts are quite abundant in the sediment of the urine, but of course their amount and variety undergo quite great variations in different cases and at different times in the same case. They are the direct sign of the presence of an inflammatory exudative process in the kidneys, although the deposits on the casts are more important for the diagnosis of the special form of renal disease than are the casts themselves. Those formed constituents of the sediment are most characteristic in this respect which point directly to the processes of fatty degeneration in the kidneys: the fatty granules and fatty granular cells, free or attached to the casts. The number of these elements is especially great in the chronic inflammatory fatty kidney, the "large white kidney." The usually clear, non-hæmorrhagic urine may in some cases have even a fatty lustrous surface. Renal epithelium is, on the whole, more rarely present in the sediment in chronic nephritis than in acute, but it occurs at times in some cases.

Of the other symptoms, the one that usually most strikes the eye is dropsy. It usually comes on, as we have said, in the beginning of the disease, and slowly or rapidly reaches a great extent and intensity. A medium or even a high degree of general dropsy may often persist almost unchanged for months. In other cases it shows either spontaneous variations or variations influenced by treatment; it decreases for a time only to increase anew. The severer and more acute the case, the greater in general is the dropsy. In the more chronic cases, in secondary contracted kidney, its intensity may be slight for a time or even permanently. The dropsy may even be absent in some cases, as we learn especially from the observations reported by Wagner under the name of "chronic hæmorrhagic Bright's disease without œdema." In regard to the different localizations of the dropsy, and to dropsy of the internal cavities, hydrothorax, ascites, and hydropericardium, and their results, the same holds true as was described in the account of acute nephritis.

Of the internal organs, the condition of the heart lays claim to the most interest. In all cases of chronic nephritis, in which we do not have to do with especially weak and run-down patients, who can not save the necessary nutritive material for the formation of a cardiac hypertrophy, we find a pronounced and often a very marked hypertrophy of the left ventricle, either with or without a co-existing dilatation of its cavity. A chronic nephritis without cardiac hypertrophy, which was put forward by Bartels and others as the type of "chronic parenchymatous nephritis," does not exist, as we have said, except under the above-mentioned conditions. The detection of cardiac hypertrophy during the patient's life is sometimes difficult, especially when there is general dropsy, but the diagnosis can usually be correctly made with proper attention to the abnormally tense radial pulse, the accentuated, valvular aortic second sound, and the displacement outward of the apex-beat, or at least its increased strength. We often find in the cadaver, and can sometimes make out during life, a hypertrophy of the right ventricle. This is usually a sign of a disturbance of compensation—that is, the paralyzed left ventricle can no longer send forward in a sufficient manner all the blood coming from the pulmonary veins, so that there ensues a stasis in the pulmonary circulation, and a consequent hypertrophy of the right ventricle.

A second important sequel of chronic nephritis consists of the changes in the retina—albuminuric retinitis. Although very rare in acute nephritis, these changes are present in by far the greater majority of the cases of this class. Sometimes the patient's subjective visual disturbance (dimness of vision, defects in the field of vision), point to a disease of the retina, but the existence of disease can be established with certainty only by ophthalmoscopic examination. In these cases we find two changes, in varying numbers and combinations: first, retinal hæmorrhages; and, second, white spots and streaks, especially in the vicinity of the optic nerves. The origin of the spots, which may appear and disappear again, is not yet entirely clear. At any rate, there are circumscribed fatty degenerations of the special retinal elements. The degree of amblyopia depends, of course, chiefly upon the localization of the changes, whether in the macula lutea, or other parts.

We can say little in regard to the other symptoms, since they agree essentially with those of acute nephritis. The general anæmia is very pronounced in many cases, but it is less marked in the very chronic forms. The cerebral symptoms, especially the headache and the mild vertigo, may depend in part upon the cerebral anæmia; otherwise, they are a uræmic symptom (*vide infra*). Cerebral hæmorrhages have been observed in a very few cases. Hæmorrhages on the inner surface of the dura mater are more frequent, but they are usually without clinical significance. The mouth, larynx, and pharynx usually show nothing particular, except accidentally complicating inflammations. We must, however, remember the occasional occurrence of a very distressing or even dangerous œdema of the soft palate, or of the arytæno-epiglottic ligaments—œdema of the glottis. Similar forms of bronchitis and pneumonia, as in acute nephritis, are found in the bronchi and lungs. Bronchitis and chronic œdema of the lungs also make their appearance in the more advanced stages of the disease as a result of cardiac insufficiency. Finally, we must remember the hindrance to respiration from hydrothorax, and also from uræmic dyspnœa. The changes in the heart have already been spoken of. Endocarditis or pericarditis may occur, but they are very rare.

Loss of appetite is a very common symptom on the part of the stomach. Very persistent vomiting is usually to be regarded as a chronic uræmic symptom. The bowels, as a rule, are constipated, but there may also be severe diarrhœa, as in acute nephritis. In severe cases, especially in the last stages of the disease, ulcerative and dysenteric processes have repeatedly been observed in the large intestine and the ileum. Peritonitis may occur, but it is at all events extremely rare. The liver and spleen usually show no peculiarities.

Uræmic symptoms, both of the milder chronic variety and also in their severest acute form, may come on at any time, although they do not by any means attain their full development in all cases, and are somewhat rarer than in genuine contracted kidney.

The temperature remains normal, as a rule, as long as it is not influenced by complicating inflammations, or by the appearance of uræmia.

Course, Duration, and Termination of Chronic Nephritis.—In general, the whole course of chronic nephritis presents quite a great uniformity. The different symptoms may show certain variations within long periods, but the patient often presents almost the same appearance day after day for months. The duration of the disease shows all the transitions from three to six months, in the subacute cases, to two or three years, or even more, in the very chronic cases. The cases of long duration are almost all cases of secondary contracted kidney. They sometimes show in their clinical relations the transition from the enlarged to the granular kidney, since the picture in many of its details is more like that in the genuine contracted kidney: the œdema decreases, disappears completely, or, at least, continues in a lesser degree; the amount of urine becomes more abundant, and the specific gravity and the amount of albumen become correspondingly less. The condition thus lasts for a long time until it grows worse again, through uræmia, or disturbance of the compensation in the heart.

The final termination of chronic nephritis is in most cases unfavorable. In the severe forms death ensues in from three months to a year, either in consequence of general dropsy or from uræmia, from complicating inflammations, etc. The conditions when the nephritis goes on to secondary contraction are comparatively more favorable, inasmuch as the patient may then find himself in a tolerable condition for a time at least. Complete recoveries doubtless occur in chronic nephritis, but they are rare. An apparent recovery may be simulated by the appearance of secondary contraction. Even after signal improvement, however, relapses are always to be feared. There are even genuine acute attacks in the course of chronic nephritis.

Diagnosis.—On careful examination of the urine in all suspicious cases of œdema, anæmia, etc., the diagnosis of chronic nephritis can always be correctly made. In regard to the more exact distinction of the different anatomical forms, we will here give a brief schematic glance at the most important conditions:

Chronic Hæmorrhagic Nephritis (large variegated or mottled kidney).—Duration from six to eighteen months. Urine often hæmorrhagic; usually quite rich in red blood-corpuscles and casts. Œdema. Cardiac hypertrophy. Retinal changes. Quite frequently uræmia.

Inflammatory Fatty Kidney (large white kidney).—Duration also six to eighteen months, but usually somewhat shorter than in the preceding form. Urine not at all, or only slightly, hæmorrhagic. Frequently many white blood-corpuscles, and especially signs of fatty degeneration in the kidneys, fatty granular cells, fat-drops in the urine, etc. Significant amount of albumen in the urine. Marked œdema. Cardiac hypertrophy. Very often retinal changes. Death by uræmia frequent.

Secondary Contracted Kidney.—Longer duration of the disease, from a year and a half to three years. At first the symptoms of the preceding forms; later, urine more abundant, less œdema, etc. Death from an increase of the dropsical symptoms due to cardiac insufficiency, uræmia, etc.

Treatment.—The treatment of chronic nephritis corresponds in all its details so closely to that of acute nephritis that we can refer almost entirely to the preceding chapter.

The main thing here is also regimen and symptomatic treatment. The patient must always keep himself warm, wear flannels, or stay in bed. Under some circumstances climatic cures, like Italy, Egypt, etc., are indicated in the more chronic forms. A milk diet is to be carried out as far as possible.

The treatment of dropsy follows entirely the methods previously described, and so does the treatment of any uræmic symptoms.

In the more chronic cases with great anæmia preparations of iron, such as iodide of iron, are often to be used, and also frequently stomachics, cathartics, etc. The condition of the heart always deserves careful attention (digitalis !). The retinitis rarely demands a special treatment.

CHAPTER IV.

CONTRACTED KIDNEY.

(*Genuine Contracted Kidney. Granular Atrophy of the Kidney. Granular Kidney. Renal Sclerosis. "Third Stage of Bright's Disease." Chronic Interstitial Nephritis.*)

Definition and Ætiology.—The genuine contracted kidney is the result of an extremely chronic and very slowly but constantly progressive atrophy of the renal tissue. The term "chronic nephritis" is also used for contracted kidney, but the special inflammatory processes are very subordinate here, for the anatomical process consists essentially in nothing but a simple degenerative atrophy of the renal parenchyma, and in a corresponding gradual increase of the interstitial connective tissue. From a general pathological point of view the process is to be regarded as wholly analogous to the corresponding changes in the liver in cirrhosis of that organ, in the spinal cord in the chronic degenerations of the different systems of fibers, etc. In all these cases we have a primary destruction of the special tissue-elements as a result of some deleterious action, and, following a general pathological law (Weigert), a partial replacement of the parts destroyed by a newly formed cicatricial connective tissue.

In the "genuine" contracted kidney the atrophy of the renal parenchyma begins in a previously healthy kidney. Cell after cell of epithelium, islet after islet of tissue, are slowly attacked, while other parts still remain intact. It was therefore an error of the older pathologists to regard the contracted kidney as the "third stage of Bright's disease," as if every granular kidney were first found in the stage of acute inflammation, and then passed into the stage of chronic enlargement, and lastly into that of contraction. This theory, of course, suits certain cases in part, for chronic nephritis at least may often finally pass into contraction, but these "secondary contracted kidneys" (*vide supra*) can clinically, and almost always anatomically, be differentiated from the genuine contracted kidneys. On careful examination, as it seems to us, the contracted kidney may of course arise from an acute nephritis in some cases, which perhaps are not very rare; but then the process hardly ever passes through the three stages mentioned above, for the acute nephritis apparently recovers. A slight remnant of it is left—a little fire, as it were, glimmering under the ashes; its work of destruction advances, wholly in secret, and perhaps only after many years do the symptoms of a pronounced renal contraction appear.

If we inquire into the causes which produce the atrophy of the renal tissue in the ordinary cases of contracted kidney, which are chronic from the first, we are very often unable to make out any special causes. Of course, we must again bear in mind first the two great groups of injurious influences, the chemico-toxic and the organized parasitic, but at present only a small number of ætiological factors have a more or less definite significance.

Experience teaches us that there are three chemical substances to be mentioned which may favor the development of contracted kidney: alcohol, lead, and uric acid. Chronic alcoholism is often to be regarded as the most probable cause of renal contraction, especially in people who have "lived well" otherwise, and have become corpulent. This is the explanation of the combination of contracted kidney and cirrhosis of the liver repeatedly observed. The connection between contracted kidney and chronic lead-poisoning, in type-setters, painters, etc., is also incontestable. It is also a remarkable circumstance, and one not yet fully explained, that in these cases we very often see at the same time a genuine gout (*arthritis uratica*). Gout, however, alone, without any co-existing chronic lead-poisoning, often leads to the development of contracted kidney, "gouty kidney," in which we probably have to do with the noxious action of an abnormal amount of uric acid on the renal parenchyma.

Infectious influences are, probably, first to be considered in those cases where the contracted kidney can be referred to a former infectious nephritis, as after scarlet fever. We may also mention here the appearance of contracted kidney sometimes observed after severe acute articular rheumatism. We may perhaps imagine a similar connection in the cases where contracted kidney is found combined with chronic endocarditis (valvular heart disease), or with chronic arthritis not of gouty origin. Of the chronic infectious diseases, which may probably sometimes have a connection with the origin of contracted kidney, we may mention malaria and syphilis. The latter ought especially to be considered more than it is at present, because we may have either an immediate action of the syphilitic poison, or a renal atrophy as the result of specific disease of the renal arteries.

We must here spend a little time in the general consideration of the connection between renal contraction and primary disease of the vessels, which has been much discussed. It is true that we often find general arterio-sclerosis, and also atheroma, especially in the renal arteries, in the bodies of persons who have died from contracted kidney, but this frequent coincidence can not be remarkable in such cases, because contracted kidney is seen chiefly in advanced age, and in those persons in whom atheroma of the arteries is also a very common symptom. The theory advanced by the English authors, Gull and Sutton and others, that the vascular disease, "arterio-capillary fibrosis," always represents the primary process, to which the renal atrophy is only secondary, is, however, utterly untenable. We often find the most pronounced contraction of the kidneys without any vascular changes sufficient to explain the atrophy; and where the latter can be found in the small renal arteries, we usually have not a primary but a secondary process—namely, the well-known obliterating arteritis, which is seen in almost all chronic inflammations and degenerative atrophies of various organs.

Of course, it can not be denied that under some circumstances primary vascular diseases of the renal arteries may lead to secondary atrophy in circumscribed spots by checking the blood-supply to certain parts of the tissue—"vascular contracted kidney"—just as indurated myocarditis arises after primary arterio-sclerosis of the coronary arteries. The "senile kidney"—that is, the granular kidney of old people—is also due to vascular changes like atheroma, as are also, which is worthy of special note, the rare cases of unilateral contraction of the kidney, which is observed chiefly in syphilis.

The relation of contracted kidney to amyloid disease of the kidney, and to chronic diseases of the urinary passages, especially of the pelvis of the kidney, will be spoken of later in the appropriate chapters.

Pathological Anatomy.—In the genuine contraction of the kidney, both kidneys are always diminished in about the same degree. Their size is sometimes reduced to one half or even one third of the normal, so that it is almost difficult to find the little kidney in the very abundant and thick fatty capsule that is often present. The kidneys feel firm and dense, and show on their surface a very plain, coarse or fine, uniform or irregular, granulation. On pulling off the somewhat thickened fibrous capsule, these granulations become more prominent, and the capsule usually adheres quite firmly to the depressed portions. The raised portions are almost always darker and redder—that is, richer in blood—than the lighter and grayer depressions. Whether the whole kidney appears more red or more white depends only upon the amount of blood in the organ, and there is no reason for separating the "small red" from the "small white" contracted kidney.

On section of the contracted kidney, we find the cortex much smaller, and pale atrophic streaks alternating with the darker portions. The pyramids are also somewhat smaller, and, as a rule, are darker than the cortex. In the pelvis of the kidney, which is often somewhat dilated, there are frequently a number of uric-acid concretions. Striated uric-acid infarctions in the pyramids are a very characteristic mark of the gouty contracted kidney. The microscope shows an advanced destruction of the renal parenchyma, which is replaced by a cicatricial connective tissue which is still granular or which has begun to be poor in granules. We can always make out signs of degeneration and atrophy of the epithelium, and the formation of casts in the uriniferous tubules which still remain, but which are always diseased. Atrophy, thickening of the capsule, etc., are found in many of the glomeruli. The uriniferous tubules that are still preserved in some places are often in part dilated. We can not here go more fully into the manifold histological details, especially the formation of cysts, the changes in the vessels (*vide supra*), the deposition of lime-salts, etc. Hæmorrhages are only very rarely present.

Thus the contracted kidney may be regarded as the form of chronic nephritis with by far the longest course (lasting from three to five years, and even much longer), and also the form with the widest extent. Its essential nature can in no way be contrasted with "chronic parenchymatous nephritis" as a "chronic interstitial nephritis"; for we always find interstitial processes in the former, which have reached a far higher degree in the contracted kidney only because the slow atrophy of tissue is compatible with a much longer duration of life, and hence can attain a much greater extent.

The anatomical changes in the other organs of the body beside the kidneys will be spoken of in connection with the symptomatology of contracted kidney.

Clinical Symptoms.—Except in the comparatively rare cases where we can refer the origin of a contracted kidney to a previous acute or chronic nephritis, the clinical symptoms of contracted kidney develop as gradually and unnoticeably as the anatomical process itself. There is no doubt but that a contraction of the kidney may exist for years, without calling the patient's attention to his disease by a single serious subjective symptom. This follows in part from the chance discoveries on autopsy of a contraction of the kidney in people who have lost their lives in some other way, but especially from the cases where the severest symptoms, like uræmia, cerebral hæmorrhage, etc., which often lead immediately to death, suddenly come on in persons previously regarded as perfectly healthy, while the autopsy often shows quite a far advanced contraction of the kidney as the special cause of these symptoms. The less prominent the subjective symptoms of renal contraction are in the earlier stages of the disease, the more we should consider the objective changes, which in fact usually permit the diagnosis of the disease quite early on careful examination of the patient.

The condition of the urine is most important in this respect. As soon as changes have taken place in the epithelium in different parts of the kidneys, the results previously spoken of must make themselves manifest in the secretion of the urine, although still in a slight degree, and the diseased patient will secrete a urine diminished in amount and in solid constituents, but containing albumen. Since, however, many normal uriniferous tubules and glomeruli are still present, and since the whole process, as we have seen, develops only very slowly, the body gains time for the formation of one of those judicious compensatory contrivances which we recognize in so many pathological processes, and which we must regard in a teleological sense. This compensatory process consists of an increase in the arterial pressure, coming on as gradually as the renal contraction itself, and constantly increasing, and of a hypertrophy of the left ventricle dependent upon it. The blood therefore courses through the many normal glomeruli of the contracting kidney under an increased pressure, and the consequence is that in these portions the secretion of the urine, especially of the water, is much more abundant. This is the reason why, as a rule, in contraction of the kidney, the patient passes an abnormally large amount of urine, which is more watery, and therefore lighter and of a lower specific gravity, and which contains only a slight amount of albumen coming from the diseased portions. The daily amount of urine is often seventy to a hundred and twenty ounces (2000–3500 c. c.) or more ; the urine looks light-yellow and clear, contains scarcely any morphological constituents, has a specific gravity of 1010–1005 or even lower, and gives, on heating, only a slight precipitate of albumen, the amount excreted in the twenty-four hours being about half a drachm to a drachm (two to five grammes). On careful microscopic examination of the urine, we usually succeed in finding a few hyaline casts, which only exceptionally may be more abundant. The urine also frequently contains some white, and more rarely a few red blood-corpuscles. In rare but definitely attested cases it may happen that for a time, or even during the main part of the disease, the urine contains no albumen at all, or only a trace of it. This is probably explained by the fact that the diseased glomeruli have wholly ceased secreting, and that therefore the urine is secreted only by the healthy portions of the kidney.

It is apparent of how great significance this abundant secretion of water, as a result of the abnormally high blood-pressure, must be for the whole morbid process ; for, in spite of the renal disease, there is now absolutely no retention of water in the body, and we therefore understand why there is no œdema in contracted kidney, even after a course of years. The secretion of the solid constituents of the urine is not quite in such a favorable condition as the secretion of water. It is self-evident that the percentage of the former decreases with the increased amount of urine, but the whole amount of urea, uric acid, phosphoric acid, etc., eliminated is also at times somewhat less than normal in relation to the food. This diminution, however, is not very great, as long as the work of the heart is sufficient, and at certain times, especially in the earlier periods of the disease, it may certainly be entirely absent. We accordingly see that the symptoms dependent upon an accumulation of the urinary constituents in the blood do not appear at all for a long time. Thus it happens that the patient may still feel perfectly well up to a time when the objective examination of the urine discovers marked pathological changes. Many patients, of course, notice the polyuria, but often no further attention is paid to it, and it is attributed to drinking a good deal of fluid. The patient gets accustomed to it, even if, as often happens, he has to pass his urine much more frequently than formerly, and even during the night.

We need not go into detail here in regard to the special causes of cardiac hypertrophy (compare page 834). It was with regard to contracted kidney that Traube advanced his mechanical theory of cardiac hypertrophy, which, however, brought up the considerations previously mentioned, and therefore was properly replaced by the chemical theory, which was also very applicable to this form of renal disease. In its clinical relations it is especially important that the cardiac hypertrophy causes no subjective symptoms at all, as long as the heart can suffice for the work put upon it without strain, a condition which is perfectly analogous to that of any fully compensated valvular disease. We can usually recognize the condition correctly only by a careful physical examination of the heart and the vascular apparatus, although in contracted kidney the percussion and palpation of the heart are often rendered difficult by a co-existing pulmonary emphysema. We can often perceive, however, the displacement and the increased strength of the apex-beat, the extension of the cardiac dullness to the left, and almost invariably the abnormal tension of the radial pulse, and the accentuation of the aortic second sound. In the later stages of the disease a hypertrophy of the right ventricle is often added to that of the left (compare page 853). Complete, or almost complete, absence of the cardiac hypertrophy is observed, as we have said, in weak and cachectic patients.

As long, therefore, as the high arterial pressure kept up by the cardiac hypertrophy regulates the condition of the renal secretion in the way above described, the condition of the patient as a rule shows no special abnormality. At most it happens that certain cerebral symptoms now appear, especially attacks of headache and occasional vertigo, which are probably to be referred to active cerebral hyperæmia. Frequent nose-bleed is also sometimes the result of the abnormally high blood-pressure.

The picture is quite different as soon as the first signs of a beginning cardiac insufficiency appear. Here, as in most diseases of the heart, the disturbance of compensation does not usually come on suddenly. Its results begin quite gradually, disappear for a time, to come on afresh and to increase quite slowly. In the first place, the abatement of the heart's energy usually makes itself manifest by symptoms on the part of the heart itself, and of the lungs. The pulse loses in tension, and becomes smaller, more frequent, and sometimes a little irregular toward the end of the disease. The patient begins to be short of breath, comparatively slight physical exertion affects him more than formerly, and there is often palpitation. Certain anatomical results of stasis may also temporarily or permanently develop in the lungs, especially a mild transitory catarrh, or a more obstinate and recurring bronchitis. In the more advanced stages of the disease the dyspnœa often comes on in pronounced paroxysms, which have an asthmatic character. This long-known asthma of renal disease, often termed, without reason, "uræmic asthma," does not always have the same origin. It usually depends distinctly upon the attacks of cardiac weakness, and is, accordingly, a genuine cardiac asthma, and corresponds precisely to angina pectoris in its different symptoms (see page 318); but in other cases there is a transudation into the lungs from stasis, coming on as a result of the cardiac weakness, so that the dyspnœa is associated with the signs of an acute pulmonary œdema, and is sometimes accompanied by a copious expectoration of a frothy, serous, and often somewhat bloody sputum. This is the condition which may pass away again and be often repeated, which was formerly termed humid asthma. In the last stage of the disease the dyspnœa often becomes continuous, and forms the patient's chief disturbance. It is then usually due, not merely to the stasis in the lungs, but often, besides, to co-existing lobular pneumonia (*vide infra*), to hydrothorax, etc.

As a further sequel of the disturbance of compensation, œdema often appears in different parts of the body, in the later course of the disease. It has, indeed, been repeatedly observed that dropsy may be entirely absent in contracted kidney; but this is the case only when death ensues from some intercurrent attack before the pronounced cardiac insufficiency. Otherwise œdema is by no means rare in contracted kidney. It usually appears first in the ankles, the eyelids, or the prepuce, disappears again when the patient is in a quiet condition, and, after a longer or a shorter pause, comes on anew, until finally, in the last period of the disease, a high degree of general dropsy may develop.

Among the results of the cardiac insufficiency on the internal organs we must first mention the cerebral symptoms. While at first, as we have said, these have more of an active hyperæmic character, the frequent and very violent headaches that come on later, in so far as they are not of a uræmic nature, certainly depend mainly upon the passive hyperæmia of stasis, or the arterial anæmia of the brain. The pain sometimes shoots into the back of the neck, and sometimes is localized chiefly in one half of the head; it is often associated with symptoms of vertigo, with a gloomy or morose disposition, with troubled sleep, etc. The stasis is also apparent in the abdominal organs. Chronic dyspeptic disturbances appear, the appetite fails, the bowels become irregular, and we can even make out a moderate enlargement of the liver. The influence which the altered activity of the heart exerts upon the function of the kidneys themselves is, however, particularly important. From what has been previously said of the dependence of the secretion of urine upon the arterial pressure, it follows directly that any compensatory activity of the still normal renal territory must at once experience a reduction, as soon as the blood-pressure can no longer be kept at the same level. Corresponding to this we see, in fact, that the secretion of urine also usually suffers a decline at the same time with the other symptoms of stasis already mentioned. The amount of urine is less abundant: it falls to forty or fifty ounces (1500–1000 c. c.), and even lower; the specific gravity rises, rarely to a high figure, but still up to 1010 or 1012, or over. The urine often retains its clear color for quite a long time, but may finally more and more resemble the genuine urine of stasis. The point, however, which is especially to be considered, is the co-existing and increasing retention of the solid constituents of the urine in the blood, and the consequent allied possibility of the onset of uræmic symptoms.

It must be stated that, in contracted kidney, the immediate exciting causes of uræmia are not always clear. Thus, it is a well-known and very important fact, clinically, that very severe and often fatal uræmic convulsions may sometimes attack the patient quite suddenly, apparently when in the best of health. Cases have been repeatedly seen, by other observers and by ourselves, where the daily amount of urine has shown no discoverable diminution in the days preceding the uræmia. The precise explanation of these cases must remain undecided ; we do not know whether there has been a previous retention of the solid constituents, in spite of the abundant secretion of water, or whether other changes, like œdema of the brain, are to be considered. It is certain, however, that in many cases at least, the onset of the uræmia is connected with the cessation of the secretion of urine, produced either gradually or suddenly by the cardiac insufficiency. In the former case the type of chronic uræmia (see page 829) develops with especial frequency; this consists of headache, vomiting, diarrhœa, severe pruritus of the skin, etc., but these symptoms are, of course, often combined with the immediate symptoms of stasis, and are not always to be easily and distinctly separated from them. Such a condition of chronic uræmia, in patients with contracted kidney, often presents a very mournful picture, since the unrestrainable and constantly recurring vomiting, the headache, and the general mental anxiety may last for weeks. The severe acute uræmia either follows the preceding chronic uræmic symptoms, or comes on at once with the severest symptoms, general and often-recurring convulsions, and coma. The uræmia may pass off again, even in contracted kidney, but quite frequently it is the immediate cause of death (*vide infra*).

Beside the symptoms so far described, we must now mention a set of anatomical complications which may appear in the course of contraction of the kidney. From its diagnostic and clinical importance the albuminuric retinitis, already known to us from the preceding chapter, takes the first place. It may come on at any time in the course of the disease ; but it often develops so early that the patient, up to this time, knows nothing at all of his other disease. He merely consults an oculist, who often first recognizes, from the ophthalmoscopic picture (see page 854), the special seat of the primary disease. Even in the cases where no subjective visual disturbance is present, the retinal examination sometimes shows a positive image. In general, the contracted kidney is that form of renal disease in which retinal changes are comparatively the most frequent.

Another rarer but clinically important complication consists of the hæmorrhages of internal organs, whose cause is to be found either in the increased arterial pressure, or in an abnormal weakness of the walls of the vessels—arteriosclerosis in older persons, defective nutrition of the vascular walls in young and anæmic patients. Hæmorrhages into the brain are comparatively the most frequent. They cause both mild and severe apoplectic attacks, which pass off completely or leave a hemiplegia behind, and sometimes they are the direct cause of death. Beside the hæmorrhages into the brain itself, there may also be hæmorrhages on the inner surface of the dura mater—hæmatoma. Nose-bleed is also of significance; in many patients it is frequent and very stubborn; we have ourselves seen two cases where the fatal termination was caused directly by an unrestrainable nose-bleed. Hæmorrhages into the other organs are more rare, but they have also been observed in the skin, the stomach, the intestines, or the lungs. In a few cases, indeed, a sort of hæmorrhagic diathesis seems to develop.

Among the complicating inflammations of internal organs, pneumonia is the most frequent; it appears in the lobar croupous form or in the lobular form peculiar to all varieties of nephritis. Inflammations of the serous membranes, pleurisy, or pericarditis, occur, but they are rare. The catarrhal inflammatory affections—laryngitis, bronchitis, gastric catarrh, intestinal catarrh—are either to be regarded as catarrhs from stasis, or they are perhaps connected with the retention of the urinary constituents in the body. We must also refer here to the acute exacerbations of inflammation in the kidneys themselves (acute recurrent nephritis), which is, of course, rare.

Quite great variations appear in regard to the general nutrition. In most cases where the disease develops quite gradually in persons in middle or advanced life, the general nutrition for a long time shows no striking anomaly. The patient is often very well nourished, and even corpulent, at the period when the first cardiac symptoms begin. To the more practiced and attentive eye, of course, he shows a certain appearance of suffering, which later becomes more pronounced. He becomes emaciated, and has a faded and often somewhat cyanotic color to his skin. Marked anæmia usually develops only in younger patients, who then show the pallid exterior characteristic of so many patients with renal disease.

General Course, Duration, and Termination.—The most important peculiarities in the course of renal contraction have already been spoken of above. We have stated that the disease may be latent for a long time; that the severest symptoms—like uræmia or apoplexy—sometimes come on suddenly and unexpectedly; that in other cases the disturbances of compensation in the heart, dyspnœa, palpitation, or slight œdema, are the first symptoms; that, under some circumstances, certain complicating conditions, such as retinitis, or frequent nose-bleed, first direct suspicion to a renal disease, and demand an examination of the urine; while, finally, in a last class of cases, only general disturbances, loss of appetite, pallor, general physical weakness, and similar symptoms induce the patient to consult a physician. It is usually hard to decide how long the disease has lasted before a diagnosis is made. Beside any mild symptoms we must especially inquire into the existence of polyuria, which is often not observed, but which many patients notice.

The further course may also vary according to the onset of complications, the external conditions under which the patient lives, etc. In general, as we must repeatedly emphasize, almost everything depends upon the heart's capacity for work and its staying qualities. If death does not ensue sooner from some intercurrent disease, the last stage of the disease almost always presents itself under the picture of cardiac insufficiency with predominant symptoms of dyspnœa and general dropsy.

As has been said, we usually can not determine with any accuracy the whole duration of the disease. It may, at any rate, last many years, probably sometimes even ten years or more, although there may be many variations in its course. It is not impossible that, during the earlier period of the disease, there may be a cessation in the process of renal atrophy, but it is hard to decide with certainty. At all events, the disease must generally be termed completely incurable, although life may not only be preserved for a long time, but the patient may even exist without much discomfort. We need not refer especially here to the different intercurrent attacks, the possibility of which must always be kept in mind in regard to prognosis.

Diagnosis.—The diagnosis of contracted kidney can be made with certainty only by examining the urine. We must, therefore, dwell again on the necessity of making this examination in all suspicious cases, because only in this way can we avoid overlooking the condition. The suspicion of a developing renal contraction should demand an examination of the urine, especially in all cases where the patient complains of frequent headache, of congestive conditions, of palpitation and dyspnœa, asthmatic attacks, disturbances of vision, general dullness, and dyspeptic symptoms, without finding any other reason for these symptoms. The polyuria, the clear urine of low specific gravity, containing a slight amount of albumen, in connection with the signs in the circulatory apparatus, the tense pulse, and the hypertrophy of the left ventricle, permit us to recognize the disease correctly in most cases. If retinal changes are present, they may sometimes be of much aid in confirming the diagnosis. The ætiological conditions—lead, gout, alcoholism, etc.—of course also merit attention.

The diagnosis presents great difficulty in the quite rare cases where albuminuria is absent. In these cases we are sometimes able to reach the correct interpretation of the morbid condition only by repeated examinations of the urine. Otherwise we can scarcely avoid mistaking it for chronic affections of the heart, such as myocarditis or idiopathic hypertrophy.

The diagnosis is also very difficult if the patient does not come under observation until the stage of fully developed disturbance of compensation. The characteristic features of the urine of contracted kidney are then absent, the urine is scantier, darker, richer in albumen, and it is often scarcely possible to decide whether we have a primary renal affection with secondary cardiac hypertrophy or a primary heart disease with a secondary congested kidney. If general arteriosclerosis or marked pulmonary emphysema is present at the same time, the judgment as to the condition is still more difficult. In such cases a correct diagnosis is possible only by very carefully balancing all the different symptoms, and considering the whole course of the disease.

Finally, the diagnosis of contracted kidney is very difficult in cases where the first examination of the patient is made during a sudden attack of uræmia or after an apoplectic seizure. Here the albuminuria is the symptom which points most to the existence of a renal disease, although, in spite of this symptom, the judgment as to the condition, and its differentiation from other acute cerebral affections, often presents great difficulties.

Treatment.—As soon as the diagnosis of renal contraction is established, the whole hygienic condition of the patient must be regulated so as to prevent the advance of the affection in every possible way. In this respect two indications are to be fulfilled, to guard against any irritation which may have an injurious action on the kidneys, and to relieve the work of the heart as much as possible, in order to keep off cardiac insufficiency as long as we can. The diet must be carefully regulated, and must be of scant measure or abundant and strengthening, according to the patient's physical constitution. In these cases, too, milk is the chief food to be considered. Alcoholic beverages are to be permitted only in a moderate degree. All physical over-exertion is to be avoided, although moderate methodical exercise is to be recommended for corpulent patients. We should always provide for regular evacuation of the bowels by appropriate remedies, dietetic prescriptions, fruits, bitter waters, etc. The general condition is often materially improved by proper air and recreation, and in this way the use of a bath may be of service, chosen according to the individual conditions, such as iron baths, Marienbad, Carlsbad, Kissingen, Ems, or Baden-Baden.

If disturbances of compensation appear, the dietetic *régime* and the utmost bodily rest possible are still more strictly to be advised, and a symptomatic treatment should be instituted according to the prevailing symptoms. First of all we must try to excite the heart's energy anew by the exhibition of digitalis. The details of treatment here are almost precisely the same as are considered in the treatment of chronic heart disease (*q. v.*) and the other renal affections. [The nitrites, especially nitroglycerine, are of great service in some cases of contracted kidney, and may be used persistently when the arterial tension is unduly high, and the compensatory hypertrophy of the left ventricle shows signs of failing. In these cases strophanthus, which is said not to increase arterial tension, may at times be substituted for digitalis with advantage.]

At present it is impossible to influence the process of contraction in the kidney favorably by the direct use of drugs. The iodine preparations—iodide of potassium, and in anæmic patients iodide of iron—are alone recommended, and are worth trying in this respect.

The prophylaxis of renal contraction is evident; we should consider as far as we can the known ætiological conditions. We may compare, in this regard, the chapter on gout.

CHAPTER V.

AMYLOID KIDNEY.

Ætiology.—The amyloid kidney is invariably a part of the more or less extensive amyloid degeneration of the organs in the rest of the body. In its clinical relations, however, it claims the most interest of all amyloid diseases, since it has by far the greatest significance for the whole clinical picture of amyloid degeneration.

As is well known, we understand by amyloid degeneration a peculiar change which, under certain pathological conditions, is observed in the connective tissue, and especially in the smaller vessels. The walls of the vessels are thicker, they have a lustrous, homogeneous appearance, and they show peculiar reactions on treatment with certain coloring agents. These reactions are due to the presence of an albuminoid substance—amyloid—which is either deposited in the tissue from the blood, or, as is much more probable, is developed in that place and spot from the albuminoid substances present. In marked amyloid degeneration the diseased organs often show macroscopically an altered, "bacon-like" appearance, and assume a characteristic red-brown color on treating the affected parts with Lugol's solution of iodine. The microscopic examination alone affords more accurate conclusions as to the presence and distribution of the degeneration. Here we make use chiefly of staining the tissues with methyl-violet or gentian-violet. The amyloid portions thus take on a very characteristic and easily defined red color. In this way we can discover that the amyloid degeneration begins everywhere in the walls of the small vessels, that the interparenchymatous connective tissue may also be affected later, but that the parenchymatous cells proper, liver cells, renal epithelium, etc., almost always remain perfectly free. The latter often show atrophic and fatty degenerative changes (*vide infra*), but never amyloid degeneration.

Nothing is known as to the special causes which effect that peculiar metamorphosis of the albumen of the connective substance into amyloid. We know only that there are a number of primary diseases in which it is known empirically that amyloid degeneration quite frequently develops as a secondary condition in the different organs. These conditions have mainly the common feature that they go along with a general cachexia and weakness of the body. The following conditions (arranged in about the frequency of the occurrence of amyloid degeneration) are the special ones in which amyloid degeneration in general, and therefore amyloid kidney in particular, are chiefly observed :

1. Chronic pulmonary tuberculosis, particularly the ordinary ulcerative phthisis. Tubercular ulcers of the intestines, with or without co-existing marked pulmonary tuberculosis, may also lead to amyloid disease.

2. Long-continued chronic suppuration in the bones or soft parts, especially chronic fungous processes with fistulæ into the bones or joints, empyema with fistulæ, vertebral caries, etc.

3. Constitutional syphilis, chiefly the cases with ulcerative and usually tertiary processes in the bones and mucous membranes.

4. Other ulcerative processes or processes associated with chronic suppuration: saccular bronchiectases, chronic intestinal ulcers (for example, of dysenteric origin), purulent pyelo-cystitis, vesico-vaginal fistula, ulcerated new growths, like cancer, etc.

5. In rare cases amyloid has also been observed in other chronic diseases, as in malaria, gout, and other chronic articular affections. In the medical clinique here at Leipsic we once saw a case of marked amyloid kidney in a girl of twenty-one with aortic insufficiency.

6. Finally, in a small class of cases, of which we have ourselves seen some examples, no discoverable cause at all may be found at the autopsy for a usually quite extensive amyloid degeneration. In these cases there accordingly seems to be a primary amyloid disease.

Pathological Anatomy.—Since the text-books on pathological anatomy have been referred to in regard to the anatomical conditions of amyloid in other organs (compare also page 498), we must here describe in detail only the pathological anatomy of amyloid kidney.

In very slight and not extensive amyloid degeneration in the kidneys the latter present a perfectly normal appearance to the naked eye. Careful microscopic examination alone shows amyloid degeneration of the walls of different vessels in the cortex, and especially in the medullary substance.

The commonest and most characteristic form of amyloid kidney is the so-called large white amyloid kidney (waxy kidney, lardaceous kidney). The kidney is usually enlarged, and the surface is smooth and of a grayish-white or yellowish color, and usually somewhat mottled. On section, the cortex is wider and also of a yellowish-white color, and the glomeruli may often be recognized with the naked eye as dull, lustrous, translucent points. Hæmorrhages are scarcely ever seen. The medullary substance is either also pale or darker. In many cases the cortex may also have a darker reddish or mottled appearance, which is due merely to the greater amount of blood in the organ. The pale-yellow color is due either to the anæmia or to the fatty degeneration, while the amyloid spots show a more translucent character with a bacon-like luster.

If we examine the kidney microscopically, we find first the amyloid degeneration, which, in varying extent and combination, affects most frequently the glomeruli and also the capillaries of the cortex, the vasa recta, and sometimes the membranæ propriæ of the uriniferous tubules. In pure amyloid kidney the rest of the renal tissue is normal, but in many cases we also find changes in the epithelium, fatty degeneration, desquamation and disintegration, and also not infrequently interstitial cellular infiltration.

Thus amyloid degeneration is often combined with degenerative inflammatory changes in the kidneys. If the process has lasted for a long time, it leads, as in ordinary nephritis, to a complete atrophy of tissue in some parts, with a corresponding increase of connective tissue. Then the renal tissue sinks in at the affected parts, and there is a decided unevenness to the surface of the kidney. There is even a completely developed red or white contracted kidney, in which we find abundant amyloid, and which is, therefore, termed amyloid contracted kidney. In this form the parenchymatous and interstitial changes correspond precisely to those in ordinary contracted kidney, only the amyloid degeneration is added to them.

At present differences of opinion prevail as to the precise connection between amyloid and the inflammatory degenerative processes in the kidney. Probably we have an actual combination of both conditions, sometimes perhaps even co-effects of the same cause. For since genuine nephritis, as well as amyloid kidney, occurs, as we have seen, in syphilis, tuberculosis, and other diseases, it can not be remarkable that in these diseases both sequelæ, nephritis and amyloid, develop side by side, and that we may, therefore, find at the same time, beside the changes of an inflammatory large white kidney, of a secondary contraction, or of a genuine contracted kidney, a more or less extensive amyloid degeneration in the kidneys. On the other hand, it, of course, can not be questioned that the disturbance of circulation, which must arise in consequence of a marked amyloid of the vessels, is of influence on the nutrition of the renal tissue, and that, therefore, many changes in it, especially fatty degeneration of the epithelium, are, under some circumstances, the direct result of the amyloid disease.

Clinical History.—If we consider the great differences which the distribution of the amyloid in the kidneys shows, and its manifold combinations with inflammatory processes, it is clear from the outset that we can not set up a uniform picture of the symptoms of amyloid disease in general. To this we must add that the symptoms of amyloid disease, which is almost always a secondary condition, are also modified in various ways by the primary disease.

We must first state that many cases, where the amyloid in the kidneys is of comparatively slight extent, can not be recognized by any clinical symptom. The albuminuria in particular may be entirely absent, as has been repeatedly proved (Litten and others), which is perhaps due to the fact that in such cases the vasa recta and not the glomeruli are chiefly affected by the amyloid degeneration.

Except in these cases, however, the urine secreted from the amyloid kidneys shows marked changes, which, of course, present quite great variations according to the form of the individual case. The amount of urine is most frequently about normal, or somewhat diminished—in some cases much diminished—but in others it is decidedly increased, so that the patient may pass eighty to a hundred and twenty ounces (2500–3500 c. c.) in the twenty-four hours. We quite frequently see considerable variations in the amount of the urine in the same patient at different times. All these differences are easily explained if we remember how many circumstances may act on the amount of urine—the presence or absence of inflammatory changes in the kidney, the presence or absence of cardiac hypertrophy, co-existing perspiration, diarrhœa, œdema, fever, etc.

The color of the urine is almost always light yellow. Only exceptionally, in amyloid nephritis, does it contain an abundant sediment; usually it is entirely, or almost entirely, clear. The very considerable amount of albumen in the urine, which is often one or two per cent., is also characteristic of amyloid kidney. In many cases, of course, especially in amyloid contracted kidney, the amount of albumen is but slight. It sometimes, but by no means regularly, happens that the urine in amyloid kidney contains a comparatively large amount of paraglobuline beside the ordinary serum albumen (Senator).

The specific gravity of the urine varies very much according to the amount of water and albumen in it. It may be increased (1015–1020) or diminished (1010–1003).

If we examine the urine under the microscope, we usually find only a few hyaline casts, and also most frequently a small number of white blood-corpuscles. In the combination of amyloid with more marked nephritic changes the sediment is more abundant, so that the urine is cloudy. The microscope then shows more numerous hyaline or moderately fatty casts, more abundant white blood-corpuscles, sometimes a little renal epithelium, and in quite rare cases even red blood-corpuscles. Amyloid reaction occurs in the casts, but it is very rare, and therefore of no value in diagnosis.

The other morbid symptoms which are observed in amyloid kidney depend either upon the change in the kidneys themselves, or upon co-existing amyloid degeneration in other organs; or, lastly, upon the primary disease. The symptoms of the latter are, of course, extremely varied, but in many cases they may be entirely subordinate.

In regard to the directly resulting symptoms of amyloid kidney, their occurrence is of interest, especially in comparison with the analogous conditions in acute nephritis. Dropsy of a moderate, or even a severe degree, is often present in amyloid kidney, but it may also be entirely absent. We must remember that an œdema independent of a renal affection may be produced by marantic venous thrombosis. Uræmic symptoms are distinctly rare in amyloid kidney, but they are sometimes observed, especially in their milder forms, such as vomiting. It is a very important point that a hypertrophy of the left ventricle is absent in most cases of amyloid kidney. This is because we have weak and cachectic individuals, in whom a cardiac hypertrophy can not develop for lack of an excess of nutritive material. Where there is no debility, a cardiac hypertrophy may doubtless develop, as we see especially in amyloid contracted kidney. At the autopsy we often find the heart, of course, in a condition of brown or simple atrophy.

Albuminuric retinitis hardly ever appears in pure amyloid kidney. In the amyloid contracted kidney it has sometimes been observed, however, in cases where there has probably been originally a pure contracted kidney, with amyloid coming on later. The secondary inflammations in the internal organs, such as renal pneumonia, and the hæmorrhages, like cerebral hæmorrhage, are also rare.

The patient's general condition is also dependent in part upon the renal disease, but usually upon the primary disease. The patient with amyloid kidney is usually correspondingly cachectic, and shows in high degree a pallid, anæmic color of the skin. In some cases, however, such as syphilis, bronchiectasis, or unilateral contraction of the lung, the condition of the nutrition may remain tolerably good for a long time.

The symptoms which point to a co-existing amyloid degeneration in other organs beside the kidneys are of great diagnostic significance. The symptoms in the liver (enlargement, abnormal firmness, and a hard, sharp lower edge to the organ), the spleen (enlargement and hardness), and intestines (diarrhœa) are clinically important in this respect. The explanation of the diarrhœa is, of course, usually difficult, since it may often depend upon tubercular intestinal ulcers as well as upon amyloid of the intestines.

We can scarcely make more general statements in regard to the whole course and the duration of amyloid kidney, since the form of the primary disease is to be especially considered in these cases. In regard to the time that it takes for an amyloid degeneration to develop in an existing primary disease, the degeneration is certainly present sometimes after a few months. Of course, it is hardly ever possible to determine its onset accurately, since the first beginnings of amyloid degeneration in the kidneys do not usually permit themselves to be recognized at once by the appearance of albuminuria (*vide supra*). The duration of amyloid kidney varies very much according to the severity of the case; it may last only a few weeks or months until death, while other cases have certainly lasted for a year, especially in amyloid contracted kidney.

The **prognosis** of amyloid kidney is in most cases utterly unfavorable, which is due mainly to the incurability of the primary disease; but trustworthy observers have repeatedly proved that, when the primary disease is curable, as with syphilis and many chronic suppurations, an already developed amyloid kidney *can* be completely restored.

The **diagnosis** of amyloid kidney can be made with quite great certainty when the evident signs of a renal affection are added to those diseases which we know empirically often give rise to the development of amyloid degeneration. Whether in such cases we have a pure amyloid or a pure nephritis, or a combination of the two, can be decided with some certainty only from the condition of the urine: a clear urine, containing but few morphological elements, but rich in albumen, points to amyloid, while a large number of casts and red and white blood-corpuscles in the urine, point to the presence of inflammatory changes in the kidney. A very characteristic symptom of many cases of amyloid kidney, and one that is therefore of diagnostic value, is the rapid and frequent change in the amount of the urine and in the amount of albumen in it (Wagner). An accurate diagnosis of the anatomical changes, however, is hardly ever to be made with certainty, and at most can be made only by attention to the whole course of the disease.

A very material support for the diagnosis of amyloid kidney, and therefore one which should always be looked for, is the discovery of amyloid in other organs. We have briefly mentioned above the most important symptoms in the liver, the spleen, and the intestines referable to this point.

Treatment.—Only the treatment of the primary disease can, of course, be considered, both as a prophylactic and also as a causal indication. In many surgical cases, and also in the cases of amyloid in syphilis, there is a possibility of this (as by iodide of potassium) ; but otherwise we try to improve the primary disease as far as is possible.

In other respects the treatment is purely hygienic and symptomatic. We must try to strengthen the patient as much as possible by good food and the exhibition of preparations of iron and quinine. The use of iodide of iron is to be recommended. In a symptomatic point of view the same remedies are used as in other renal diseases.

CHAPTER VI.

PURULENT NEPHRITIS AND PERINEPHRITIS.

(*Renal Abscess.*)

Ætiology.—Although in the forms of nephritis so far described the occurrence of interstitial accumulations of granular matter has been repeatedly mentioned, none of them ever come to genuine suppuration—that is, to a purulent liquefaction of tissue, a true abscess-formation. The origin of a purulent nephritis is, rather, always associated with the entrance of perfectly definite morbid irritants into the kidneys. These are invariably organized and their special peculiarity is to excite a purulent inflammation.

There are two chief ways through which the morbid irritants may reach the kidneys—the arterial blood-current and the urinary passages. The first-mentioned means of entrance is to be considered in all the cases of purulent nephritis which come on as one symptom of pyæmic processes and certain forms of ulcerative endocarditis (see page 112 on the point). Far more rarely purulent nephritis develops in this way as a complication in other diseases, such as dysentery. Purulent nephritis also occurs in actinomycosis (Israel).

The excitants of inflammation take the second path in those cases where a purulent nephritis follows an inflammation of the lower urinary passages, the pelvis of the kidney, the bladder, etc. Here the bacteria, which almost always enter directly into the urinary passages (the urethra and bladder) from without, pass gradually upward from the bladder through the ureters to the pelvis of the kidney; from this they enter the apertures of the collecting tubes and the uriniferous tubules of the kidney, everywhere exciting a purulent inflammation. We therefore term these forms of purulent nephritis—with regard to their origin—purulent pyelo-nephritis.

We must remark in conclusion that a purulent nephritis may arise in direct wounds of the kidney from infection of the wound; this is usually associated with a perinephritic suppuration (*vide infra*).

Pathology.—Purulent nephritis shows quite characteristic peculiarities and differences according to its mode of origin. (We omit traumatic abscesses here.)

The renal abscesses in pyæmia and analogous diseases are usually focal suppurations, which only exceptionally attain a great extent, but which are usually to be recognized with the naked eye as numerous little yellowish dots or lines, scattered over the whole kidney, about half a millimetre or a millimetre in diameter. On microscopic examination, these nodules prove to be genuine little abscesses, in whose territory the renal tissue proper is completely destroyed. In the middle of them we often find the originating colony of micrococci, the "micrococci embolus," seated in a central vessel. The conditions are still plainer if we examine the earlier stage of the process. We find vessels (the loops of the glomeruli, or the encircling capillaries), which are completely plugged with micrococci, and in whose vicinity the renal tissue is still perfectly normal. We further see analogous spots where the renal tissue is already necrosed in the vicinity of the colony of micrococci, and is infiltrated with emigrated cells. These nodules show, finally, a continuous transition to the completed abscess, which is often surrounded by a hyperæmic or even a hæmorrhagic areola.

In purulent pyelo-nephritis the renal abscesses appear somewhat different. The abscesses also have a characteristic striated appearance, corresponding to the distribution of the inflammation along the straight tubules. They often extend from the point of the renal papilla through the cortex to the surface of the organ, so that from the outside we see the abscesses, appearing through as yellowish points. The broader abscesses arise from the confluence of neighboring striæ. The microscope shows that the purulent inflammation arises from the vessels of the interstitial tissue, in whose territory the uriniferous tubules are of course destroyed. The clusters of micrococci form the most interesting feature. These settle originally in the uriniferous tubules and are the special cause of the necrosis of epithelium and the inflammation. Pyelo-nephritis, indeed, was one of the first diseases for which Klebs discovered a bacterial origin.

Clinical Symptoms.—We can speak very briefly here in regard to the clinical symptoms of purulent nephritis, since they can never be sharply separated from the symptoms of the primary disease. The pyæmic renal abscesses, and the abscesses in ulcerative endocarditis, hardly ever cause special clinical symptoms, so that their presence is first recognized on the autopsy-table. Since the abscesses do not usually communicate with the uriniferous tubules, there is usually not even a large amount of pus in the urine.

The clinical symptoms of pyelo-nephritis also depend less upon the nephritic abscesses than upon the previous and accompanying pyelitis and cystitis. We will therefore return to renal abscesses in the description of these diseases.

PERINEPHRITIC (PARANEPHRITIC) ABSCESS.

Perinephritic abscess is the name given to suppurations in the vicinity of the kidney, especially in its fatty capsule or in the peri-renal connective tissue. Apart from any traumatic origin for such abscesses, they develop most frequently as a result of purulent nephritis or purulent pyelitis. The escape of pus, which involves the surrounding tissue in the inflammation, may come from the ureter or pelvis of the kidney, or from the kidney. The special form of primary disease differs very much; it may be either simple purulent pyelitis, or pyelitis from renal calculi, or sometimes tubercular processes and new growths that finally suppurate, such as cancer, or echinococci. The peri-renal suppuration may also take its start from the other organs in the neighborhood. Thus cases have been seen in which the perinephritis followed a perityphlitic abscess, a hepatic abscess, or a psoas abscess after vertebral disease. Perinephritic suppuration may also be due to actinomycosis.

In many cases of this sort the accumulation of pus is so considerable that there is a protrusion in the lumbar region exactly like a tumor. This is at first only obscure; but later the skin becomes œdematous there, it constantly protrudes more and more, it assumes an inflammatory hyperæmic redness, until finally a definite fluctuation shows the advance of the abscess up to the skin. In other cases the inflammatory swelling extends forward into the iliac fossa; then there is also abnormal resistance and dullness above Poupart's ligament. The swelling may also extend upward toward the diaphragm, so that the diaphragm is crowded upward, giving rise to marked dyspnœa. The relations of the swelling to the descending colon are sometimes the same as in new growths of the kidney (compare Chapter VIII).

Beside the swelling, there is almost invariably a very great pain in the affected region, either spontaneous or on pressure. If the swelling presses on the large nerve-trunks in the vicinity, it produces severe shooting pains in the leg of the same side, and sometimes a numb feeling and paresis. The leg is then often kept in a position similar to that in coxitis.

The condition is almost always associated with fever, which shows the characteristic remitting or intermitting type of most suppurative fevers, and may be interrupted by occasional chills with a marked rise of temperature. The patient becomes very much debilitated and emaciated by the fever and the pain, and he may finally fall into a sad general condition. The urine contains pus only when the abscess communicates in some way with the urinary passages.

Recovery can take place only when the abscess is evacuated through some external channel. Except for operative procedures, the spontaneous escape of pus through the skin is the most favorable; this most frequently takes place in the lumbar region, or more rarely, like a psoas abscess, under Poupart's ligament. Sometimes persistent fistulæ are left after such a rupture. The rupture of the abscess into the intestine (the colon) has also been observed, with an evacuation of pus by the bowels, and also ruptures into the bladder, the pleural cavity, and the peritoneum. We need not here discuss in detail under what circumstances death may ensue; it comes on, in many cases, after the disease has lasted a considerable time.

The diagnosis is based chiefly upon the swelling, the tenderness, the fever, and a consideration of the ætiological factors. The disease may be confused with hydro-nephrosis, psoas abscess, or solid renal tumors. The result of an exploratory puncture is sometimes decisive in such cases.

The only treatment, apart from the fulfillment of any symptomatic indications, is surgical, and consists, if possible, in opening and draining the abscess. The success depends chiefly upon the patient's general condition, and the form of the primary disease. The details are to be found in the text-books of surgery.

CHAPTER VII.

DISTURBANCES OF CIRCULATION IN THE KIDNEYS.

1. **The Congested Kidney.**—Although local impediments to the flow of venous blood from the kidneys, as from thrombosis of the renal veins, hardly ever attain a clinical significance, the participation of the kidneys in a general venous stasis, as it is chiefly seen in heart disease (compare page 300), pulmonary emphysema, etc., is of great diagnostic importance, since we possess in the condition of the urine quite an accurate measure of the intensity as well as of the increase and decrease of the stasis.

The congested kidney is easily recognized anatomically. The organ is often somewhat enlarged, it feels firmer than normal, and shows, both on its surface and on section, a dark, bluish-red color—" cyanotic induration." The medullary substance is usually darker than the cortex. Under the microscope we see considerable dilatation and a tense fullness of the veins and capillaries. The parenchyma is normal, but in more advanced cases it may show a beginning fatty degeneration of the epithelium, which is a result of the defective arterial blood-supply. Interstitial changes are usually absent.

The clinical symptoms of congested kidney concern only the changes in the urine. The amount of urine diminishes, corresponding to the diminution of the arterial pressure and the diminished rapidity of the blood-current. Only twenty or twenty-five ounces (800–500 c. c.), or less, are secreted daily. The urine is also more concentrated and darker than normal, and often has an abundant sediment of uric acid or urates. If nutritive disturbances have begun in the epithelium of the glomeruli as a result of stasis, the urine is also albuminous, but the amount of albumen in pure congested kidney is always slight—about one tenth to one sixth of the volume. The urine often contains, besides, a few hyaline casts, and a few white and red blood-corpuscles, the latter pointing to little congestive hæmorrhages.

If the changes mentioned come on as one symptom of a general venous stasis, and are, accordingly, often associated with cyanosis, and dropsy, the diagnosis of congested kidney can be made with certainty. If we succeed in restoring the circulation by appropriate remedies, such as digitalis, the urine at once becomes more abundant and clearer and its albumen disappears. Otherwise the symptoms of the urine of passive congestion last until the patient's death.

2. **Embolic Infarction in the Kidneys.**—Since the renal infarction, although it has great pathological interest, is hardly ever of clinical significance, we will limit ourselves here to a brief description of the most essential points.

If one of the smaller renal arteries is plugged by an embolus in heart disease, the affected portion of the organ cut off from the circulation must perish, since all the renal arteries are terminal arteries. The epithelium undergoes the well-known changes of coagulation necrosis, disappearance of the nuclei of the cells, and disintegration, and the tissue becomes entirely or in part a hæmorrhagic infarction (compare page 300). In this way arise the characteristic wedge-shaped, red, hæmorrhagic infarctions in the kidney, or far more frequently the yellowish-gray, anæmic infarctions, often surrounded by a hæmorrhagic areola, the base of which is at the surface of the kidney; the base may reach a width of half a centimetre to a centimetre or more, while its apex extends a varying distance into the cortex, or even into the medullary substance. Later on the gradually disintegrated tissue of the infarction is absorbed, round cells emigrate from without into the region destroyed, and a shrunken connective-tissue cicatrix gradually develops in place of the former infarction. Many kidneys may have such a granular surface from numerous infarction cicatrices that they may be appropriately termed "embolic contracted kidneys."

The anatomical processes just briefly described cause in most cases no special clinical symptoms at all. Only in a few cases does a slight amount of blood in the urine seem to depend on the development of a hæmorrhagic infarction in the kidneys, so that when a cause for embolic processes, like heart disease, is present, we may sometimes entertain the suspicion of the development of a renal infarction during life. In a few cases the development of a large renal infarction may be accompanied by a sudden severe pain in the renal region.

The embolic processes in the kidney never demand special treatment.

CHAPTER VIII.

NEW GROWTHS IN THE KIDNEYS.

Of the primary forms of tumor occurring in the kidney, two especially claim our interest: cancer of the kidney and congenital sarcoma. The latter is of great importance in regard to the general theory of tumors, since it points definitely to the development of the new growth from scattered portions of embryonic tissue. Striped muscular fibers have been repeatedly found in tumors consisting otherwise of round or spindle cells, from which the name of "*rhabdomyoma*" has been chosen for these tumors. Since there are no muscular fibers in the kidney itself, their occurrence in the tumors points undeniably to disturbances of development. This theory obtains a further interesting confirmation from our own observation of the development of left-sided, and, probably, congenital renal sarcoma in two brothers. Both children died when between two and three years of age, and the autopsy gave almost precisely the same lesion in both: numerous metastases in the liver and lungs, beside a new growth almost as large as a child's head in place of the left kidney.

Renal cancer is also remarkably frequent, comparatively speaking, in children under four years of age, and about equally common in the two sexes, although, of course, we find renal cancer in persons of more advanced years. Usually only one kidney is affected, chiefly the left, as it seems, but the new growth has sometimes been found in both kidneys. In its character, renal cancer belongs either to the denser or to the softer, medullary form. It may permeate the whole kidney and change it to a large tumor, weighing fifteen or twenty pounds (five or ten kilogrammes). Softening and hæmorrhage very often take place within the tumor. The proliferation has been repeatedly observed to extend to the neighboring parts, especially the pelvis of the kidney, and metastases also form in other organs, as in the liver or the lungs.

The clinical symptoms of renal tumor are entirely absent, or of a very indefinite nature, in the first period of the disease. Dull pain in the renal region is repeatedly given as the first symptom, but, of course, this is hardly ever of definite significance. The diagnosis almost always first takes a definite direction by the appearance of a palpable tumor. This develops in the lumbar and lower lateral abdominal region, constantly extending from this point upward and inward. As stated above, both carcinoma and sarcoma of the kidney may cause enormous tumors, especially in children, which may make the whole abdomen protrude to a marked degree. The tumor feels dense, and either smooth or rough, and it does not move with the respiration. The relation of the tumor to the descending colon in left-sided renal tumors is not unimportant in diagnosis. Since the latter is pressed forward by the growth of the tumor, and thus gets into a position between the new growth and the anterior abdominal wall, we can often succeed in making out by percussion, and sometimes even by palpation, that the affected portion of intestine, and sometimes even a loop of small intestine besides, is drawn forward over the tumor; the percussion may vary as the large intestine is empty or full. In right-sided renal tumors analogous conditions may also be present, but they are rarer. The liver is then sometimes pushed to the left.

In many cases of renal tumor the urine shows no abnormal conditions at all, since its secretion is performed compensatorily by the other healthy kidney. In carcinoma of the kidney it sometimes presents, however, one valuable sign for diagnosis—namely, an admixture of blood. This hæmaturia often comes on very early, even before there is any tumor to be felt. It is repeated either frequently or only rarely in different cases, and sometimes it is entirely absent. The hæmorrhage is associated with colicky pains only when large clots have to pass through the urinary passages. It is a remarkable fact that in a few cases the blood, as it seems, may come from stasis in the healthy kidney, which is very hyperæmic. Sometimes, but very rarely, small particles and shreds of tissue from the disintegrated new growth may be found in the urine.

The general symptoms are often very late at first, especially in children; but finally a general condition of marasmus almost always develops. A constant and great frequency of the pulse is often striking. We must also mention the peculiar symptoms several times observed, that in girls with congenital renal tumors there is an abnormally early development of the pubic and axillary hair, and sometimes a peculiar pigmentation of the skin (Kühn). We need not mention in detail the symptoms of compression of neighboring organs by the tumor, which may develop in different ways.

The diagnosis of renal tumor may be made with quite great certainty in many cases. The position of the tumor, its immobility, its relation to the large intestine, and, especially, our general experience as to the occurrence of renal tumors in children, lead us at once to think of the correct diagnosis. In older people renal hæmorrhages, which are otherwise inexplicable, must direct our suspicions toward the possibility of a renal cancer. Renal tumors may, of course, often be mistaken for retroperitoneal glandular tumors, ovarian tumors, large psoas abscesses, or splenic tumors. The differential diagnosis must accordingly be carefully considered in every case.

The prognosis is, of course, always unfavorable. The disease sometimes lasts only a few months, sometimes a year or two, rarely longer.

The treatment must in most cases be purely symptomatic. The only expectation of success lies in the operative removal of the new growth, the details of which are to be found in the later writings on renal surgery.

CHAPTER IX.

PARASITES OF THE KIDNEYS AND OF THE URINARY PASSAGES. CHYLURIA.

Echinococcus of the Kidney.*—Echinococcus cysts have been repeatedly found in the kidney, although much more rarely than in the liver. Usually only one kidney is affected, and the parasite is generally situated in the renal substance itself, only exceptionally between it and the capsule of the kidney. The size of the echinococcus cysts may be very considerable, the diameter reaching to twenty centimetres or more.

Clinical symptoms usually first appear when the tumor can be felt through the abdominal walls. Subjective symptoms may even then be entirely wanting. Pain on pressure develops gradually later. The tumor usually has an approximately globular shape. Its relations to the neighboring organs, especially to the colon, are the same as we have learned to recognize in the preceding chapter, in the description of cancer of the kidney. The feeling of the so-called "hydatid thrill," from a sudden impulse on the tumor with the palm of the hand against it on palpation, is characteristic of echinococcus, but, unfortunately, it is but seldom manifest.

It happens rather frequently that the sac of the echinococcus bursts into the pelvis of the kidney. Then single echinococcus cysts, or at least bits of membrane, hooks, etc., are passed with the urine, usually with severe colicky pains, which are exactly like the renal colic from the passage of a calculus. Such attacks may be often repeated, and may form a very severe type of disease by obstructing the urinary passages—the bladder and urethra. In such cases the symptoms of a secondary pyelitis and cystitis are often added.

Perforations in other directions are much rarer. The rupture of a renal echinococcus into the lungs has sometimes been observed, the patient coughing up echinococcus cysts.

Sometimes, especially after injuries, the sac of the echinococcus inflames, suppurates, and thus leads to a general pyæmic condition.

The diagnosis of renal echinococcus is possible only when a tumor can be made out belonging to the kidney, and when portions of echinococcus are passed with the urine, or through an exploratory puncture. They are most frequently confounded with hydronephrosis (*vide infra*), and, in women, with ovarian tumors.

The prognosis is not wholly unfavorable. Permanent recovery has been repeatedly observed, especially after the rupture, or single or repeated evacuations of the sac of the echinococcus; but, of course, echinococcus of the kidney may also be attended with numerous dangers, such as suppuration of the sac. The whole course of the disease is always very tedious.

A radical treatment is possible only by surgical means. Symptomatically, ice and local blood-letting are used when there are symptoms of local inflammation; and morphine, warm baths, and sometimes mechanical aids, like the catheter, when there are symptoms of colic.

2. **Distoma Hæmatobium** (see Fig. 113) is a parasite, occurring frequently in Egypt and Abyssinia; it is one of the fluke-worms (*hematodes*), and has its seat in the branches of the portal vein, the splenic vein, the vesical plexus, etc., and is nourished by the blood. Its eggs are often deposited in great numbers in the mucous membrane of the pelvis of the kidney, the ureters, and the bladder, and

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* In regard to the general natural history of the echinococcus, compare page 494.

there cause very intense inflammation, ulceration with subsequent stricture, deposit of concretions, etc. Many cases of the so-called tropical hæmaturia are caused by the distoma. The diagnosis of the disease can be made with certainty by finding the eggs in the urine.

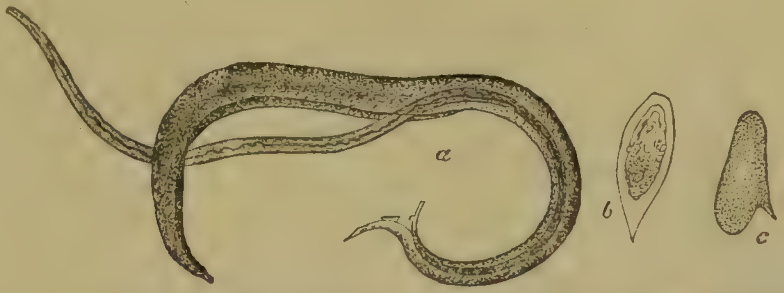

FIG. 113.—Distoma hæmatobium (from LEUCKART). *a.* Male and female, the latter in the canalis gynæcophorus of the former. Ten diameters. *b.* Egg with a terminal spine. *c.* Egg with a lateral spine. 150 diameters.

3. **Strongylus or Eustrongylus Gigas** (*palisade worm*) is a parasite occurring in the pelvis of the kidney in many animals—the dog, the wolf, the marten, and very rarely in man. In size and color it is not unlike an ordinary earth-worm. It may produce symptoms of severe pyelitis, with hæmorrhages, and colicky pains.

4. **Filaria Sanguinis Chyluria.**—The blood filaria of man, belonging to the round-worms, has obtained a special clinical interest, since it is recognized, from the investigations of Wucherer in Bahia in 1868, and of Lewis in the East Indies in 1870, as the cause of the tropical chyluria and some allied diseases, such as lymph scrotum, elephantiasis Arabum, and chylous ascites.

The full-grown filaria, "*filaria Bancrofti*," a very thin worm, about three or four inches long, has been found only a few times in man. Its seat is in the larger lymphatics, where it gives rise to chronic stasis of the lymph with its consequences—chronic hyperplasia of the connective tissue, etc. In the affection which here especially interests us, chyluria, the parasites are probably situated in the main branches of the thoracic duct—at any rate, in such a place that a stasis of the lymph ensues in the lymphatics of the bladder, or perhaps, in some cases, of the pelvis of the kidney and the other urinary passages. If the distended lymph-sac ruptures, the lymph or chyle is poured out into the urinary passages and is evacuated with the urine. Since this process may be often repeated, the intermittent course of chyluria is thus explained. The individual attacks of the disease may come on during years at intervals of weeks or months. They are often associated with pain and febrile symptoms.

The condition of the urine, which in many cases may look almost exactly like milk, is most characteristic. A creamy layer of fat forms upon the surface. If we shake the urine with ether, the greater part of the fat can be removed, and the urine rendered clear. The fat in the urine may amount to two or three per cent. The chyluria is often associated with a hæmaturia coming from the ruptured veins. The urine then looks bloody red, and shows under the microscope many red blood-corpuscles beside the fat-drops. Large clots often form in the urine.

The embryos of filaria, found in the urine in very many cases, although not in all, form the most important diagnostic feature in the urine. These (see Fig. 114) are objects two to three tenths of a millimetre long, with a diameter about equal to that of a red blood-corpuscle. They are usually imbedded in a very delicate sheath, which often projects at the end of the animal, and show a constant, vigorously vibrating motion. They have also been found in the blood of the patient, as well as in the urine, and, strange to say, especially during the night.

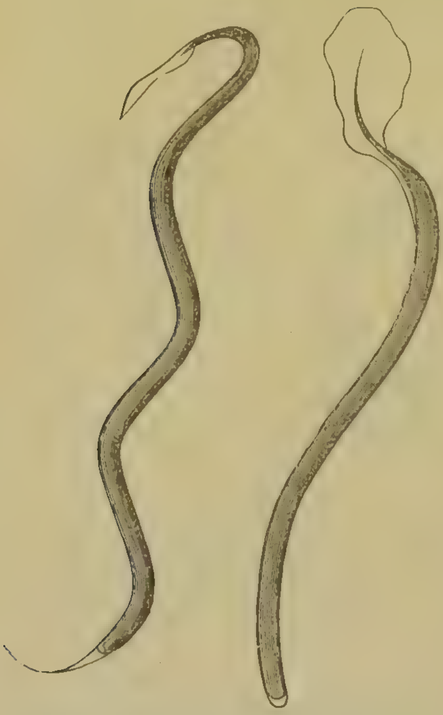

FIG. 114.—(From SCHEUBE.) Embryos of filaria.

The course of the filaria disease may vary considerably. Many patients reach an advanced age; in others, severe general symptoms, like anæmia and emaciation, finally come on. The different forms in which the disease occurs — chyluria, elephantiasis, etc.—are combined in manifold ways.

The region of the geographical distribution of the disease lies almost wholly in hot countries. It has so far been most frequently observed in Brazil, the Antilles, the East Indies, China, Japan, Egypt, Cape Colony, and Australia. Nothing definite is yet known of the precise mode of invasion of the parasites. According to Manson's investigations, mosquitoes play an important part here.

In regard to treatment, apart from any surgical interference, we may try picronitrate of potassium, three to ten grains (grm. 0·2–0·5), in pills or capsules, several times a day (Scheube).

CHAPTER X.

MOVABLE KIDNEY (FLOATING KIDNEY, REN MOBILIS).

Ætiology.—Although under normal conditions the kidney is fixed firmly in its position by its fatty capsule, over which the peritoneum is tightly drawn, and by the diaphragm, there is not infrequently a pathological condition, in which the kidney shows quite a high degree of displacement and mobility. The causes of this anomaly are not always quite clear, and there are probably different factors which may give rise to movable kidney.

First, perhaps, a congenital predisposition is often to be considered; this may be based chiefly upon an abnormally loose character of the tissue surrounding the kidney, and also upon an abnormal length of the renal artery; but in later life it is chiefly the factors that lead to a marked distention and laxity of the abdominal cavity, which further the development of a movable kidney. Frequent pregnancies are especially unfavorable in this respect, which explains the fact that floating kidney is very much commoner in women than in men. Severe and persistent bodily labor may also give rise to floating kidney; in some cases, too, injuries which affect the abdomen, and especially the region of the kidney, also play a part. Too tight lacing, and the wearing of corsets, have been repeatedly accused of gradually causing a mobility of the kidney. Finally, a loss in the general nutrition, by which the fatty capsule of the kidney undergoes a reduction, sometimes seems to favor the occurrence of movable kidney.

We have just mentioned the frequent occurrence of floating kidney in women but some cases have also been observed in men, and even in children. It is remarkable that the right kidney shows the anomaly in question much oftener than the left, which is possibly connected with the fact that the right kidney may be more easily displaced by the respiratory movements of the liver. Litten has lately pointed out that we often find the right kidney dislocated upward in simple gastrectasia, arising from immoderate distention of the stomach, relaxation of the muscular coat of the stomach as a result of chronic gastric catarrh, etc. In such cases the distended stomach and sometimes the duodenum push up the liver, and with it the right kidney. Movable kidney can be confirmed in the cadaver only when the kidney is found in an abnormal position—for instance, in front of the vertebral column.

Clinical Symptoms are not necessarily present in every case of movable kidney. If we examine old women with special regard to this point, we not very rarely find movable kidneys without any symptoms caused by them. In other cases, however, the floating kidney gives rise to a whole set of symptoms which are inexplicable except by the discovery of their cause. Drawing and pressing pains in the abdomen are the most frequent; these may shoot into the epigastrium or into the sacral and lumbar region, and may sometimes assume almost a colicky character. They are also frequently associated with nausea and even vomiting. All these disagreeable sensations usually increase still more as the patient moves, in walking or riding, while they are slightest or even disappear entirely when the patient lies down.

In many cases of floating kidney more severe attacks come on periodically, often at the time of the menses; they have been termed by Dietl "incarceration symptoms." They consist of the sudden onset of pain, chills which may increase almost to a rigor, vomiting, and symptoms of general collapse. Diuresis is usually diminished during this period, and increases only when the attack ceases—that is, three or four days later. The special cause of these symptoms is to be found partly in circumscribed inflammatory changes in the vicinity of the kidney, but chiefly in a sudden urinary stasis from bending or twisting the ureter. There then arises an acute hydronephrosis whose attendant symptoms disappear when micturition has again become possible. In some cases floating kidney seems to cause persistent hydronephrosis, with secondary pyelitis.

We find quite frequently in women with floating kidney a line of general nervous "hysterical" symptoms—headache, backache, mental irritability, paræsthesiæ, etc. It is often hard to decide whether these symptoms are due to floating kidney or are only co-ordinate with it. They often arise, at any rate, only as a result of the mental condition, for the mere idea of possessing a "floating kidney" may be enough in nervously disposed women to provoke a host of subjective sensations. Hence we must be cautious in informing the patient of the diagnosis.

Movable kidney also seems able sometimes to produce certain resultant conditions by pressure on neighboring organs. Thus Bartels has claimed that many cases of dilatation of the stomach are due to a compression of the descending part of the duodenum by the movable kidney. In an analogous way, jaundice may arise from pressure on the gall-ducts, constipation from pressure on the colon, œdema of the legs from pressure on the inferior vena cava, and finally it has been stated that floating kidney in women may be the cause of abortion. All these accidents, at any rate, are only rarely to be considered.

The **diagnosis** of floating kidney can be made only by its objective discovery. It is rather a theoretical assumption, which is only rarely of value in practice, that we can recognize the displacement of the kidney from its normal position, by the sinking in of the affected lumbar region (almost always the right), and by the abnormal clearness of the percussion-note there. The only certain proof is feeling the kidney as a movable tumor of the proper size and shape beneath the edge of the right ribs or toward the umbilicus or the inguinal region. Occasionally we can succeed in feeling the pulsation of the renal artery. Palpation of the movable kidney, however, is not always equally easy, and generally demands a certain amount of practice. Bimanual palpation and counterpressure in the lumbar region with the left hand is very advisable. If we can reach the kidney with the fingers, we can push it about and often bring it back into its proper place.

In general, the diagnosis is not very difficult in patients whose abdominal walls are lax, if the attention be directed to the possibility of floating kidney. Of course a positive result is alone decisive, while a negative result, or finding it only once, proves nothing. In many cases, of course, floating kidney is confused with other sorts of tumors, with pedunculated ovarian cysts, fæcal tumors, enlargements of the gall-bladder, or echinococci.

The prognosis is so far favorable that the patient's life is never threatened by an uncomplicated floating kidney. The symptoms, are of course, very obstinate, and may resist any therapeutic attempts for years. In advanced age they often cease of themselves.

Treatment.—If there are severe disturbances or "incarceration symptoms" on the part of a floating kidney, a quiet dorsal decubitus is to be prescribed; and if the kidney does not return to its proper place of itself, we should try artificial reposition, which can be performed in many cases, and which is then attended with success. If we can not relieve the symptoms in this way, prolonged warm baths, warm poultices, and opium must be used as indicated. Various bandages provided with pads and supports are recommended to guard against a new displacement of the kidney; these, of course, sometimes do good service, but they are often useless. At all events, they should be tried. The hope of "removing the laxity of tissue" by a "tonic treatment," iron preparations, cold rubbing, etc., must be illusory; nevertheless, these remedies are very much recommended in practice, since they often act very favorably, especially on the general nervous symptoms, as mental remedies. Certain general hygienic measures, especially the avoidance of any great bodily movements, and care for easy dejections, are also of service.

In cases with very severe, distressing symptoms, surgeons who are fond of operations have repeatedly stitched the kidney to the parietes, sometimes with success, sometimes, too, without the desired result. At any rate, the indication for operation is furnished only when the symptoms are very marked, and when all other remedies have been unsuccessful.

APPENDIX.

THE DISEASES OF THE SUPRA-RENAL CAPSULES AND ADDISON'S DISEASE (BRONZED SKIN).

In the year 1855 the English physician Addison published for the first time a list of cases in which, besides the symptoms of a general bodily weakness and anæmia, a peculiar dark pigmentation of the skin had gradually developed. Since disease of the supra-renal capsule was found at the autopsy in all cases, Addison concluded that this was the immediate cause of the bronze coloring of the skin. Observations similar to Addison's were soon made in greater numbers, so that the fact itself can not be doubted; but even at present nothing definite is known as to the special cause of the disease or the theory of this remarkable connection between disease of the supra-renal capsules and pigmentation of the skin.

Attempts have been made in different quarters to obtain an explanation from experiments on animals; but up to the present time these attempts have remained quite fruitless, and even lately Nothnagel, in spite of having destroyed both supra-renal capsules in a large number of rabbits, has not succeeded a single time with certainty in provoking the symptoms of the disease artificially. The anatomical lesions in man are up to the present time not calculated to add clearness to the case, since they seem to contradict one another in various points. In the first place, some observations must be mentioned in which the supra-renal capsules were found perfectly normal in spite of an existing pigmentation of the skin. Such cases, however, prove little, since it of course can not be put in question that a staining of the skin may sometimes develop from some other reason besides disease of the supra-renal capsules. On the other hand, it has been asserted that extensive changes are sometimes found in the supra-renal capsules on autopsy without the existence of the symptoms of bronzed skin during the patient's life; but these cases are also open to the objection that the disease has perhaps not been extensive and intense enough to cause the bronze coloring of the skin. The contradictions just mentioned, however, have led to many other attempts at an explanation of Addison's disease, of which one especially deserves consideration. According to this, the symptoms met with are produced not by the disease of the supra-renal capsules themselves but by the invasion of the solar plexus and the semilunar ganglia of the sympathetic by the morbid process (Risel, Burger, and others). According to this theory, the symptoms of Addison's disease may arise when the aforesaid nervous parts are diseased independently, or by the extension of a pathological process from some other neighboring organ. A number of anatomical lesions are cited to favor this view, but it is not certainly proven, and the internal connection between the symptoms is by no means clear by this hypothesis.

Pathological Anatomy and Ætiology of Addison's Disease.—Addison himself has pointed with emphasis to the fact that the special form of disease in the supra-renal capsules is by no means always the same. At any rate, the disease named from him is not to be regarded as a definite anatomical affection, but rather as a particular group of symptoms. By far the most frequently it is tuberculosis of the supra-renal capsules which lies at the bottom of Addison's disease. The capsules then are either enlarged and studded with caseous tubercular new growths, or they are in part cicatricially contracted. Other tubercular affections are almost always present in the body at the same time, especially caseation of the mesenteric lymph-glands and pulmonary tuberculosis. Other morbid processes besides tuberculosis may also be found in the supra-renal capsules: simple chronic inflammations, enlargement of the organ (which Virchow has called "struma of the supra-renal capsules"), hæmorrhages, new growths such as cancer, and even echinococci. It is self-evident, from what has been said above, that in every case we must consider some implication of the neighboring sympathetic ganglia, by compression, cicatricial contraction, or chronic inflammation. Both supra-renal capsules are almost always diseased at the same time, rarely only one is affected.

Of the lesions in other organs we must also mention that Peyer's patches and the solitary follicles of the intestine are as a rule swollen. The spleen is somewhat enlarged in some cases, but not in others. There is no striking pigmentation of the internal organs. The changes in the skin and in certain mucous membranes will be mentioned below.

Considering the variety of the anatomical causes there can be no question of a uniform ætiology of the disease. Among the causal factors, those most frequently reported are defective nutrition, care and anxiety, and finally traumatic action on the abdomen. The majority of cases are met with in the male sex and in middle life. Addison's disease, however, must be regarded as a rare affection, of which but few cases are observed even in the larger cliniques.

Symptomatology.—The purest type of Addison's disease appears in those cases where the symptoms are apparently primary in their development, and do not come on in the course of some other disease, such as phthisis or cancer.

The first symptoms of the disease are usually of a general nature, and are referable to a gradually increasing anæmia and to general weakness and physical lassitude. The anæmia shows itself objectively through the pallor of the skin and the diminution in the number of red blood-corpuscles, but without other definite anomalies of the blood that can be made out. A number of symptoms appear besides, which are due secondarily to the anæmia, especially to the anæmia of the brain. Among them are mental lassitude and loss of energy, frequent headaches, attacks of vertigo and faintness, and tinnitus aurium. The patient's general nutrition often suffers very considerably; but it must be added that in Addison's disease, as in other anæmias, the fatty layer, especially over the abdomen, often remains remarkably well developed.

Besides the anæmic symptoms, there frequently are disturbances of the stomach. The appetite is poor, and there is very often vomiting. The latter may sometimes be almost unrestrainable, and then it is one of the most distressing symptoms of the disease. It is usually due not to an anatomical change in the stomach, but probably to the anæmia of the brain, or to other nervous influences. Cardialgic symptoms are also frequent. The bowels are sluggish as a rule, but there is sometimes diarrhœa. We sometimes hear functional murmurs in the heart, but as a rule its sounds are low and pure. The pulse is usually moderately accelerated. The liver and spleen present no constant changes. Albuminuria is only exceptionally found, and is due to complications, such as amyloid kidney.

The special characteristic symptom, which alone renders the diagnosis possible, is the gradual onset of a peculiar pigmentation of the skin. This usually shows itself first in the face and on the backs of the hands, and also in those parts which normally present a greater pigmentation (the areola of the nipples, the axillæ, and the genitals), or which are exposed to greater pressure by the clothing, as the hips and shoulders. It is especially noteworthy that dark pigmented spots usually develop on the mucous membrane of the lips and mouth. The intensity of the coloring differs in different cases. It usually increases as the general condition grows worse. In the most intense cases the whole skin may become dark brown or black, like that of a mulatto or negro. Sometimes, however, the pigmentation remains limited to separate large or small spots, and in other parts of the skin there may then be even a marked loss of pigment. The nails and the sclera always remain white, and frequently the palms of the hands and the soles of the feet also. The pigmentation of the skin usually increases during the whole disease; only exceptionally does the skin become light again in the later stages.

The special cause of the accumulation of pigment in the skin is wholly unknown. We find on microscopic examination of the skin that the pigment lies not only in the cells of the rete Malpighii, but also in the corium, especially along its blood-vessels. It is probably formed from the blood-pigment, and is carried by wandering cells from the cutis into the epithelial layers of the skin (Demiéville, Nothnagel).

The course of Addison's disease is almost always chronic, and may last for years, but cases have been described with a more acute course. The disease sometimes begins with violent initial febrile symptoms, vomiting and diarrhœa. The disease then has a comparatively rapid termination after a few months, or a second chronic stage may follow the first acute one.

The final termination of Addison's disease is always unfavorable. Temporary remissions are often observed, but the disease always becomes worse again after them. Death usually ensues gradually amid the signs of increasing general uræmia and weakness. In some cases severe nervous symptoms also come on toward the end of the disease—coma, delirium, or epileptiform attacks. Such conditions, which, according to Jaksch, may depend upon acetonuria, may sometimes develop quite rapidly and unexpectedly.

Treatment.—There can be no question of a specific treatment of Addison's disease, especially considering the differences in the primary diseases and in the complications. Tonic remedies, strengthening diet, iron, quinine, and arsenic, are most indicated, although iodide and bromide of potassium, electricity, etc., have been tried by some physicians. Otherwise the treatment is purely symptomatic; the vomiting, diarrhœa, and nervous attacks demand special measures. We know by experience that great caution is to be used in prescribing cathartics, since patients have repeatedly been observed to grow considerably worse after them.

SECTION II.

Diseases of the Pelvis of the Kidney and of the Bladder.

CHAPTER I.

INFLAMMATION OF THE PELVIS OF THE KIDNEY. PYELITIS.

Ætiology.—Isolated primary pyelitis hardly ever occurs as an independent disease. Pyelitis is rather in most cases either a complication or a result of other diseases, and in such cases often claims but little clinical interest.

We sometimes find a rather moderate pyelitis in the bodies of persons who have died of severe general infectious diseases, typhoid fever, smallpox diphtheria, or pyæmia. The affection depends, in all probability, upon the elimination by the kidneys of substances that excite inflammation, and is thus to be regarded as analogous to the renal changes which often coexist. Toxic substances, such as cantharides and copaiba, which pass through the kidneys, may also cause pyelitis as well as other disturbances.

Pyelitis very often arises from a direct extension of inflammation from the neighboring organs. In many cases of acute and chronic nephritis the pelvis of the kidney takes part in the inflammation to a greater or less degree; but an ascending extension of the inflammation from primary diseases of the urethra or bladder is still more common. Any urethritis or cystitis may, if it lasts long, advance upward to the ureters and the pelvis of the kidney, so that in severe cases we often find an inflammation of the whole urinary tract, a pyelo-cystitis, and even a "ureteritis." We have already stated (compare page 868) that the inflammation may extend still farther to the kidneys themselves, and this extension will be repeatedly spoken of.

Another frequent cause of pyelitis is the presence of foreign bodies in the pelvis of the kidney, which act as direct mechanical irritants. Among these are, in the first place, renal calculi (*vide infra*), and also retained coagula, parasites (see page 874), etc. The pyelitis arising as a result of retention of urine in the pelvis of the kidney (*vide infra*, hydronephrosis), does not belong here directly, since it first develops from a decomposition of the urine.

Whether there is a primary independent pyelitis, arising in ways other than those so far mentioned, is doubtful, as we have said. The occurrence of a primary pyelitis after exposure to cold especially lacks confirmation. The pyelitis coming on in women in childbed, or following different sorts of diseases of the sexual organs, may in all cases be referred to an infection of the pelvic mucous membrane from the bladder or from the kidneys.

Pathological Anatomy.—In simple catarrhal inflammation the mucous membrane of the pelvis of the kidney is reddened, swollen, and covered with an abundant secretion, which contains varying amounts of pus-corpuscles and epithelium. In severer inflammations we often find quite numerous little hæmorrhages in the mucous membrane, and sometimes little gray nodules, which correspond to the swollen lymph-follicles.

In severe cases, which are seen almost solely as a complication of a more extensive affection of the urinary passages, such as pyelocystitis, we have a purulent, ulcerative inflammation, which may even assume a diphtheritic character. In these cases the kidneys are almost always coincidently involved—pyelonephritis. If the nephritic abscesses break into the pelvis of the kidney, there arises an ulcerative destruction of the renal tissue, so that the pelvis of the kidney is filled with pus and bounded by extensive ulcers, which often penetrate deeply into the substance of the kidney—pyonephrosis. The usually striated, pyelonephritic abscesses reaching to the surface of the kidney have already been described in the previous section (see page 869), where their bacterial origin has also been mentioned.

The condition differs when the kidney is involved, as in many cases of chronic pyelitis. This appears most frequently as a result of retention of urine, and hence it is usually associated with a dilatation of the pelvis of the kidney. In these cases we sometimes find pronounced processes of contraction in the kidneys—that is, a partial atrophy of the renal tissue, increase of the interstitial connective tissue, and evident cicatricial depressions on the surface—in a word, a secondary contracted kidney, arising as a result of pyelitis, which differs from genuine contraction of the kidney only in its ætiology.

Clinical Symptoms.—Since in most cases pyelitis develops only as one symptom of a more extensive morbid process, its clinical symptoms are usually but slightly prominent in the whole course of the disease. In what follows, therefore, we can not give any complete description of the clinical course of pyelitis, but we must mention only those symptoms from which, when there is an affection of the urinary passages, we can conclude that the pelvis of the kidney takes part in the morbid process.

The most essential sign which the urine presents in all inflammatory affections of the urinary passages, the presence of mucus and pus, will be described more fully in the chapter on cystitis (*vide infra*). In pyelitis, also, the muco-purulent secretion of the pelvic mucous membrane must mix with the urine, and in every severe purulent inflammation the amount of pus in the urine must be quite considerable. We can never decide with certainty, from the mere presence of pus in the urine, as to the place where the pus mixes with the urine, whether in the pelvis of the kidney or in the bladder, or even in the urethra. Only when the urine contains other characteristic morphological elements besides the pus-corpuscles can we decide upon the portion of the urinary tract which must be especially affected. Among these morphological constituents is, first of all, the epithelium, which in the pelvis of the kidney shows in part another shape than in the bladder. If we find in the urine, then, the triangular, long caudate pelvic epithelium (see Fig. 115), with its cells sometimes laid over one another in the form of tiles, we may assume that the pelvis of the kidney is involved in the inflammation. Of course, the reverse of this law does not obtain; for, even in severe purulent pyelitis and pyelonephritis, we often fail entirely to find the caudate epithelium in the urine. We must also add that similar forms of epithelium are also found in the bladder, so that great caution is always to be enjoined in estimating the diagnostic value of the epithelium found. Blood is only rarely found in the urine in simple pyelitis, but it is common in pyelitis calculosa (see the following chapter). The reaction of the urine in pyelitis is usually acid, but it is not correct to assume that there is in this a comprehensive mark of distinction between pyelitis and cystitis, in which the urine is often alkaline.

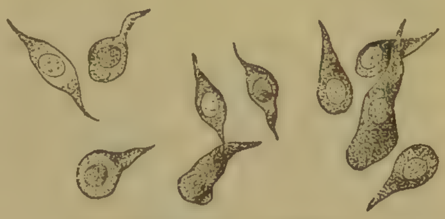
FIG. 115.—Pelvic epithelium.

Another symptom to be referred directly to the pyelitis, is the local pain in the region of the kidney, which sometimes passes from this point down along the ureters to the bladder. This symptom has not, therefore, a great diagnostic value, because only its presence is in favor of pyelitis, while its absence proves nothing against the existence of the disease.

All the other symptoms may be directly dependent upon the pyelitis, but they may usually be referred in great degree to the other co-existing affections. First among these is fever, which either shows an irregularly remitting course, or appears in single high elevations of temperature, usually associated with rigors. The fever, however, seldom shows this latter pyæmic character except in the severe purulent forms, where we usually also have the formation of renal abscesses—that is, a pyelo-nephritis. Besides the fever there are often in severe cases, general nervous symptoms, such as headache, delirium, and sopor, which are to be referred partly to the general pyæmic infection of the body and partly, perhaps, to the absorption of ammonia from the urine decomposing in the blood—the "ammoniæmia" of Treitz and Jaksch.

The whole course of pyelitis differs so much according to the primary disease present that nothing of general application can be said about it. The milder forms, which often pass off rapidly, are found most commonly in childbed, and sometimes in acute infectious diseases, poisonings, and as a result of mild cystitis. The severe forms occur chiefly as cysto-pyelitis and pyelo-nephritis, as a result of strictures of the urinary tract (*vide infra*), of severe cystitis in diseases of the spinal cord, and in other severe diseases of the kidney and of the pelvis of the kidney, such as new growths, and parasites. They usually form then a very tedious and often incurable affection, which lasts until the patient's death.

The signs of pyelitis which are important in diagnosis have already been mentioned above. The main point is always to pay careful attention to the ætiology of the case, and, next to that, to the changes in the urine. If the whole condition points to a severe affection of the urinary tract, we can often decide correctly on a pyelitis or a pyelo-nephritis without the presence of direct signs of these diseases, because we know by experience that such an extension of the affection is the rule in all severe and long-continued cases.

The implication of the kidneys is sometimes shown directly by the presence of casts in the urine in addition to the pus-corpuscles. In the cases above mentioned, where a chronic cysto-pyelitis is complicated with a contracted kidney, the condition of the urine is the same in many respects as in genuine contracted kidney.

It is abundant, usually has a low specific gravity, and contains, besides the pus-corpuscles, a few short hyaline casts.

Treatment.—The treatment of pyelitis coincides mainly with the treatment of the primary disease, and therefore it needs no detailed description here. Ordinarily only the accompanying cystitis (*vide infra*) is accessible to a direct local treatment, and here an important prophylactic factor is discovered, since by a timely treatment of the cystitis we can certainly hinder the advance of the inflammation to the pelvis of the kidney.

Among the internal remedies to which we ascribe a favorable influence on the mucous membrane of the urinary tract, and which are therefore used in like manner both in pyelitis and in cystitis, we may mention the astringents, tannin, alum, and acetate of lead. Balsams, such as copaiba, are sometimes prescribed, and also antiseptic substances, such as salicylic acid and chlorate of potassium. The details in regard to all these remedies will be found in the treatment of cystitis. The copious ingestion of fluids sometimes acts favorably, especially the use of certain mineral waters, among which the waters of Carlsbad, Vichy, Ems, Neuenahr, and Wildungen have obtained the most reputation. A methodical milk cure is also greatly to be recommended, especially when there are symptoms of irritation, such as pain on micturition.

Local applications to the region of the kidneys, warm poultices, or exceptionally local bloodletting, are indicated only when there is severe pain, where, of course, narcotics must also be used under some circumstances. In this respect warm baths also do good service at times.

CHAPTER II.

NEPHROLITHIASIS.

(*Renal Calculus. Renal Gravel. Pyelitis Calculosa.*)

Occurrence, Chemical Composition, and Ætiology of Renal Concretions.—The precipitated concretions of the urinary constituents which form in the pelvis of the kidney, and which, under some circumstances, may be passed from it with the urine, are designated, according to their size and nature, as renal sand, a fine, pulverized precipitate; renal gravel, gravel-like, granular concretions about the size of the ordinary coarse grains of sand, which can usually pass through the ureters without special difficulty; or renal calculi, the larger concretions. The latter are about the size of a pea or bean, but larger stones are occasionally seen which may even resemble actual casts of the pelvis of the kidney. We usually find a calculus only in one kidney, although both kidneys may be affected.

In regard to the chemical nature of renal concretions, they consist most frequently of uric acid. They then have a brown-red or blackish color, and have a crystalline breakage, which in large stones is usually plainly laminated; and, on the whole, a smooth although irregularly-shaped surface. More rarely the renal concretions consist of calcic oxalate. The oxalate calculi are extremely hard, have a dark-brown color and a rough surface, often furnished with many prickles, from which reason they are often called "mulberry calculi." Their breakage sometimes has a radiated but never a laminated arrangement. Stones are also frequently seen, which consist of alternating layers of uric acid and calcic oxalate, or which have a nucleus of uric acid and a coating of calcic oxalate. The phosphatic calculi are another variety of renal concretions. We only very rarely have to do, however, with stones which consist exclusively of basic calcic phosphate or ammonio-magnesic phosphate, but we usually have secondary deposits of layers of phosphate which are precipitated on uric acid or mulberry calculi in urine which has become alkaline. The phosphatic calculi have a grayish-white color, and are comparatively soft. The largest examples of them are found, not in the pelvis of the kidney, but in the bladder. The cystine and xanthine calculi are the rarest of all.

But little is definitely known as to the exact causes for the origin of all these concretions. We must suppose for the deposition of uric acid an abnormally great acidity of the urine, but we are not in a position to state by what circumstances (the patient's food and manner of life, acid fermentation of the urine within the urinary tract?) this may be provoked. It is a very probable theory that some solid body usually affords the nucleus and the first cause for the formation of at least the larger stones, among these bodies being mucous coagula, shreds of epithelium, and perhaps bacteria. The oxalate, cystine, and xanthine calculi are also deposited from acid urine, but scarcely anything is known as to the precise conditions to be here considered. We can point only to the fact that with the close chemical alliance between uric acid and oxalic acid the origin of the latter from uric acid seems possible, and the frequent co-existence of the two substances in the calculi therefore seems plausible. We have already indicated above that the cause of the deposition of phosphatic concretions can be found only in the existence of an alkaline reaction in the urine.

In regard to the predisposing causes of calculus formation we must mention, first of all, that they are often found in children, and next in frequency in advanced life. Men show a greater disposition to renal calculi than women. Heredity also seems to play a certain part, since the disease has been repeatedly observed in different members of the same family. The many relations which have been imagined between the formation of calculi and certain conditions in the manner of life and in the food taken, all lack accurate foundation. In regard to this the chief blame is laid upon an excessive meat-diet, drinking copiously of new sour wines, and drinking water containing lime. In regard to the occurrence of uric-acid concretions in gouty patients, compare the chapter on arthritis uratica.

The Anatomical Changes caused by Renal Calculi.—The most frequent change which the presence of concretions in the pelvis of the kidney excites is pyelitis. This may exhibit all degrees, from a simple catarrhal inflammation to a diphtheritic or severe purulent inflammation of the pelvic mucous membrane. As a result of the mechanical irritation, there are quite frequently large or small hæmorrhages.

If a severe purulent pyelitis has developed, this may bring with it all the sequelæ with which we have previously become acquainted. In severe cases the process may involve the kidneys, and there arises a pyelo-nephritis, with a purulent breaking down of the renal tissue, and, under some circumstances, even a perinephritis, with extensive suppuration into the vicinity of the kidney, and with occasionally perforation into the neighboring organs. If the renal calculi have previously passed outward, they are not found at the autopsy, although they form the special starting point of the disease. Sometimes, however, the pus-cavity is entirely filled with calculi.

A second important sequel of a renal calculus which sometimes develops is hydronephrosis (*vide infra*). It arises when a large stone blocks the passage from the pelvis of the kidney into the ureter, or when a smaller stone remains fast in the ureter and completely shuts off the passage of the urine. In the latter case there may also arise a pressure necrosis and perforation of the ureter. It goes without saying that inflammation and hydronephrosis or pyonephrosis may be combined with each other.

Clinical Symptoms.—If there is merely the formation of renal sand or renal gravel in the urinary tract, this condition is sometimes associated with no symptoms at all. The little granules are washed away by the urine and evacuated, and at most they may give rise to slight pain in the region of the kidney. Larger stones, however, may sometimes be wholly, or almost wholly, without symptoms, if from their position and their smooth surface they can cause no special bad results.

The characteristic clinical symptoms of nephrolithiasis do not appear until the results of mechanical irritation of the pelvis of the kidney arise, or until there is an incarceration of a calculus in the ureter. It is the latter circumstance which, after the analogy of gall-stones, causes the most important symptom in the diagnosis of renal calculi—the pains, the so-called renal colic. Such an attack of colic sometimes comes on quite suddenly and unexpectedly; in other cases it is produced by some exciting cause—jumping, running, walking, or riding. The pain often attains an uncommonly severe intensity; it shoots from the lumbar region along the course of the ureters, but sometimes extends still farther, to the testicles, the thighs, or the back. In severe attacks there may be a general state of collapse with a small rapid pulse, cold sweat, and attacks of fainting. The temperature may be somewhat raised. We often see nausea and repeated vomiting. The urine is sometimes entirely normal, inasmuch as it comes exclusively from the other free kidney; but oliguria, or even complete anuria, with its consequences, invariably sets in if both ureters be stopped. Even when one kidney remains normal, the evacuation of urine may be inhibited by a reflex spasm of the bladder. Sometimes the urine passed contains pus and blood. The duration of renal colic depends upon the duration of the incarceration; it may last for a few hours or several days. The attack often ends with the passage of the stone outward into the bladder.

The other symptoms occurring in nephrolithiasis refer mainly to the results of the mechanical irritation of the pelvis of the kidney. The urine then shows an admixture of pus, and contains pelvic epithelium and often blood. The frequent appearance of blood in the urine, which usually has its cause in purely mechanical lesions of the mucous membrane, is a characteristic symptom of pyelitis calculosa. If we find, as sometimes happens, the urine at many times perfectly clear and normal, but at other times containing pus, we may suspect an occasional blocking of the ureter coming from the diseased kidney by a renal calculus.

The symptoms are much more severe if the trouble goes on to a severe purulent pyelitis and pyelo-nephritis. We need not describe the details again here—the pain, fever, swelling, and perforation internally or externally—since they agree completely with what has been said before (see the previous chapter and Chapter VI in the previous section). A special chapter is devoted below to the symptomatology of hydronephrosis.

The whole course of nephrolithiasis is, as a rule, very tedious. Since the disposition to the formation of calculi usually persists, and since also the sequelæ which have once developed may last for a long time, a very chronic state often develops, which, in varying ways and with manifold exacerbations and remissions, is composed of attacks of colic, hæmorrhages, and symptoms of pyelocystitis.

In many cases, of course, complete recovery may finally ensue. The calculi present are passed, new ones are not formed, the pyelitis that has arisen disappears, and, of course, all the morbid symptoms cease; but, on the other hand, nephrolithiasis has also a number of dangers in itself, which threaten life very seriously.

These are, besides the rare occurrence of uræmia, first of all the development of pyelo-nephritis and of still more extensive suppurations, with a general decline in strength, pyæmic states, etc. There is also a possible danger in such chronic suppurations that there may be the appearance of a general amyloid degeneration of the internal organs.

Among the complications occurring in other organs, only one circumstance has especial interest: that gall-stones and renal calculi are found quite frequently in the same individual. We, of course, can not well speak of a complication with vesical calculi, since a great part at least of the vesical calculi are originally formed in the pelvis of the kidney, and undergo merely a further growth in the bladder.

Diagnosis.—The diagnosis is made perfectly certain only by finding the special *corpora delicti* in the urine. For this purpose the urine must always be examined as soon as possible after its passage, and it is best to pour it through a fine sieve. In many cases, however, we can diagnosticate nephrolithiasis quite certainly, without the direct evidence of concretions, from the characteristic symptoms, especially from the periodical renal hæmorrhages and from the attacks of colic. Of course it may be confused with renal cancer, parasites of the kidney such as echinococci, and similar affections, but this does not happen very often, because renal calculus is a far commoner affection than the diseases just mentioned. Finally, we must state that these diseases may comparatively often be combined with nephrolithiasis.

Treatment.—Since the uric-acid concretions are by far the commonest, the methods of treatment most in use for nephrolithiasis refer first to these; but they are to be used in like manner in the allied oxalate calculi.

If the tendency to the formation of urinary gravel be confirmed in a patient, or if the severer symptoms of nephrolithiasis have already appeared, we must first give a number of general dietetic directions, which check the formation of uric acid in general, and aid the solubility of the uric acid already formed as far as possible. Without entering too much on theoretical reasoning, we will give in what follows the measures which have been proven practically and quite generally acknowledged. In the first place, the patient must avoid any immoderate indulgence in food in general, and particularly too great indulgence in meat. We should recommend for him a diet mainly, though not exclusively, vegetable, with a moderate supply of meat; milk is also a proper food. Alcoholic beverages should be taken only in slight amount, and acid foods and drinks, except lemonade, should not be taken at all. It is a good plan to control the supply of food by weighing the patient regularly, in order to avoid any further addition to the weight in all persons in a state of normal nutrition, and to obtain a loss of weight in the corpulent. We should also aid the using up of tissue by regular physical exercise and muscular work, gymnastics, sawing wood, or gardening; and, finally, the urine must be diluted by an abundant supply of fluid, and its soluble power be thus increased.

This latter indication will usually correspond at the same time with those which diminish the acid reaction of the urine by a supply of alkalies and thus hinder the deposition of uric acid as far as possible. From this comes the very extensive use of the alkaline mineral waters in nephrolithiasis. The simplest plan is to dissolve some alkaline salt in a large amount of plain water, soda water, or lemonade, and let the patient drink it. Among these salts we may mention sodic phosphate, one to four drachms (grm. 5–15) a day, or better, sodic carbonate, one to two drachms (grm. 5–10), or carbonate of lithium, which has lately been especially recommended, two to five grains (grm. 0·1–0·3) several times a day. Of course, the special mineral waters enjoy a greater reputation; these may be used in conformity with the treatment at home, or especially at the appropriate springs. The following springs enjoy the greatest reputation in this respect: Carlsbad, Vichy, Salzbrunn, Fachinger, Tarasp, Neuenahr, Ems, and Wildungen.

The symptomatic treatment is also very important. In so far as this relates to the accompanying pelvic and vesical catarrh, we may refer to the appropriate chapters in this book, while the surgical methods of treatment in the severer sequelæ—hydronephrosis, pyonephrosis, or perinephritic abscess—are to be found in the special treatises. Against renal hæmorrhages some internal remedies, such as ergotine or tannin, have been recommended, but their action is quite doubtful. The treatment of the attacks of colic is of greater practical significance. The chief remedies are the narcotics, opium, and morphine, internally, or, with very severe pain, better subcutaneously. Warm baths, warm poultices, or narcotic embrocations, such as chloroform liniment, also frequently give relief. Local blood-letting is only rarely indicated. An abundant supply of fluid is always advisable, in order to aid the washing out of the incarcerated stone by an increased secretion of urine.

As we have stated, what has thus far been said obtains chiefly in the treatment of uric-acid and oxalic-acid calculi. We know no special prescriptions to be considered for the occasional cystine calculi. When there are phosphatic calculi, however, which can be deposited only from alkaline urine, the use of acids has been recommended, especially of lactic acid, seven to fifteen grains (grm. 0·5–1), internally in an aqueous solution. The main thing, of course, is always the treatment of the catarrh of the urinary tract, which usually lies at the bottom of the calculus formation.

CHAPTER III.

TUBERCULOSIS OF THE GENITO-URINARY APPARATUS.

Ætiology and Pathological Anatomy.—It does not seem remarkable that, with the presence of many tubercular processes in the body, tubercle bacilli should quite easily reach the kidneys by way of the blood-current, and there give rise to an eruption of tubercle. Accordingly, we quite frequently find a few or many miliary tubercles in the kidneys in acute miliary tuberculosis, in pulmonary tuberculosis, etc., which are distributed over the whole kidney, or sometimes only in the territory of one arterial branch.

While miliary tuberculosis of the kidney, however, is without any clinical significance, there is also an extensive local tuberculosis of the kidney, as well as of the urinary tract and the sexual organs. Such affections sometimes occur as a result of pronounced pre-existing tuberculosis of other organs, especially the lungs, or they arise as an apparently independent disease, which is termed genito-urinary tuberculosis. In such cases the infection with the tubercular poison often seems to take place by means of the blood from some previously existing—perhaps concealed—tubercular foci in the body, such as glands, tubercular bone or joint disease, etc. In other cases, the tubercle bacilli perhaps enter the urinary tract from without, but the point of the first anatomical lesion need not, apparently, always be the same. The kidneys often seem to be first diseased, in other cases the bladder, and quite frequently, as it seems, the prostate, and sometimes perhaps the vesiculæ seminales or the testicles. From the organ first affected the process then extends continuously or by leaps to the neighboring parts. If the cases come to autopsy, the tuberculosis is often so extensive that we can no longer make out with certainty the place where it first began. In women the urinary apparatus is only very rarely affected by tuberculosis, while uterine and ovarian tuberculosis represents a localization of the tubercular poison of clinical importance (compare the chapter on tuberculosis of the lungs).

In the kidneys the tubercular infiltration develops either chiefly from the pelvis of the kidney or in the renal substance itself. Yellow cheesy nodules arise, which finally break down and thus lead to an actual "nephro-phthisis." If the disease arise from the pelvis, the infiltrated renal papillæ are usually first affected, from which the whole pelvis of the kidney is changed into an ulcerating surface covered with necrotic tissue and cheesy detritus. In very advanced cases almost the whole kidney is destroyed. The process is usually bilateral, but it is often more advanced on one side than on the other.

If the process invade the ureters, their walls are also infiltrated with tubercular deposits, and hence they are thickened, while the mucous membrane is often changed in great part to a necrotic ulcerating surface. Precisely analogous conditions are found in the bladder, and in some cases even in the urethra; while in the prostate, the vesiculæ seminales, and the testicles there is more frequently the formation of cheesy tubercular nodules, and more rarely disintegration and perforation.

Clinical Symptoms.—The picture of genito-urinary tuberculosis corresponds in most of its details completely to that of a severe chronic pyelo-cystitis. The occasional local symptoms are pain in the region of the kidneys and bladder. This may sometimes assume great severity, like colic, if the ureter become plugged by a broken-down, crumbling mass; yet in other cases the pain is but slight during the whole disease.

The urine shows the most important changes. It almost invariably contains an abundant sediment, consisting of pus-corpuscles and detritus. Its amount usually remains normal for a long time; its reaction is faintly acid, but in severe cases it may become alkaline through complication with an alkaline fermentation of the urine. The discovery of shreds of tissue in the urine, elastic fibers and connective tissue, is sometimes possible, and is of diagnostic value because it is direct evidence of an ulcerative process. The discovery of tubercle bacilli in the purulent urinary sediment (Rosenstein and others) is, however, far more important. This is performed by the same method as in the sputum; it succeeds in almost all cases, and is an infallible and absolutely decisive sign in diagnosis. Admixtures of blood in the urine are also seen in genito-urinary tuberculosis, but they may often be entirely absent. In one of our cases a slight hæmaturia was the first symptom which called the patient's attention to the trouble with the bladder.

The local objective examination of the kidneys usually gives a negative result. Only in a few cases have we been able to feel the diseased kidney as a tumor through the abdominal walls. This is usually due less to the tubercular infiltration of the kidney itself than to the dilatation of the pelvis of the kidney from hydronephrosis. We can sometimes feel the thickened walls of the bladder. The local examination of the prostate and the testicles is far more important in diagnosis. Especially in the latter we often feel the hardening corresponding to the tubercular infiltration, and manifesting itself chiefly in the epididymis, while the hardening and enlargement of the prostate can usually be easily detected by rectal palpation.

Among the general symptoms we must mention, first of all, fever, which is only exceptionally absent, and usually, in the severe cases, shows a pronounced remitting, hectic character. The other general symptoms are the same as in most of the other tubercular diseases—anæmia, emaciation, loss of appetite, increasing bodily weakness, etc. We have a special sign in the occasional co-existence of other tubercular diseases in the body, the lungs, the intestines, the bones, etc., but these may also be wholly absent, so that we have to do with a purely local genito-urinary tuberculosis.

The course of the disease is steadily progressive. Recovery does not occur, at least not in any cases where the disease has attained any extent. The disease lasts from a few months to a year or two, but sometimes much longer. The fatal termination usually ensues from the increasing general weakness, more rarely under the symptoms of ammoniæmia, or sometimes from a miliary tuberculosis or some other tubercular disease, such as pulmonary tuberculosis, tubercular meningitis, etc.

Diagnosis.—The diagnosis of genito-urinary tuberculosis is now usually no longer difficult in fully developed cases, since it can be made with complete certainty by the discovery of the tubercle bacilli joined to the presence of pus in the urine. Of course this gives no information as to the more special extent of the process. In order to judge of this, we must add the local symptoms and the physical examination of the different organs. We are aided in the confirmation of our first suspicion of a tubercular disease chiefly by the consideration of the general condition and the habit of the patient, the discovery of a hereditary taint, or at least the approximate possibility of tubercular infection, and also the discovery of other tubercular affections, especially in the testicles, the hectic fever, and the tedious course, upon which nothing has a favorable influence. At any rate we must make it a rule, in every case of persistent pyuria which can not be otherwise explained, to examine the purulent sediment for tubercle bacilli. We may then often be able to recognize with certainty the milder and incipient cases of this not very rare affection.

Treatment.—Since we do not know at present any efficient remedy to combat the tubercular process, the treatment has merely the task of improving the patient's general condition as far as possible, and also of undertaking a local symptomatic treatment in the same way as in ordinary pyelitis and cystitis (*q. v.*). Of internal remedies we have most frequently used chlorate of potassium and turpentine, and have sometimes seen good results, especially from the latter. The internal exhibition of creosote sometimes seems to us to act well. In vesical tuberculosis it is well to wash out the bladder. As an operative procedure we may remove tubercular testicles and epididymes, but we must remember that in such cases there is usually co-existing disease of other parts of the genito-urinary apparatus, such as the prostate, etc.

CHAPTER IV.

HYDRONEPHROSIS.

(*Dilatation of the Pelvis of the Kidney.*)

Ætiology.—If a contraction arises in any part of the urinary tract and checks the flow of urine, there is a stasis of the urine in the portion behind the stenosis, which gradually leads to a constantly increasing dilatation of the tract as a result of the pressure of the retained fluid. If the obstacle be situated in a ureter, the pelvis of the kidney, as well as the part of the ureter, dilates, and there arises a so-called hydronephrosis. If, however, the obstacle have its seat in the urethra, the bladder and both ureters gradually dilate, and there finally arises a bilateral hydronephrosis.

A closure of the ureter arises most frequently in adults from impacted renal calculi, and also from new growths in the vicinity, in the uterus or ovaries, which compress the ureter from without. So great a pressure may also be exerted on the ureters by the gravid uterus as to be followed by a hydronephrosis, which is usually bilateral. Cicatricial strictures, valve-formations and bends, also are found in the ureter, and form an obstacle to the flow of urine. Finally, in cancer of the bladder the lower opening of the ureter may be contracted or entirely closed.

Constrictions of the urethra, which finally lead to a bilateral hydronephrosis, arise most frequently from strictures as a result of gonorrhœa, and also from enlargements of the prostate. In a rare cases a phimosis may even form the obstacle.

In general, it is evident that gradual constrictions of the urinary tract and periodic obstructions, as from calculi, interrupted by free intervals, lead to more marked degrees of hydronephrosis than rapid and complete obstructions. Under the first-named circumstances the renal secretion persists much longer and is more abundant than in the latter case, when it usually soon ceases. Nevertheless, even then there still follows a further slow distention of the pelvis of the kidney, since its mucous membrane continues to secrete.

It is worthy of note that hydronephrosis may also be congenital, and then it is usually due to congenital defects of development in the ureters or other urinary passages. In later life hydronephrosis is in general more frequently observed in women than in men.

Pathological Anatomy.—The pathological anatomy of hydronephrosis is on the whole very simple. We have a dilatation of the pelvis of the kidney, which is associated with a pressure atrophy of the renal tissue. The papillæ are flattened, the uriniferous tubules and the glomeruli are gradually more and more obliterated, and finally the whole kidney may be changed to a connective-tissue sac filled with fluid. The size of such a hydronephrotic sac may sometimes be so large as to contain ten or twenty quarts (litres) of fluid. The latter consists, of course, at first of urine, but the farther the atrophy of the kidney advances, the more it contains merely the secretion of the mucous membrane. Inflammatory conditions are found in hydronephrosis only when they have existed previously, as in pyelitis calculosa, or when excitants of inflammation in addition have reached the pelvis of the kidney.

Clinical Symptoms.—Since the whole type of the disease is, of course, dependent in many respects upon the nature of the primary disease, we have here to describe only those symptoms which point particularly to the development of hydronephrosis. Such a condition often causes no special clinical symptoms at all, so that we can at most suspect its existence from the presence of an ætiological cause.

The appearance of a visible and palpable tumor is the first definite point in the diagnosis of hydronephrosis. This first shows itself in the region of the affected kidney, but then it gradually enlarges toward the hypochondrium and the median line of the body, and it may finally show very considerable dimensions. The tumor is not movable on respiration. Its resistance is usually quite considerable, but sometimes a marked feeling of fluctuation may be present. On percussion, the tumor gives a dull note, from which the tympanitic note of the colon in front of the tumor is sometimes distinct (see page 873). It is an important diagnostic sign if the tumor show variations in its size at times, since it decreases in size with a co-existing increase in diuresis, and increases again when the amount of urine becomes smaller.

In doubtful cases an exploratory puncture of the tumor may also be of significance in diagnosis. It of course favors the existence of hydronephrosis if urinary constituents, especially urea, can be found in the fluid evacuated; but if the hydronephrosis be of long standing, its contents, as we have said, will be simply seromucous, and then chemical examination gives no definite data for distinguishing hydronephrosis from ovarian tumors, or other cystic tumors of the kidney. In regard to the procedure first devised by Simon, which is also important in regard to palliative therapeutics—namely, catheterization of the ureter in women after having previously dilated the urethra artificially, and in this way confirming the diagnosis—the details may be found in the special works on surgery.

The secretion of urine in unilateral hydronephrosis may be completely normal if the other healthy kidney act vicariously. In stricture of the urethra, and also in bilateral constrictions of the ureters, however, there is, of course, an obstacle for the passage of urine, so that the amount of urine may be abnormally small. There may be at times complete anuria, and even uræmic symptoms. The composition of the urine depends entirely upon the form of the primary disease. If only the healthy kidney secrete, the urine passed is normal. If there be at the same time pyelitis or cystitis, the urine may contain pus or blood. If the urine can also come from the diseased kidney at one time and not at another, the urine also exhibits a varying composition, as we have said before (page 886).

In many cases of hydronephrosis quite severe local symptoms are present; there are frequently severe pains in the tumor, which shoot chiefly toward the thigh. Of course, these local symptoms are sometimes only slight. In regard to the symptoms on the part of other organs, gastric disturbances appear to be of the most frequent occurrence; among them are nausea, loss of appetite, vomiting, and eructations. In some cases the bowels are constipated, in others there is obstinate diarrhœa.

The whole course of the disease is always chronic. There are often variations in its course, but no general statements can be given, because the conditions vary in the different cases according to the form of the primary disease. Most cases of hydronephrosis end fatally, either in consequence of the primary disease or in consequence of secondary pyelo-nephritic or perinephritic inflammations, of uræmia, etc. Recovery takes place in rare cases, especially if one kidney be perfectly normal, and there be no incurable primary disease. Recovery may ensue spontaneously from perforation or obliteration, or it may be brought about artificially from operative procedures.

In the **diagnosis** of hydronephrosis, the points especially to be considered have already been mentioned. The diagnosis is usually not easy, especially if the ætiological factors be unknown; and the disease is often confused with other renal tumors and echinococci of the kidneys, with ovarian tumors, and even with splenic and hepatic tumors.

Treatment.—Except for the symptomatic treatment of the pain and any accompanying pyelo-cystitis, an efficient treatment of hydronephrosis can be attempted only by surgical means. Puncture, incision, extirpation of the kidney, and the establishment of a renal fistula, are the methods of operation most in use—the details of which are to be found in the special surgical treatises.

CHAPTER V.

CYSTITIS.

(Vesical Catarrh.)

Ætiology.—In most cases of vesical catarrh the agents of inflammation reach the bladder from without through the urethra. The most unequivocal experiment in this regard is, unfortunately, often made by the physician himself, when he excites a cystitis by the use of an insufficiently purified and disinfected catheter or bougie. The development of the vesical catarrh is generally aided in such cases by the fact that there is usually a defective evacuation of urine, from stricture of the urethra or paralysis of the detrusor, and that there is at the same time retention of urine, in which the bacteria can develop undisturbed. The agents of inflammation may also enter from the urethra into the bladder in incontinence of urine. On account of the imperfect closure of the sphincter, a stagnating column of urine, directly connected with the contents of the bladder, forms in the urethra, and to this column the air and the bacteria that excite decomposition of the urine have direct access. In this way many cases of cystitis arise in patients with nervous disease who have paralysis of the bladder, and also many of the frequent cases of cystitis arise in this way in persons who are severely ill and stupid from some other disease, such as typhoid fever.

Cystitis often follows diseases of the neighboring urinary tract. Gonorrhœal urethritis is the most common, and this invades the bladder directly and leads to a gonorrhœal cystitis. In women, the agents of inflammation may quite easily enter the bladder from the vagina through the short female urethra. Thus arise especially the frequent cases of cystitis in childbed. In some cases communications may develop between the bladder and certain neighboring organs, as in vesico-rectal or vesico-vaginal fistulæ, by which again access to the bladder is open to the agents of inflammation.

Another group of cases is due to the presence of foreign bodies, which irritate the vesical mucous membrane mechanically. Among these is, first of all, the cystitis which so often accompanies stone in the bladder. It must be stated, however, that many cases of the vesical catarrh which here exists are not directly dependent upon the calculi, but are first excited by examination with catheters and sounds.

In distinction from the methods of the origin of cystitis so far described, the production of inflammation by way of the blood-supply is much rarer. Certain chemical substances, already mentioned (page 837), which are eliminated by the kidneys and provoke an inflammation of the urinary tract, are the most important in this respect. Cantharides shows the most intense action of this sort, and it may cause an actual croupous cystitis. Slight irritative states of the bladder also frequently appear after taking certain foods and drinks, as after drinking new beer. Infectious substances only rarely come under consideration in this regard. Most of the cases of cystitis in severe acute infectious diseases are secondary complications (*vide supra*). It can not be doubted that in some cases an apparently idiopathic primary cystitis appears after exposure to cold, but it is very rare. In such cases we usually have to do with acute exacerbations of an old chronic cystitis—for example, of gonorrhœal origin.

It has been stated in the previous chapters how frequently cystitis is only one symptom of a more extensive disease of the urinary tract. As cystitis may further invade the pelvis of the kidney through the ureters, so, on the other hand, any pyelitis of primary origin may extend downward and involve the bladder.

Pathological Anatomy.—The pathological anatomy of cystitis presents the same conditions as the inflammation of any other mucous membrane. In simple catarrhal cystitis the mucous membrane is swollen and covered with pus, and is often studded with hæmorrhages. In old chronic cystitis the mucous membrane often takes on a slaty, grayish-black color as a result of the hæmorrhages. The severer forms of cystitis, such as are often observed in diseases of the spinal cord, are termed vesical diphtheria. These cases come to a necrotic destruction of the superficial layers of the mucous membrane, ulcerations, etc. In such severe cases submucous and pericystitic abscesses sometimes develop, which may perforate into the surrounding parts in various ways. The incrustation of the mucous membrane with urinary salts, especially with ammonio-magnesic phosphate, is also frequently found in chronic cystitis, and is worthy of mention.

Clinical Symptoms.—The local symptoms in the bladder are sometimes quite severe in cystitis, but in other cases they are only slight. In general, they show a greater intensity in acute cases than in chronic cystitis. The pain in the region of the bladder is rarely entirely continuous; it usually comes on only on micturition, but it is often very distressing then, and shoots to the opening of the urethra. Since the inflamed vesical mucous membrane shows an increased irritability, and since the morbidly altered urine (*vide infra*) also exerts an abnormal irritation on the mucous membrane, there is very often an increased desire to micturate. The patient has to empty the bladder much oftener than normal, and in severe cases there is an almost constant, painful "vesical tenesmus," from which, at any attempt to micturate, only a very small amount of urine is passed, with severe burning. As a result of the increased irritability of the vesical mucous membrane, there sometimes comes on a very troublesome reflex spasm of the sphincter, by which the symptoms are increased.

Only the character of the urine is decisive in the diagnosis. This is secreted in a perfectly normal amount and character, in case there is no complication on the part of the kidneys; but in the bladder it is mixed with the products of the diseased mucous membrane, and it is here exposed to the action of the bacteria in a way that will presently be described. The abnormal admixtures in the urine consist chiefly of pus-corpuscles and bladder epithelium, and sometimes of some of the mucus produced by the mucous membrane. The specific action of the bacteria, which have reached the bladder from without, consists of the so-called alkaline fermentation of the urine—that is, in the fermentative change of urea into carbonate of ammonia. This process is associated entirely with the presence of certain micro-organisms, the "*micrococci ureæ*," and the retention of urine as such never leads to an alkaline fermentation. As Lépine and Roux have shown, we can produce a very severe cystitis, and even nephritis, by injecting very small amounts of a pure culture of micrococcus ureæ (Cohn) into the bladder of a guinea-pig. The stagnation is only a factor, which greatly aids the whole process since the activity of the bacteria, as we have said, can develop much better here than if the bladder were to a certain degree constantly purified and washed out by the urine that is always coming afresh. As soon as a part of the urea is changed to carbonate of ammonia, the acid reaction of the urine must be less. The urine has a faintly acid or neutral reaction, and sometimes it is even already decidedly alkaline when passed. The latter, however, is only rarely the case, but it is often simulated by the fact that the urine is not examined until it has stood for some time. Since during this time the alkaline fermentation which has once begun makes rapid progress, the cystitic urine that has stood is very often alkaline. Many crystals of ammonio-magnesic phosphate and urate of ammonia then form in it; the former are easily recognized by their "coffin-lid shape," and the latter by their "thorn-apple shape" (see Fig. 116).

If we then briefly put together what has been said, the urine is passed in about the normal amount in cystitis. It usually looks clear, and has an abundant sediment, which can often be recognized as purulent with the naked eye, and in which, microscopically, we can find pus-corpuscles, often bladder epithelium, and constantly innumerable bacteria—usually short rods in vigorous motion. The alkaline fermentation may usually be recognized by the strong ammoniacal odor, and also, as we have said, by the reaction of the urine. In the severe diphtheritic forms of cystitis we find entire shreds of necrotic tissue in the urine. If there are hæmorrhages in the bladder, the urine often contains red blood-corpuscles and sometimes even large blood-clots. The mucus in the urine appears in milder cases as a cloudy opacity—"*nubecula.*" The viscid masses which can be drawn out into threads, and which are usually abundant in the urine in severe cystitis, are not the special product of the mucous membrane, mucine, but they arise from the pus-corpuscles and the epithelium dissolved in the alkaline urine, and hence give the reactions for albumen. It goes without saying (compare page 824) that every cystitic urine is albuminous from its mixture with pus-serum. The presence of slimy threads in the urine—the so-called "clap-threads" (*Tripperfäden*)—is characteristic of gonorrhœal cystitis.

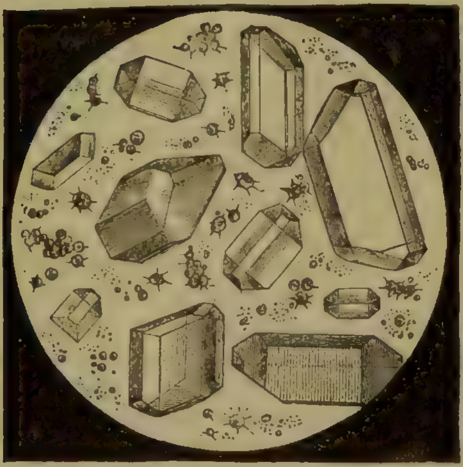

FIG. 116.—Crystals of triple phosphate and ammonic urate. (From FUNKE.)

There can be no doubt that the decomposing alkaline urine acts as a chemical excitant of inflammation on the vesical mucous membrane. Hence cystitis often arises perhaps in this way, that the bacteria which have entered the bladder first excite only an alkaline fermentation, and that then the mucous membrane is affected by the irritation of the ammonia salts that are formed. It is, however, at present hard to decide, and it is also without special practical interest, whether the bacteria as such can not directly excite inflammation.

The other morbid symptoms associated with cystitis usually depend only in part upon the disease itself and in part upon some existing primary disease. The most important symptom is the fever, which is often to be referred directly to the cystitis. In severer cases it may be very intense, and often assumes a pyæmic intermittent character, especially if there have arisen pericystitic suppurations or if the cystitis has extended to the pelvis of the kidney and the kidneys (see page 881). An acute cystitis may also begin with a chill and high fever. If the escape of the purulent urine, however, always remains undisturbed, the fever may be entirely absent in spite of the existence of cystitis.

Sometimes in severe cystitis with a marked alkaline fermentation certain nervous symptoms appear, such as headache, vertigo, stupor, and nausea. The idea has been advanced that in these cases we have to do with an auto-intoxication of the body, since ammonia and perhaps other products of decomposition, like sulphuretted hydrogen (?), are absorbed from the bladder into the blood (ammoniæmia), and in this way excite the symptoms of poisoning mentioned.

According to the course of the disease we distinguish an acute and a chronic cystitis. The former, which may come on, for example, after catheterization, in gonorrhœa, etc., often passes off favorably after a few days. The amount of mucus and pus in the urine remains slight. Chronic cystitis is observed especially as a complication in other diseases of the urinary tract, like stricture, in chronic diseases of the spinal cord with paralysis of the bladder, etc. It is very often incurable because the primary disease is incapable of improvement and the cause of the disease therefore persists. The longer a cystitis lasts, the nearer is the possibility of the development of more severe and dangerous complications, especially the development of a pyelo-nephritis, and the formation of pericystitic suppurations. In this way cystitis, especially in chronic nervous diseases, may become the immediate cause of death.

Treatment.—The dangers last mentioned must urgently impress upon us the prophylaxis of cystitis. Fortunately, a good deal can be done in this respect, in the first place, by the avoidance of all unnecessary use of bougies and catheters, by the greatest care for cleanliness in the use of all instruments of this sort, and by the timely treatment of all those conditions which may lead to cystitis.

The treatment of cystitis is, in the milder and acute cases, hygienic and medicinal, but in the severer cases only a careful local treatment can be useful.

In any severe, and especially in any acute cystitis, the greatest bodily rest (if possible rest in bed) is urgently desirable, since otherwise an increase of the symptoms and a prolongation of the course of the disease is the almost inevitable result. The diet must be mild and unirritating. Spiced food and alcoholic drinks are to be avoided, but we should recommend an abundant supply of fluid, by which the urine is diluted and the bladder washed out. We have the patient drink plenty of ordinary water, tea, or a suitable mineral-water, like Wildunger, Selters, or Fachinger. A diet mainly of milk is very good; by it the cystitic symptoms often very rapidly cease.

Among internal remedies those are to be considered which are eliminated with the urine, and are thus able to act on the diseased mucous membrane, or directly upon the agents of inflammation. One of the most efficient drugs, which never does harm with necessary caution, is chlorate of potassium, of whose favorable influence on vesical catarrh we have often convinced ourselves. It is prescribed in an aqueous solution, forty to seventy-five grains a day (grm. 3–5), and should never be taken on an empty stomach. Salicylic acid is sometimes used with good results in doses of half a drachm to a drachm (grm. 2–4) a day in ten-grain (grm. 0·5) capsules. The two remedies mentioned have largely replaced tannin, which was formerly in great favor. At present a decoction of uva ursi is still more frequently prescribed, 10 or 15 to 150, whose active principle, arbutine, in doses of forty-five to sixty grains a day (grm. 3–4) in an aqueous solution, seems worthy of further trial (Lewin and others). In more advanced stages of vesical catarrh, if the initial symptoms of irritation have ceased, the resinous drugs are to be used, of which oil of turpentine and balsam of copaiba especially sometimes show a very good action. Both are best given in gelatine capsules.

If there are severe local symptoms, we prescribe warm compresses and poultices to the region of the bladder. In robust persons with acute cystitis, local blood-letting, three to six leeches to the perineum, sometimes has a decidedly favorable symptomatic action in such a case. In other respects narcotics, especially subcutaneous injections of morphine, are the best remedy where there is severe pain and tenesmus. Camphor, extract of belladonna, etc., are much more uncertain in their action. The frequent use of protracted warm baths may, however, be greatly recommended.

In chronic cystitis all the remedies previously mentioned are also to be considered; but they are usually not sufficient alone, and at any rate they are far less effective than a methodical local treatment. This consists in washing out the bladder regularly every day by the aid of an elastic catheter, to which a T-tube is fastened by means of rubber tubing, one arm being connected with an irrigator and the other with the escape-tube. We thus let a moderate amount of fluid, eight or ten ounces (200–300 c. c.), run into the bladder and run out again, frequently repeating the process, until it comes away perfectly clear. For this purpose we use either pure warm water, or, better, a dilute solution of plumbic acetate (1 to 1000), permanganate of potassium (1 to 1000), boric and salicylic water, and the like. By such a treatment many cases of chronic vesical catarrh may recover, and others may at least be kept constantly in check.

Attention to the causal indication is sometimes very important also in chronic vesical catarrh—for example, the treatment of any strictures, the removal of calculi, or the improvement of paralytic states of the bladder.

In pericystitic suppuration surgical treatment is only rarely possible. We must, therefore, confine ourselves to purely symptomatic procedures.

CHAPTER VI.

NEW GROWTHS IN THE BLADDER.

Primary new growths in the bladder are quite rare. The commonest is the so-called villous cancer (which is properly a papillary fibroma), which may attain the size of a walnut, and is usually situated in the lower portion of the bladder near the entrance of the urethra. Since the tumor is usually very vascular, there are often hæmorrhages into the bladder, and repeated hæmaturia is therefore one of the commonest symptoms of vesical papilloma. In this affection the blood-clots often assume a peculiar long, worm-like shape, from their passage through the urethra. Severe symptoms on micturition sometimes appear, since portions of the tumor may lie in front of the opening of the urethra. A definite diagnosis of a villous tumor is possible only when single portions of the tumor are thrown off, and are found in the urine passed. Examination of the bladder by the catheter may also give information as to the presence and seat of the tumor.

Primary carcinoma of the bladder is rare. It is usually spread diffusely over the wall of the bladder, and leads to so considerable a thickening of it that we can often feel the bladder from without through the abdominal walls. Otherwise the symptoms are the same as in severe chronic cystitis. The urine contains much pus, and is sometimes bloody. The general cancerous cachexia developed rather late in the cases which we have seen, of which one was in a man still quite young. The diagnosis is not always easy. Except from attention to the general course of the disease and any vesical tumor that may be felt, it must aim to be based chiefly upon the discovery of particles of cancer in the urine.

A secondary invasion of the bladder by carcinomatous new growths from the uterus, rectum, and vagina is quite frequently observed.

The treatment can usually be only symptomatic, since surgical procedures are possible only in rare cases.

CHAPTER VII.

ENURESIS NOCTURNA.

(Nocturnal incontinence of Urine.)

ENURESIS nocturna is a nervous affection of the bladder by no means rare in children of both sexes, and therefore quite important in its practical relations. Of course, in small children there is no sharp boundary to be drawn between normal and pathological conditions; but it is decidedly pathological if larger children, from four to ten years of age and even older, pass their urine in bed more or less frequently during sleep, in spite of well-developed reasoning powers and professedly the best intentions. This anomaly may extend to the years of puberty and even beyond it, and then it frequently produces a very depressing mental affection for the patient. Special causes for it are not to be discovered in most cases. We are compelled to assume either an abnormal weakness of the sphincter, which is probably sometimes congenital, or an abnormal irritability of the detrusor. At any rate, in wetting the bed at night the process of micturition comes on in a purely reflex way, but it is often accompanied by certain ideas in dreams referable to micturition. It does not hold in all cases that the sleep is especially deep. Many patients, of course, first notice the trouble in the morning, but others almost always wake directly after. The involuntary micturition usually occurs in the first hours after going to sleep, but sometimes it is later, and even first toward morning. By day micturition is often perfectly normal ; but in many cases there is even then a noticeable weakness of the bladder, so that the child has to make water oftener than usual, and sometimes wets its clothes even by day.

Although, as we have said, we can usually find no special cause for the trouble, still, in some cases, certain morbid changes in the urinary organs may give rise to the incontinence. We should, therefore, in every case at least think of the possibility of stone in the bladder, of congenital phimosis and adhesions of the prepuce to the glans penis, of ascarides, and of inflammatory conditions, and make a special examination of these points. We must also bear in mind polyuria caused by diabetes or renal disease, and finally, of course, in the diagnosis of a purely nervous nocturnal incontinence of urine, we must exclude the existence of any actual anatomical spinal affection.

In all the cases just mentioned, the treatment must of course refer first to the primary disease ; but in the ordinary nocturnal incontinence the treatment must first take into consideration the prevention of the appearance of nocturnal micturition as far as possible. The child must take only a very little fluid in the evening, and he should be made to empty his bladder immediately before going to sleep, and once again later. He should not be covered up too warmly, and, if possible, he should not lie on his back during sleep. Tying a brush to the back is therefore a well-known domestic remedy. A somewhat strict mental treatment is often effective, since thus the attention to the process is increased, although unconsciously, and the child often learns to wake up at the right time. The use of the rod is of course on the whole only rarely admissible. On the contrary, we often have to protect the child against unreasoning parents.

Internal remedies, especially belladonna and tincture of nux vomica, which were formerly recommended, rarely avail. Iron preparations are indicated only in anæmic children. Electrical treatment, however, is often, if not always, very effective. We put the broad anode over the lumbar cord, and the smaller kathode over the region of the bladder or on the perineum, and let quite a strong constant current pass through for two or three minutes. Then we pass the wire end of a conducting cord, which we make the kathode, into the mouth of the urethra for one or two centimetres, and let quite a strong and somewhat painful faradic current act for one or two minutes (Seeligmüller). The sittings must at first be repeated daily. It is also a very good plan to let the whole body be well rubbed with cold water before going to sleep.

The prognosis of these forms of incontinence, which have no organic disease at the bottom of them, is almost always favorable, since in the worst case the anomalous condition usually disappears gradually of itself with increasing years.

[Belladonna, strychnia or nux vomica, or a combination of the two, are often of unquestionable service. If the enuresis is only nocturnal, belladonna alone may be used, either in a single dose at bedtime or three times a day. If the enuresis is diurnal also, the two drugs should be combined and given three or four times a day.]

DISEASES OF THE ORGANS OF LOCOMOTION.

CHAPTER I.

ACUTE ARTICULAR RHEUMATISM.

Ætiology.—Acute articular rheumatism is an infectious disease. This is shown by all the clinical and anatomical peculiarities of the disease; and, although the specific organic pathogenetic poison can not yet be demonstrated, still this view of the disease, which was first brought forward by Hüter, is the only one which enables us properly to understand its symptoms and course.

Like many other infectious diseases, acute articular rheumatism is often indisputably endemic and epidemic. According to Hirsch, the disease is most prevalent in the temperate zones, being much rarer in cold and tropical latitudes; but even in Europe it is by no means uniform in its frequency, and certain districts of England, Belgium, and Russia are said to be almost exempt from it. It is also possible to observe epidemic influences with regard to the frequency of its appearance, as already intimated. Here in Leipsic, where articular rheumatism is one of the most frequent of acute diseases, we have observed for years that at certain times there are only a few cases, while at others there is a striking increase in their number. Usually attacks are most prevalent in the winter and spring months, but again it is sometimes in summer that the disease is especially common.

Among the exciting causes of the disease, taking cold is always mentioned as of first importance; and in fact it can not be denied that the influence of cold does often seem to contribute to the occurrence of the disease.

This result, however, seldom follows a single severe exposure, but it rather follows persistent causes, and in particular the long-continued influence of wet and cold, as in certain occupations—for example, washing and scrubbing, or inhabiting unhealthy damp dwellings, and the like. This explains why those who follow certain callings are especially subject to articular rheumatism; thus servant-girls and coachmen are frequently victims to the disease. And yet it is possible to regard all these injurious influences as being merely indirect causes, inasmuch as they favor the development or action of the specific micro-organisms; and, furthermore, it is by no means exceptional to see a case of articular rheumatism where no history of exposure to cold can be obtained.

Sex exerts no special influence upon the frequency of the disease. As to age, acute articular rheumatism is most frequent in young adults between fifteen and thirty-five years of age. In later life, and particularly in old age, it is much rarer. In children six years old or more the disease is not especially infrequent, but in younger children it occurs only exceptionally. We may be permitted to mention a single interesting case which we met with here in Leipsic, where a child who died when only a few days old, and whose mother at the time of its birth was suffering from a severe attack of acute articular rheumatism, was found to have multiple purulent arthritis.

Much has been said with regard to the relations of acute articular rheumatism to other acute diseases. We must, therefore, call particular attention to the fact that the joint diseases, whether simple or multiple, which occur after scarlet fever, gonorrhœa, or in connection with puerperal and septic diseases, as well as recent cases of secondary syphilis, have nothing to do with genuine acute rheumatism. In cases of this sort the trouble in the joints is merely a special localization of the general disease; and, indeed, the circumstance that the joints are a favorite point of attack for infectious diseases may be brought forward as another proof that acute articular rheumatism is of infectious origin. There is, however, a single affection, namely, chronic endocarditis, about which the facts are different. This disease is ætiologically identical with acute endocarditis, and therefore with acute rheumatism (*vide infra*). At least this is true in many cases, although probably not in all. It may be regarded as a proof of this connection between the two diseases that patients with chronic cardiac disease are especially liable to attacks of acute articular rheumatism. Here, therefore, we have a genuine articular rheumatism as a symptom of more general disease. It may be regarded in some sense as a fresh acute exacerbation of this disease, localized mainly in the joints.

A very noteworthy fact is that acute articular rheumatism can not be numbered among those infectious diseases which usually occur but once in the same individual. This disease, on the contrary, resembles pneumonia and erysipelas, in that it is very apt to occur repeatedly in the same person. Acute rheumatism, therefore, even when it ends favorably and leaves behind it no evident lesions, seems to render the patient more liable to the disease than he was before.

Symptomatology.—The chief symptom of acute articular rheumatism is an acute febrile synovitis, which almost always affects several joints. The synovitis is associated with the usual local phenomena of swelling and tenderness in the parts affected. Often this articular affection is the first symptom, and, indeed, it may be the only symptom of the disease. It is, however, by no means exceptional for the arthritic trouble to be preceded by certain prodromal or initiatory symptoms, as is true of other infectious diseases. These prodromata consist either of a slight general malaise, or of certain local symptoms. It is not rare to have sore throat, or, as we have repeatedly had opportunity to observe, laryngitis. These premonitory symptoms are, however, generally insignificant, and may, as we have said, be entirely absent.

The articular disturbance is almost always very rapid in its development. Some of the larger joints are usually first affected, and perhaps those of the lower extremities somewhat oftener than those of the upper. It is extremely exceptional for all the joints that are affected to be attacked at one and the same time. It is somewhat characteristic of acute articular rheumatism that it "jumps from one joint to another." To-day this joint will be affected and to-morrow that, while the joint first attacked may still remain diseased or undergo rapid recovery. Thus, there may be either a few joints affected or many, in varying sequence, and they may sometimes be affected rapidly and at other times more slowly. In many of the milder cases the disease is an extremely temporary one, while in others it may attach itself most persistently to some one or more joints.

There is usually fever in addition to the arthritis; but it is not usually very high, seldom exceeding 103° (39·5° C.). The fever, on the whole, corresponds with the arthritic phenomena, and does not present a curve which is at all typical, but one which is irregularly remittent. We have scarcely ever seen the disease begin with an initial rigor, nor are the so-called "general febrile symptoms" of headache, stupor, and subjective feeling of heat as a rule at all marked in acute rheumatism.

This indicates that the constitutional infection does not as a rule attain great severity. The skin is noticeably inclined to perspiration, but the perspiration is not at all a result of any sudden fall in temperature such as is seen in other diseases.

The course of the disease is marked by alternate ameliorations and aggravations of the local symptoms and of the fever, and lasts, particularly if it is not treated, one or more weeks, or a still longer period. Then, as a rule, the symptoms gradually abate and convalescence begins; but it generally is tedious and frequently interrupted by relapses. In other cases, however, the disease contrasts strongly with this simple course, for articular rheumatism is notoriously subject to numerous complications and peculiarities in its course. The protean character of the disease will be evident upon a perusal of the following description of the symptoms referable to the different organs of the body.

Symptoms referable to the Different Organs, and Peculiarities in the Course of the Disease. 1. JOINTS AND SHEATHS OF THE TENDONS.—The favorable termination of most cases of acute articular rheumatism prevents us from often examining the anatomical changes in the affected joints; but there can be no doubt that in most instances the trouble is merely a simple serous synovitis—that is, an inflammation of the synovial membrane, with an exudation into the cavity of the joint composed mainly of serum with but little admixture of fibrine and pus. The synovial membrane itself, in the cases which do come to autopsy, is usually very little affected. It is somewhat injected, opaque, and thickened. Necrosis of the cartilages is seen only in severe cases, or in those which have lasted a rather long while. From a clinical standpoint, the articular disturbance is noticeable chiefly for the pain which it causes the patient upon every movement of the joint and any pressure upon it. The painfulness is often in striking contrast with the slight apparent change in the structure of the joint, for a joint which is extremely sensitive may appear to be scarcely at all diseased. Usually, however, the joints exhibit the signs of synovitis. The effusion into the joint produces an evident swelling, which can be seen particularly well in the knees, but also in the joints of the ankle, wrist, shoulder, and elbow, and sometimes even in the smaller joints of the fingers and toes, particularly the great toe. It is rather exceptional to detect swelling of the hip-joint. It should be remembered, however, that the swelling in the region of the joint, particularly the ankle or wrist, is often less the result of a synovial effusion than of an inflammatory periarticular œdema. This œdema, for example, may extend over almost the whole posterior surface of the hand. The joints are by no means invariably the only parts attacked. Not infrequently there are analogous inflammatory changes visible in the sheaths of the tendons, the bursæ, and perhaps, in many cases, even the fasciæ and muscles. The skin over the affected joints often has an inflammatory blush, which is usually pale red and spotted, and can be best seen at the ankle, knee, and wrist. It has been maintained that the cutaneous sensibility is diminished over the joints affected, but we regard this as a mistake.

As might be expected, the number of joints attacked and the sequence in which they are attacked differ greatly in different cases; but almost invariably a number of joints suffer, so that any monarticular arthritis should not be regarded as rheumatic except after careful deliberation (*vide infra*, diagnosis). It should be said that, in mild cases, there may be only two or three joints affected, these being usually some of the larger joints of the extremities; and of these, one may be so much worse than the others that their participation in the trouble can be ascertained only by careful questioning and examination. In severe cases, on the other hand, the number of joints attacked is often very great. Such patients become extremely helpless, because any movement is possible, if at all, only under the penalty of very severe suffering. The patient usually lies with bended knees and feet curved so as to be concave on the plantar surface, and screams with pain at any attempt to change his position. The joints of the trunk sometimes participate in the disease, but hardly ever except in the severe cases. The articulations of the vertebræ, the sterno-clavicular joint, the articulation of the lower jaw, and the symphysis pubis, are particularly apt to be affected. The fugitive character of the arthritis has been spoken of as characteristic of acute articular rheumatism, and, indeed, it is not infrequently the case that comparatively large swellings of the joints soon abate and yield to new disturbances in other joints; but, on the other hand, the disease may persist very obstinately in a single joint. In this case one joint, or rarely several, are attacked with marked severity, either from the start or subsequently to milder affections of other joints, and often remain for weeks swollen or painful long after all other symptoms have vanished.

2. CARDIAC SYMPTOMS.—The condition of the heart in acute articular rheumatism is next in importance to that of the joints. The physician should therefore, in every case, even the mildest, maintain a continuous watch over this organ. In 1836 Bouillaud made careful auscultatory investigations in this disease, and was thus the first to discover that the course of acute articular rheumatism is, with noticeable frequency, accompanied by endocarditis, and sometimes even by pericarditis. Complications of this sort may occur in any case, whether mild or severe, or may be absent in any case, even the worst. They may develop at the beginning or later on in the course of the disease. Their development is often unattended by any subjective symptoms, so that they can be recognized only by careful physical examination. In many cases, however, the onset of cardiac disease is marked by a fresh exacerbation of the fever, or possibly by palpitation, or by painful sensations in the præcordia, or by dyspnœa.

We will consider first rheumatic endocarditis. This is almost always the benign verrucous variety (see page 278).

It is far more prone to attack the mitral than the aortic valves, and is accordingly usually betrayed by a blowing systolic murmur at the heart's apex. Uncertainty may be cast upon the diagnosis by the fact that functional murmurs are not very infrequent at the apex of the heart in cases of acute articular rheumatism. We once observed a case of "hyperpyretic rheumatism" (*vide infra*) where there was an evident murmur of this sort during life, and yet at the autopsy we were able to assure ourselves of the complete integrity of the cardiac valves. Even an expert may for a time be in doubt as to the significance of many cardiac murmurs, and this explains in part the conflicting statements as to the frequency of cardiac complications in acute rheumatism. In general, one may say that such complications occur in 25 to 33 per cent. of the cases. The ultimate results of this endocarditis we do not need to describe over again in this connection (see the chapters on acute and chronic endocarditis). Complete recovery is possible. Often, however, the lesion gives rise to a chronic endocarditis—that is, to a cardiac valvular disease, which lasts through life.

The close connection between endocarditis and the arthritis must formerly have seemed very puzzling, despite the many hypotheses made to explain it. If, however, we regard acute articular rheumatism as an infectious disease, this obscurity vanishes. Acute articular rheumatism is plainly not a merely local disease, but the result of a general infection. The specific pathogenetic matter is not only present in the joints affected, but it also circulates in the blood. It is therefore easy to understand why the valves of the heart should be attacked by it, as is the case in so many infectious diseases (see page 276). Often the specific properties of the micro-organisms in question produce a typical endocarditis. The endocarditis, therefore, is not strictly a "complication," but a symptom of the disease.

Rheumatic pericarditis is not infrequent, although less common than endocarditis. The only certain way to recognize it is by a characteristic friction-sound; and even when this is heard there may be a doubt as to its significance, inasmuch as functional murmurs are not infrequently heard at the base of the heart. The pericarditis is of a sero-fibrinous nature. Sometimes it is of slight severity; but it may be extremely severe, with a large effusion and the most urgent dyspnœa (see page 323). In rare instances this pericarditis proves fatal. Usually, however, recovery ensues, although in severe cases there may be obliteration of the pericardial sac, with the consequences described on page 326.

With regard to the origin of the pericarditis, it may be said that it would not be impossible for the pericardium to be directly infected by the blood. We have reason, however, to believe that in most cases the infection proceeds from the endocardium, and probably in the great majority of cases from the aortic valves (see page 298). It is not always possible to prove that an endocarditis precedes the pericarditis, but yet this does not overthrow the opinion we have expressed, inasmuch as many cases of acute endocarditis do not betray themselves by any audible murmurs.

We should also mention that there may be functional cardiac derangement without any grave anatomical lesion. We have already spoken of the functional murmurs. There may also be a rapid and irregular pulse, and, in rare instances, attacks of angina pectoris of apparently purely nervous origin.

3. Serous and Mucous Membranes.—The pleura and peritoneum, as well as the pericardium, may be affected in articular rheumatism; so that it was formerly often maintained that acute articular rheumatism is a disease of the serous membranes of the body in general, inclusive of the joints. Rheumatic pleurisy is much rarer than either endocarditis or pericarditis; and rheumatic peritonitis is rarer still. The pleurisy, at least in most instances, is propagated directly from an inflamed pericardium; and in the same way the peritoneum may become infected from the pleura by way of the diaphragm. Few of these severe cases, presenting a simultaneous inflammation of several serous membranes, occur, now that the salicylic-acid treatment has been introduced. We do not say that it is absolutely impossible for a pleurisy or a peritonitis to occur in rheumatism without inflammation in any other serous cavity, but such an occurrence is extremely rare.

The mucous membranes are seldom greatly affected in acute articular rheumatism. As has already been stated, a catarrh of the pharynx or larynx sometimes occurs in the beginning of the disease. Bronchitis is often spoken of by the older authors; but it is probably in many cases not due directly to the rheumatism, but it is a complication, just as in any disease attended by great prostration. The stomach and intestinal canal are seldom especially affected.

4. Skin.—Cutaneous phenomena are not infrequent in the course of acute articular rheumatism. A prominent symptom of the disease is the tendency to profuse perspiration. The perspiration often has a strongly acid odor and reaction. Many patients exhibit an abundant crop of sudamina, the back in particular being sometimes entirely covered. Sometimes there are other cutaneous eruptions. In a whole series of cases we observed erythema nodosum. This affected the lower extremities more than the upper. Urticaria is not very infrequent, while herpes labialis has been very rare in our experience. It is well known that arthritic affections and the so-called "hæmorrhagic diseases" are in many ways related to each other; and it is therefore an interesting fact that extensive hæmorrhagic disturbance of the skin also occurs in connection with acute articular rheumatism, as we have repeatedly had opportunity to observe. We have seen several cases of hæmorrhagic urticaria: wheals appear upon the

skin, and a hæmorrhage takes place into their centers and spreads gradually. There may also be simple cutaneous ecchymoses. These may in severe cases be merely one symptom of a general hæmorrhagic diathesis, with hæmorrhages from mucous membranes. The occurrence of these symptoms again points most clearly to the infectious character of acute articular rheumatism.

5. THE MUSCLES AND NERVOUS SYSTEM.—The condition of the muscles is very important in many cases of acute polyarthritis. They are often quite painful on pressure, and apparently somewhat swollen about a joint that has been long affected. The muscular atrophies and muscular paralyses that often remain after the arthritis is well are especially important.

According to a general law, there are certain definite trophic relations between a joint and the muscles belonging to it, whereby almost every severe and persistent disease of the joint is necessarily followed by an atrophy of the affected muscles. The extensors of the joint are often most affected by this atrophy. This atrophy has long been recognized, and was formerly regarded, particularly by surgeons, as merely the result of inactivity of the muscles—"atrophy from disuse"; but this view is certainly erroneous. We do not know its precise cause, but it is without doubt the result of the disease of the joint, and may therefore be termed "muscular atrophy of arthritic origin." If an attack of acute articular rheumatism affects any one joint for a long period there is a secondary atrophy of the corresponding muscles. This is seen most often, and in its most typical form, where there is obstinate trouble in the shoulder-joint, the deltoid becoming extremely atrophied. This atrophy of the muscles may contribute largely to the sum-total of functional derangement. We have repeatedly seen cases where the patient could hardly lift his arm at all, although the inflammation of the shoulder-joint had passed away, and that without leaving any anchylosis. It is therefore entirely justifiable to speak of a rheumatic paralysis. We have seen similar muscular paralyses after acute articular rheumatism in the rest of the muscles of the upper arm, also in the quadriceps extensor, and once in the serratus magnus. The explanation given by Charcot, that the cause of the atrophy is a "reflex" implication of the trophic centers in the cord which starts from the joint, seems to us hardly satisfactory. There are probably local disturbances of nutrition, and sometimes apparently a direct extension of the inflammatory process from the joint to the neighboring muscles. It is noteworthy that the atrophied muscles respond promptly to the faradic current, and never exhibit the reaction of degeneration.

Chorea (*vide supra*, page 780) may be a sequel of acute articular rheumatism, and it is one of the nervous symptoms which may arise in connection with it. This complication is seen most frequently in children. Endocarditis may accompany it, but it does not always do so.

There are certain peculiar cases of acute articular rheumatism which excite the greatest interest. In these, very severe cerebral symptoms are developed, often most acutely. They are therefore called "cerebral rheumatism"; or, as they are almost always characterized by an extraordinarily high temperature, another name is "hyperpyretic articular rheumatism." In these cases the disease may exhibit severe nervous symptoms from the start, particularly delirium; or it may at first run an apparently favorable course, and not change for the worse until after several days, or even at a later period. The change may be quite abrupt. The temperature rises to 104° or 106° (40°–41° C.). There are great uneasiness, delirium, and sometimes also signs of motor irritation, such as general convulsions, or tonic spasm of the extremities, or trismus. The face grows pale and cyanotic, the pulse small and extremely rapid. With slight interruptions, the temperature continues to rise, and attains 107·5° to 109·5° (42°–43° C.). This great rise is most apt to occur just before death, and there may be a still further increase of temperature after death occurs. As has been implied, the termination is usually unfavorable. It is only in exceptional cases that recovery takes place.

It has been stated that cerebral rheumatism attacks mainly hard drinkers and other individuals whose nervous system has been previously impaired; but our own experience does not confirm this view. No case is absolutely secure from the occurrence of hyperpyrexia; but it is a very rare phenomenon, occurring perhaps once in several hundred cases. On post-mortem examination, the brain seldom shows any change in these cases of cerebral rheumatism. We are therefore obliged to regard the condition as the result of an unusually severe infection, affecting chiefly the intellectual, motor, and thermal centers. Cases have also been reported where there have been actual anatomical lesions of the brain—in particular, purulent meningitis. Probably, however, most of these cases were falsely diagnosticated, the observer having confounded articular rheumatism with epidemic meningitis, pyæmia, and similar diseases. Of course, if there be endocarditis, cerebral embolism is possible. Mental derangements deserve a brief mention. They rarely occur during the course of the disease, but are somewhat more frequent after it has terminated. We may have either melancholia attended with marked excitement or anxiety, or a more general insanity. The prognosis is usually favorable.

6. Other Viscera.—Other parts of the body than the heart, the serous membranes, and the brain, are seldom much affected in articular rheumatism. Lobar pneumonia occurs only in especially severe cases, but in such it may attain quite a considerable development and occasion great dyspnœa. It usually requires quite a long while to recover from it. Lobular inhalation-pneumonia may also occur in severe cases. Acute nephritis has certainly sometimes occurred, but it is extremely rare. The spleen may be somewhat swollen, but it is not the rule to find a splenic tumor, such as is present in other acute infectious diseases.

7. General Symptoms.—In many cases the general condition of the patient is but little affected, but in others the disease seems to exert a peculiar influence upon the constitution. This may show itself in a striking anæmia; and we have observed this repeatedly where there was no cardiac complication. Another much more dangerous but extremely rare complication has already been briefly referred to, namely, the occurrence of a general hæmorrhagic diathesis. This is almost always associated with high fever and great prostration, and is usually fatal.

Course, Duration, and Prognosis.—Acute articular rheumatism may be described as generally a benign disease, for it usually terminates in recovery. It is only in a very few cases that an unfavorable termination takes place immediately, whether as the result of pericarditis or other severe cardiac complication, or from hyperpyrexia, or the development of a general hæmorrhagic diathesis. The entire duration of the disease varies greatly according to its severity. There are mild cases, which terminate in a few days; and, on the other hand, the disease may be very tedious, lasting for weeks and months, and finally merging into chronic articular rheumatism. Quite often the violent symptoms experienced at first disappear quite rapidly, but only to be replaced by such milder ones as pain and stiffness of the joints—these latter persisting for a long while. It is a general rule, that the severity and persistency of the case correspond with the number of joints affected; but to this rule there are numerous exceptions. The disease may persist with great obstinacy in a single joint. It need hardly be said that the duration of the disease is greatly modified by the occurrence of complications, cardiac or otherwise, and such sequelæ as muscular atrophy, anchylosis of the joints, or chorea. The most important of all sequelæ is cardiac disease, and this must always be considered in giving a prognosis; for, although the disease as such does in most instances terminate in recovery, yet too often it gives rise to a tedious and usually incurable disease of the heart. It is, however, true that complete recovery from the acute endocarditis seen in articular rheumatism is possible, but in a large majority of cases recovery is not complete, and the acute passes into a chronic endocarditis. In these cases the cardiac symptoms may be developed directly, so that the patient at once begins to complain of palpitation and shortness of breath; or he may seem to regain his health completely, and a murmur which the physician alone can detect may be the only sign of the incurable injury which the body has suffered. The patient may feel perfectly well for years after, and then at last begin to suffer from the failure of compensation (see page 283 *et seq.*).

Diagnosis.—Most cases of articular rheumatism can be easily recognized, for the acute occurrence of pain and swelling in several joints is sufficiently characteristic of the disease. It should nevertheless be borne in mind that articular swelling may also take place in the course of other diseases, and that mistakes in diagnosis are by no means impossible. Where there are grave constitutional symptoms with fever from the start, we should not forget the possibility of pyæmia, or of acute osteomyelitis, since these affections occasionally give rise to the swelling of several joints. In such cases, however, careful attention to the further course of the disease will generally enable us to see that we can not be dealing with a simple acute articular rheumatism. Again, after childbirth there may be swelling of the joints, of septic origin, and therefore entirely independent of any genuine rheumatic infection.

If a single joint be attacked, the diagnosis of articular rheumatism must be made with extreme caution. These monarticular inflammations often prove to be something entirely different, namely, fungous disease of the joint, or an osteomyelitis. The arthritis which follows gonorrhœa is also sometimes monarticular (affecting especially the knee-joint), or at any rate it is confined to the lower extremities; and, in conclusion, it should be stated that it is not very rare to observe pain and swelling in various muscles and joints at the commencement of the secondary stage of syphilis, simulating an acute articular rheumatism.

Sometimes the diagnosis is doubtful in those cases which present cutaneous ecchymoses (purpura and peliosis) and erythema nodosum, because we may be unable to determine which should be regarded as the primary symptoms and which the secondary; but in these cases, after all, it is often merely a question of nomenclature, and the best way is to follow the rule that the greater should include the less.

Genuine gout (*q. v.*) can usually be readily diagnosticated from articular rheumatism by its localization in the toe, and by the gastric and other symptoms which attend it.

Finally, we must mention that acute multiple neuritis (*q. v.*), beginning with fever and severe pain in the extremities, may sometimes, if we are not sufficiently careful, be mistaken for polyarthritis.

Treatment.—Acute articular rheumatism is one of the few diseases for which we possess an undoubtedly specific and universally accepted remedy. Kolbe suggested its use, and since 1876 it has been largely employed upon the recommendation of Stricker, Buss, and others, for articular rheumatism. This remedy is salicylic acid. Although this medicine does not in all cases produce its surprisingly favorable results with equal rapidity and completeness, yet it almost invariably does produce a decided and beneficial effect upon the disease. We might even say that this influence is so constant that where salicylic acid proves entirely inefficient in a fresh case, such failure throws doubt upon the correctness of the diagnosis. Thus, where there is monarticular arthritis dependent upon some local cause, the remedy has hardly any beneficial influence. The same is true with regard to affections of the joints connected with gonorrhœa, pyæmia, and similar troubles. In genuine acute articular rheumatism, on the other hand, the salicylic-acid treatment is so superior to any other that it is the first duty of the physician in every case to give this remedy a fair trial.

There are but two preparations of salicylic acid used in rheumatism—the pure acid and its sodium salt, salicylate of sodium. Each of these two remedies has its peculiar advantages, but the specific influence of each is about the same. Salicylic acid should never be prescribed in solution, but always in capsules, usually containing ten grains (grm. 0·50). In this way the salicylic acid can be taken by almost any patient quite easily, especially if a little water or milk be drunk after each dose. Adults should receive ten grains every hour until about one or two drachms (grm. 5–8) have been administered. Usually there will by this time be a very decided abatement of the articular pain and swelling, while on the other hand there will also usually be such toxic "salicylic symptoms" (*vide infra*) as to forbid its further use. The salicylate of sodium is best exhibited in single large doses of a drachm to a drachm and a half (grm. 4–6), each dose being given with about an ounce (grm. 20–30) of peppermint water. The quite disagreeable taste of the medicine is only exaggerated by the addition of such things as syrup or fluid extract of licorice, added for the sake of elegance; but the simple solution in peppermint water is quite well taken, at least by most patients. It is a very good plan to give salicylate of sodium in a glass of Hungarian wine, or in strong black coffee without sugar. The advantage of the salicylate over the pure acid consists in its being possible to give a larger dose at one time, so that it need not be taken more than two or three times a day. In general, the amount given in twenty-four hours should not exceed two and a half drachms (grm. 10); one and a half to two drachms (grm. 6–8) may suffice. For children the dose is, of course, smaller, say five grains of salicylic acid (grm. 0·30), or half a drachm to a drachm (grm. 2–4) of the sodium salt.

Which of these two preparations shall be employed is, as has been stated, of little consequence. We ourselves usually prescribe, at first, capsules of salicylic acid to be taken hourly, as being most agreeable to the patient; but if our first visit be made in the evening, we prescribe a single large dose of a drachm to a drachm and a half (grm. 4–6) of salicylate of sodium, so that the patient may not be disturbed every hour through the night in order to take medicine. It is often possible to give the two remedies in alternation. This is a good way later on in the disease, when the patient has already acquired a distaste for the medicine. In such cases also it may be desirable to give the salicylate of sodium as an enema. About two and a half drachms should be given, in two ounces of water (grm. 10 and 60). There is no doubt that the specific effects can be obtained in this manner.

The benign influence of this remedy upon the disease is apparent in many fresh cases as early as ten to eighteen hours after treatment begins; and it is often astonishing to see how soon a patient, who before lay helpless and complaining, becomes free from pain and able to move his limbs. It must be confessed, however, that, apart from its taste, salicylic acid may produce disagreeable incidental effects. In the first place, there may be nausea with epigastric distress, and even vomiting. Tinnitus aurium may be exceedingly troublesome, and may be attended with marked vertigo. In somewhat rarer instances the mind is especially affected. Young girls in particular are often peculiarly excited; but the frame of mind is, however, in general a cheerful one. After large doses there may be an actual "salicylic delirium." It should also be said that respiration may be affected, becoming very deep and rapid (salicylic dyspnœa). All these incidental effects, and particularly the nausea and ringing in the ears, render difficult the employment of the remedy in those large doses which alone are of any benefit. This is the more unfortunate, as it is often very desirable to employ salicylic acid persistently.

Although it is not exceptional to have the symptoms almost entirely vanish at the end of one or two days, yet it is only in the minority of cases that the entire process ends with this release from pain. There is very often, sooner or later, a relapse, with fresh pain or even fresh swelling in one or more joints. It has been recommended to continue the salicylic acid in smaller doses for some time, in order to avert such relapses; but of late we have abandoned this method, for the reason that these small doses do not prevent the return of the disorder, but are calculated to give the patient a strong dislike to the remedy, and lessen his confidence in it. We therefore recommend to stop the medicine entirely as soon as the pain ceases, and to guard the patient as much as possible from relapses by preventing his catching cold (*vide infra*). If there be fresh pain, we should at once resume the acid or its salt in large doses, and thus we will very frequently be able to cut short the relapse at once.

[One other salicylic compound deserves mention—the oil of wintergreen; this has been used largely by Kinnicutt, who finds it efficacious, easy to take, and not likely to produce the unpleasant symptoms which sometimes follow the compounds in more general use. It is given in doses of ♏ x–xv every two hours, either in milk, on sugar, or in capsules.

The salicylic treatment markedly diminishes the pain and fever, shortens the time spent in bed by four or five days, does not shorten the time spent in hospital, and seems to have little or no influence on the cardiac complications. The full alkaline treatment does not curtail the pain and fever in the same degree, but does seem to afford some protection against the heart affection, and to shorten the stay in hospital several days.

A combination of the salicylic and alkaline treatments seems therefore rational, and it has been adopted by the editor for some years. Citrate of potash is given in sufficient doses to produce and maintain alkalinity of the urine. The salicylic compound is given at first in full doses, which are diminished in size and frequency with the control of the articular symptoms, but are not omitted entirely until recovery is practically complete. The editor is inclined to believe that relapse is less common if the system be kept somewhat under the influence of the drug. Salicin is often perfectly well borne when the sodium salt causes gastric irritability, and the oil of wintergreen is another form which has advantages in some cases.]

Despite the admirable qualities of salicylic acid, it must be confessed that we can not always bring about a rapid and complete cure of the disease by this remedy. There are cases where, although at first evident improvement follows its use, relapses continually recur, or the disease fastens itself obstinately in single joints. In such, the continued use of salicylic acid proves almost unavailing, and indeed the patient can hardly be persuaded to take it. In these cases, and also when the salicylic preparations are ill borne or cause unpleasant attendant symptoms, antipyrine is the best substitute. Many physicians therefore often prescribe it from the first in place of salicylic acid, although we think the action of the latter upon acute articular rheumatism is greater than that of antipyrine. We should give antipyrine in doses of fifteen grains (grm. 1) several times a day in water or wine. Its attendant symptoms (sweating, nausea, and sometimes an eruption like measles) are usually without special significance, and its influence on the articular pains is often so favorable that the remedy is of much value in the treatment of protracted rheumatism. Antifebrine, in doses of four to seven grains (grm. 0·25–0·50), seems also to have a certain favorable action in joint affections, but it is distinctly inferior to antipyrine. Sahli has recommended salol (salicylic acid, phenyl ether), which deserves more confidence; it is given in fifteen-grain (grm. 1) powders several times a day (five to eight powders a day). We are often compelled, especially in articular rheumatism which has frequent relapses, to try in turn the different remedies mentioned.

There are still other internal remedies which should be tried, but their effects are rarely satisfactory. Most important among these are iodide of potassium and the preparations of colchicum (tincture of colchicum, twenty-five to forty drops several times a day). Numerous other remedies have been recommended, and they were formerly largely employed, but at present they are almost abandoned. Among these are large doses of the alkalies (such as bicarbonate of soda), trimethylamine, veratrine, and quinine. Local treatment of the diseased joints is far more important and effective in such cases. Properly executed massage deserves special mention, as its effects are often very brilliant. Electricity also may have a beneficial effect, particularly the galvanic current. We would caution against the too early use of warm baths, as these often aggravate the pain instead of mitigating it. Steam baths are sometimes very beneficial, but they may also do harm, and they should therefore be given only when the acute inflammatory symptoms have entirely vanished, leaving behind only stiffness and tenderness in the joints.

The application of an ice-bag in genuine articular rheumatism is seldom necessary, but it may sometimes be desirable where there are violent and obstinate symptoms of acute inflammation. Warm, moist applications are useless, if not harmful, in acute cases. In the advanced stages of subacute cases a wet pack may afford some relief. Painting the skin over the joints with tincture of iodine produces no effect in acute cases, and even in the chronic ones it is probably mainly a subjective remedy. Some observers report that injections of carbolic acid beneath the skin of the affected joints greatly relieve the pain. A Pravaz's syringeful (thirteen minims) of a one-per-cent. solution may be injected one to three times a day. We have had no personal experience with this remedy. In all severe cases careful attention should be given from the start to maintaining a correct position of the diseased joints, because of the possibility of anchylosis. Before salicylic acid was introduced, "the treatment of articular rheumatism with splints" was largely and very advantageously employed. The use of salicylic acid has greatly diminished the necessity of such procedure, but even now it is sometimes required. It is often possible to give the patient great relief by applying a suitable pasteboard or wooden splint to an affected extremity.

General hygienic and dietetic treatment should not be undervalued. An equable temperature should be carefully maintained in the sick-room, inasmuch as cold, or draughts, or moisture have very often been found to exert an evil influence upon the disease and excite fresh pain. The patient should therefore be kept warm, and it is sometimes advantageous to wrap up the affected joints in cotton batting. It is of great importance that even in the mildest cases the patient should be strictly confined to bed, and he should by no means get up too soon. If possible, we keep our own patients in bed for a week after the pain has ceased. Getting up too early will very often bring on a relapse. With regard to diet, milk is the best food. We may also allow soup, eggs, and a little meat. In France, great weight is laid upon an exclusive milk diet; but this would seem to us an extreme view.

We do not need to speak at length about the treatment of the complications and sequelæ, since we should merely repeat what has already been said in the appropriate chapters of this work. There has been much said on both sides as to the influence of salicylic acid in preventing complications, particularly cardiac complications. This much is certain, that cardiac complications are not absolutely prevented by the salicylic treatment, and that they too frequently occur while it is being employed; but we do believe that this treatment decidedly shortens the course of the disease as a whole, in many instances, and thus lessens the liability to endocarditis. If, however, a cardiac complication have made its appearance, salicylic acid does not apparently exert any appreciable influence upon it. Another important question is in regard to the efficiency of salicylic acid in the graver forms of articular rheumatism, particularly in cerebral rheumatism. It may be stated, in the first place, that in Leipsic cerebral rheumatism has apparently become much less frequent since the salicylic treatment was introduced. At any rate, not a single case of hyperpyrexia occurred in the clinique in that city out of many hundred cases treated, where the salicylic acid was properly employed from the first. We had an opportunity to observe a case in which hyperpyretic symptoms had already appeared when we first saw it, and which had not been treated with salicylic acid. Here large doses of that remedy produced no effect. We should nevertheless be inclined to employ it, first of all, in such cases; and the energetic use of cool baths would probably be the most speedy way of modifying the dangerously high temperature. Stimulants, in particular camphor, are also required in these severe cases.

In the severe hæmorrhagic varieties of rheumatism we should also give salicylic acid a trial. The milder hæmorrhagic cases (hæmorrhagic urticaria) do well under ordinary methods of treatment.

If the acute affection merge into a chronic condition of stiffness and swelling of certain joints, such as the wrist or shoulder, we must employ the same remedies as in chronic articular rheumatism. Massage furnishes the best results. Warm baths may also be ordered in such cases (see the following chapter). The patient might be sent to Teplitz or Wiesbaden. The subsequent muscular atrophies and paralyses recover most rapidly under electricity.

Prophylaxis requires, first of all, that one should avoid cold or wet, and other "rheumatogenous influences." Persons who have already had one attack of articular rheumatism must be especially careful, inasmuch as they are more than ever liable to the disease, as has already been said. It is not inconsistent, however, with the exercise of due caution, to endeavor to lessen the sensitiveness of the skin by such procedures as cold sponging, followed by friction.

CHAPTER II.

CHRONIC ARTICULAR RHEUMATISM (CHRONIC POLYARTHRITIS) AND ARTHRITIS DEFORMANS.

Ætiology.—The two diseases known as "chronic articular rheumatism" and "arthritis deformans" are considered together here, because it is impossible to draw a sharp distinction between them. It is, indeed, not unlikely that the above names are sometimes applied to diseases which differ essentially from each other; but as we do not yet understand the nature or the ætiology of many chronic diseases of the joints, we must provisionally be guided by the external changes they produce. We shall therefore embrace all chronic inflammatory processes affecting the joints under the name of chronic arthritis. Traumatic arthritis it is not intended to include, much less those chronic affections of the joints which are evidently of tubercular origin, and which have ordinarily been termed fungous arthritis. These belong to the domain of surgery. We would also exclude chronic syphilitic diseases of the joints, about which, indeed, there is still less known than about the tubercular affections; besides, they are rare. Chronic articular affections from gonorrhœa are probably more frequent.

The ætiology of those cases of chronic arthritis where the disease is a direct sequel of acute articular rheumatism is evident enough. It is hardly possible to doubt that the same specific poison which excited the acute arthritis maintains possession of the joints, and produces the chronic inflammatory changes. Cases of this sort especially deserve the name of chronic articular rheumatism. They are not very infrequent, and may be of slight or great severity. The worst cases produce macroscopic changes which fully justify the other appellation of arthritis deformans.

It is also possible that many cases which are chronic from the start have the same ætiological origin—that is, are due to the same pathogenic agents. This might be inferred from analogy with numerous other diseases, and it is rendered still more probable by the fact that the same exciting causes which promote acute articular rheumatism often play a conspicuous part in chronic arthritis. Such causes are exposure to cold and wet, and working in cold or draughty places, or dwelling in newly built and damp houses. This explains why those who follow certain callings—for instance, washerwomen—are more apt to suffer from the disease than others, and why arthritis deformans has, with some justice, been called a disease of the poor, in contrast with the gout of the wealthy. Many of the laity, and even some physicians, believe that gout and arthritis deformans are in some way related, but this view is erroneous.

It is very questionable whether all cases of chronic multiple arthritis are referable to the causes already enumerated. Such other influences, however, as are concerned in its production are not at all understood. Various authorities have maintained that arthritis deformans is the result of a primary disease of the nervous centers, and in particular of the spinal cord. We regard this statement as entirely unwarranted. It originated at a time when there was a tendency to ascribe all sorts of ills to disease of the "trophic centers," but there is no doubt that this tendency was carried very much too far. We may state in this connection that a careful microscopic examination of the spinal cord in one case of very severe arthritis deformans yielded an entirely negative result.

[Some striking cases are reported by Blake, which go to show that the arthritic changes may set up chronic, though slight, suppuration, in analogy, perhaps, with the synovitis of gonorrhœa. Careful examination should consequently be made of all the mucous membranes, especially in cases which seem to have no connection with true rheumatism.]

Predisposing Influences.—Chronic arthritis is mainly a disease of advanced years. Certain monarticular varieties, about whose ætiology, it must be confessed, we know little as yet, have been termed arthritis senilis—in particular the *malum coxæ senile.* Even the common and, in a certain sense, typical form of arthritis deformans (*vide infra*) is not apt to occur in people under thirty-five years of age. This rule, however, has exceptions, and we have ourselves seen a few perfectly characteristic cases of arthritis deformans in children between ten and fifteen years of age. Women are much oftener attacked than men. It is often said that trouble, anxiety, and other emotional influences favor the outbreak of the disease; but the proof of this is lacking. The disease does not often seem to be hereditary.

Pathology.—The process is described as simple chronic arthritis so long as it is confined mainly to the synovial membrane of the joint and the periarticular connective tissue. These parts often undergo decided inflammatory thickening; the synovial membrane becomes cloudy; and the amount of synovial fluid is more or less increased—that is, we have chronic dropsy of the joint. Sometimes different parts of the synovial membrane are connected by adhesions, which considerably interfere with the movements of the joint. There may even be complete anchylosis: for example, in the shoulder or knee.

Chronic synovitis may pass imperceptibly into arthritis deformans. In this, not only the capsules of the joint, but the articular cartilages and the articular extremities of the bones, are so much affected as to produce the most striking deformity. These changes almost always originate in the articular cartilages. The cartilage is roughened and worn away; its free borders and surfaces undergo proliferation and then disintegration, or become polished on the surface, while deeper in the newly formed layers of cartilage undergo ossification. The underlying bone undergoes inflammation and degeneration. Sometimes, also, the periosteum near the joints undergoes ossific periostitis. On microscopic examination, we find fibrous disintegration of the matrix of the cartilage, and proliferation and subdivision of the cartilage-cells, at the same time that there is destruction of the newly formed cells by simple or fatty degeneration. Analogous processes of proliferation and destruction also affect the bony structures. The synovial membrane is invariably affected in cases of any severity. Usually the most striking change is a great proliferation of the joint villi, which may cover the walls of the cavity like great fringes.

Of course the normal structure of the joint is at last completely destroyed by these various processes. The articular extremities of the bones waste away more and more, and take new relative positions, as the parts which impinge upon each other are worn away. Externally, the joint usually becomes larger and larger; and this is the more evident because the surrounding muscles undergo great atrophy. Often there is no synovial fluid whatever (*arthritis sicca*), but sometimes there is a considerable effusion—for instance, in the knee-joint.

Clinical History.—The symptoms of chronic arthritis are usually quite simple and uniform. They are almost exclusively referable to the local disturbances and their results.

Except in the cases which are preceded by acute articular rheumatism, the disease usually begins quite gradually and insidiously. The first subjective symptoms are stiffness and pain in the joints, the latter being aggravated by pressure or movement. The stiffness is most noticeable when the joint has remained quiet for some time previous, and is therefore ordinarily greatest on waking up in the morning. The pain often shoots from the joints upward and downward, and is of a burning character, or less often neuralgic. Even in advanced cases the pain usually occurs only when the affected joints are moved, although then it may be very severe. When the body is entirely at rest there is little or no pain. Motion is impaired very early. This is due at first to the pain, and to a certain reflex inhibition and ataxia of the muscles; to which are later added the purely mechanical hindrances and the ever-increasing atrophy of the muscles.

The objective changes in the affected joints begin to appear soon after the symptoms just mentioned, at least in cases of any severity. The joints seem enlarged and thickened. If we attempt to move them, we not only cause pain and meet with mechanical obstruction, but we may hear and feel the cracking and grating produced by the rubbing of the denuded and uneven surfaces upon each other. This is often noticed by the patient himself. As the disease gradually progresses, there are usually developed certain characteristic deformities, which are apt to be most strikingly exhibited in the hands (see Fig. 117). The metacarpo-phalangeal joints are thickened and swollen, and are made all the more prominent because the interossei upon the back of the hand are atrophied. The bases of the first phalanges are directed obliquely toward one side, so that the fingers assume more and more the appearance of subluxation. They are bent over toward the back of the hand, and are also displaced toward the ulnar side, so that they often actually come to rest one upon the other. The palm of the hand is frequently deeply hollowed out. Often the phalangeal joints are also distorted, so that, for example, there will be an obtuse angle on the dorsal surface of the fingers between the first and second phalanx, while the terminal phalanx is apt to be flexed, although the second phalanx preserves a position of extension. Despite these changes, many patients, if only the thumb remains tolerably movable, are able to use their hands for quite delicate work, although at the expense of much time and effort. The feet exhibit analogous deformities, but seldom to the same extent as the hands. The knees and elbows are likewise enlarged. At the hip-joint, subluxation is not infrequent, the head of the femur slipping upward. The motion of the shoulder-joint, and as a consequence the use of the arms, is gradually more and more impaired. If the joints of the lower extremities are affected, of course it becomes painful and difficult either to get up or to walk. It may finally be necessary for the patient to have the help of some other person, or of crutches.

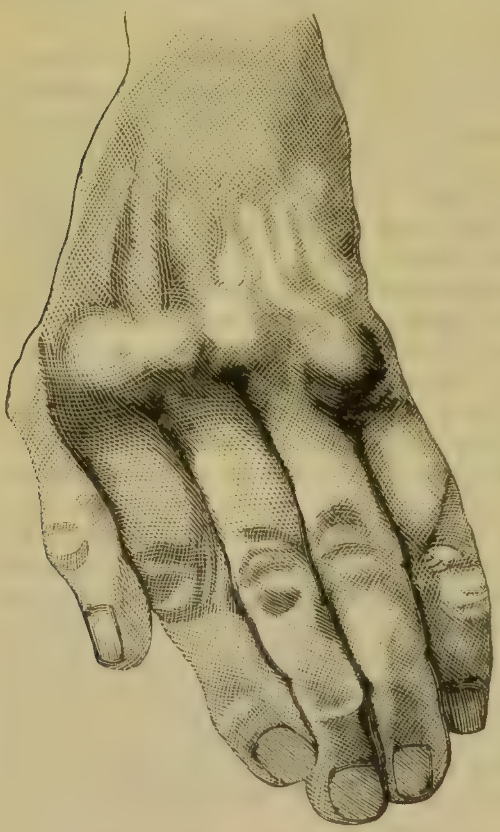

FIG. 117.—Appearance of the hand in a case of protracted arthritis deformans.

There are monarticular and polyarticular forms of the disease—the name indicating that one or several joints are affected. The monarticular form is usually regarded as a surgical trouble, and is most often located in the hip-joint (*malum coxæ senile*), or more rarely in the knee- and shoulder-joints. The polyarticular form is the characteristic one. In most of the typical cases it begins in the small joints of the hand and fingers. At a later period the larger joints are also invaded, one after the other, the invasion taking place symmetrically on both sides of the body, although the disturbance is not infrequently greater on one side than on the other. In severe cases the joints of the spinal column are also involved. This impairs particularly the movement of the head. The articulation of the lower jaw is usually very little affected, if at all.

In less frequent instances the arthritis is confined principally to the lower extremities, while the upper escape intact for a long while, or even permanently. It is very possible that such cases often have a different ætiology from ordinary arthritis deformans; and the same is true of the cases which are confined mainly to the vertebral column, and are termed spondylitis deformans. A remarkable and, as it seems to us, unique disorder may be mentioned in passing. It leads very gradually and painlessly to a complete anchylosis of the entire spinal column and the hip-joints, so that head, trunk, and thighs are firmly united and completely stiffened, while all the other joints retain their normal mobility. It need scarcely be said that this necessarily causes a peculiar modification of the carriage and gait of the sufferer. We have ourselves seen two cases of this peculiar affection which resembled each other very closely.

There is hardly any affection of parts of the body other than the joints in arthritis deformans. The muscles should be excepted, for they always undergo that muscular atrophy which we have already described (*vide* page 905) as the result of joint disease. This atrophy is most marked in the interossei, the shoulder muscles, and in the muscles of the calf and thigh. Sometimes the skin over the wrist and other affected joints appears peculiarly wrinkled and flabby. The internal organs almost always perform their functions in a perfectly normal manner. Appetite and digestion remain good, although there is often some tendency to constipation. Rarely there is valvular disease of the heart, but usually only in such cases as originate in an acute articular rheumatism. Once in a while it is seen in cases chronic from the start. This last fact is not without interest from an ætiological point of view. Sometimes there are certain nervous symptoms to be observed, such as headache, congestion, or mental depression, but these are probably not the direct result of the disease, but arise indirectly in a way to be easily imagined.

General Course of the Disease.—Arthritis deformans is an extremely chronic trouble. It may last even ten or twenty years, or more. Sometimes there is an apparent arrest of the process extending over months, or even longer. Sometimes the progress of the disease is marked by remissions and exacerbations, affecting either the general or the local manifestations. In general, however, the disease continually advances.

The prognosis is therefore unfavorable. Recovery, if it ever occurs, is extremely rare, and is possible only in the early stages. For the encouragement of the patient, it may be said that under proper care and treatment the disease often runs so gradual a course that the general condition remains at least bearable for a very long while, although there may be considerable local disturbance. The disease is not directly dangerous to life. The eventual fatal termination ensues either from general debility, or because of some intercurrent disease.

The prognosis is somewhat more favorable in the milder cases of "chronic articular rheumatism," where the anatomical changes are less severe, and are completely limited to the synovial membrane. Even here, however, recovery is by no means frequent, and it is always to be feared that grave deformities of the joints will gradually be developed.

Treatment.—With regard to regimen, it is requisite in the first place to avoid all unfavorable external influences. If possible, the dwelling should be dry and warm; and it may often seem advisable to make a change of climate. The patient must dress warmly, without, however, undermining his powers of resistance too much, as he will be in danger of doing. The diet must be abundant and nutritious.

Internal remedies may be tried, with the hope of modifying the disease, but our chief reliance must be upon local treatment of the joints. Among internal remedies, the most important are iodine and arsenic. Iodine may be given in the form of tincture (a few drops in mucilage several times a day), or a better form is in combination with potassium. As yet, we ourselves have not seen any great benefit from iodine, but we have in repeated instances witnessed a quite striking result from the use of arsenic. It is best administered in pills containing one thirtieth to one fifteenth of a grain (grm. 0·002–0·004) of arsenious acid, one pill two or three times a day. If this remedy prove beneficial, it must be continued for at least months, perhaps with occasional brief intermissions. Salicylic acid and antipyrine have no permanent effect, and are useful only when there is an acute exacerbation of the disease. The preparations of colchicum may be tried, but they will seldom be found efficient. Iron, quinine, and cod-liver oil are sometimes indicated by the general condition.

First among local methods of treatment comes massage, although the good it accomplishes is, of course, apt to be evanescent. It will, however, do much to hasten the absorption of inflammatory exudations, and also to loosen up the joints, invigorate the muscles, and improve the general health. The Swedish movement cure will be found of great benefit in all cases if begun early and methodically persevered in. It preserves the mobility of the joints as long as anything can. Electricity also has a palliative influence. The galvanic current is applied to the affected joints, and the faradic current to the atrophied muscles.

Baths are universally employed in chronic arthritis. Their value should not be overestimated, but it is, notwithstanding, undeniable in many cases. Simple warm baths, or salt baths (two to ten pounds of salt for each bath), are practicable in almost any household. As health-resorts in arthritis deformans, experience shows the following to be most desirable: The different warm baths, such as Teplitz, Wildbad, Ragatz, and Baden in Switzerland; the warm chloride-of-sodium baths in Wiesbaden; the acidulated baths of Oeynhausen and Nauheim; and the mud baths of Elster, Marienbad, Franzensbad, and Schmiedeberg. Steam baths are admissible only in the early stages of the disease, and for patients whose general condition is still vigorous. Even then they should be employed cautiously.

[The mineral springs within the limits of our own country chiefly to be recommended are Sharon and Richfield, in New York State, the Sulphur Springs of Virginia, and the Hot Springs of Arkansas. At the two former, particularly, there is every provision for comfort as well as for the use of the waters.]

We have repeatedly seen quite excellent results follow the employment of hot sand-baths. These also can be easily used at home, particularly if applied merely to the hands or feet. They are employed more elaborately in Köstritz and Blasewitz. These hot sand-baths seem to do good, not only from the temperature, but also from the uniform and persistent compression which they exert.

Stimulating or narcotic remedies may be rubbed into the joints, but they are beneficial only because of the massage which accompanies their employment. In practice it is not always possible to omit their use. The application of tincture of iodine is usually entirely without effect. As to morphine and other narcotics, the disease is so chronic that it is desirable to employ them as little as possible. A considerable number of those who suffer from chronic arthritis become opium-eaters.

We may say, therefore, that the use of the various remedies which have been suggested will enable us to oppose some obstacles to the progress of the disease. Persistent treatment will, in many cases, be rewarded by at least temporary improvement.

CHAPTER III.

ACUTE AND CHRONIC MUSCULAR RHEUMATISM.

(*Myositis, or Myalgia, Rheumatica.*)

Definition and Ætiology.—Certain acute affections may originate primarily in the muscles. These are to all appearance inflammatory in their character, and not infrequently result from taking cold, or other causes similar to those which produce acute articular rheumatism. These affections are classed as "acute muscular rheumatism," or rheumatic myositis. It is possible that this disease is also an infectious one, but the question remains entirely undecided. The analogy which this trouble bears to acute articular rheumatism is not complete. It is seldom that the two processes are seen in combination; and, furthermore, acute myositis is not "polymuscular," but is usually confined to one muscle, or to a single group of muscles; and it is never followed by acute endocarditis. The two diseases, therefore, are alike only in certain symptoms (pain and impairment of motion), and in the fact that they are often, although not always, ascribable to wet or cold, and the like. There are many cases where pain suddenly occurs in the muscles ("myalgia") without any attendant objective change. These cases can not be called genuine acute myositis. Indeed, it is sometimes diffcult to know how to regard them. In practice they are often termed muscular rheumatism, especially when they are referable to exposure; and it is possible that many such cases are really a very mild form of the genuine inflammatory disease. On the other hand, however, there must often be some different process going on. Thus, traumatic pain in the muscles is the result of some excessive strain, and in many instances is apparently due to laceration of some of the muscular fibers. This is generally occasioned by too violent muscular exertion. Any physician who sees many patients from the laboring classes meets with an abundance of cases of this sort.*

The limitations of acute muscular rheumatism are obscure; but still more so are those of "chronic muscular rheumatism." This disease is also a frequent one, and only imperfectly understood. It does not bear a close analogy to chronic articular rheumatism, except in this point, that chronic muscular rheumatism seems to be quite often occasioned by meteorological influences. While the anatomical changes in chronic articular rheumatism are almost always striking, similar lesions are very exceptional in chronic muscular rheumatism. On the contrary, the name is usually applied to cases where there is pain in various muscles all over the body, but where there is no discoverable objective disturbance. Older authorities used to speak of "rheumatic induration" of the muscles, but this or any other actual anatomical change is very exceptional.

These facts justify a doubt as to whether all cases of chronic muscular rheumatism actually deserve their name. It is certainly quite appropriate in those not infrequent cases which are due to "rheumatogenous influences," and which are so evidently aggravated upon every exposure to cold, or every period of bad weather, that the patient often asserts that he carries in his legs the best of thermometers. Such is the "old rheumatism" of those who have passed a large part of their lives in the open air, regardless of wind or weather. There are other cases, the character of which is different. In them the muscular pain is associated with a general neurasthenic condition, or with corpulence (when it is, perhaps, the result of circulatory disturbance), or possibly with chronic poisoning. An important instance is the "rheumatic pain" sometimes complained of by topers, which we are inclined to ascribe not to changes in the muscles but to nutritive disturbances of the nerves. For these and similar disorders there are no special names, and the practicing physician often terms them all "muscular rheumatism," a diagnosis with which the patient is usually quite contented.

Clinical History.—Genuine acute muscular rheumatism is usually, as has been said, limited to some one definite group of muscles. The affected muscles often seem somewhat swollen and infiltrated, are very sensitive to pressure, and, if not quite useless, are nearly so, greatly impairing the motion of the corresponding member of the body. All these symptoms are best illustrated in acute myositis of the deltoid (omalgia). The whole shoulder is swollen, the muscle is very painful, and the upper arm is almost incapable of voluntary motion, although, if caution be exercised, passive movement can be made without causing any pain.

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* Some time ago I saw an organ-player who had to work the pedals for many hours a day; he had an extremely painful affection of the lower extremities associated with swelling, which could be regarded only as an acute myositis.

The various forms of acute muscular rheumatism have received names descriptive of the locality of the affection. We have:

1. *Omalgia*, as already mentioned.

2. Acute rheumatic myositis of the cervical muscles, *myalgia cervicalis*, or rheumatic torticollis. Here the muscles of the back of the neck and throat are very painful. The head is usually held to one side, and, in severe cases, is almost perfectly immovable.

3. *Lumbago*, or *myalgia lumbalis*. This is the most frequent form of acute muscular rheumatism. The common people in Germany have termed it "witch's shot" (*Hexenschuss*), on account of its sudden onset. The entire lumbar region is very sensitive; and any motion of the trunk, such as stooping or turning, is extremely difficult and painful. The disease is more frequent in men than in women. Certain persons seem to be especially predisposed to it. It should also be stated that lumbago is not always of a rheumatic character, but of traumatic origin, as from lifting a heavy weight, or from sudden stooping. [It is very common in the traumatic neuroses, where it was once regarded as indicative of spinal injury.—K.]

4. Rheumatism of the thoracic muscles, and particularly of the intercostals. This may cause great discomfort, as it renders breathing, coughing, and sneezing very painful. It is comparatively rare; and caution should be exercised in diagnosticating it to avoid confusion with pleurisy and periostitis of the ribs. Very often, also, thoracic disturbance is regarded as rheumatic when it is really traumatic, being the result of stretching or laceration of the fibers of the pectoral or other muscles.

5 Rheumatism of the head also, probably, belongs in this category, although the affection is seldom confined to the muscles of the scalp, but involves also the fasciæ, and may even be almost confined to them. It is not infrequently excited by exposure to cold. The pain is quite violent, and greatly increased by any movement of the scalp. Of course, the diagnosis requires the previous exclusion of the various forms of headache described on pages 530 and 591.

The duration of acute muscular rheumatism is brief. Usually the pain abates in a few days; but a tendency to relapse persists for some time. In chronic muscular rheumatism there are usually no objective changes to be detected. The pain is seldom located permanently in any one place, but it is felt first here and then there. It is usually increased during bad weather, and is less severe when the weather is warm. The pains are often described as "wandering." Motion is seldom much impaired. Sometimes, however, there may be a certain stiffness of the muscles, most marked after a period of rest.

The diagnosis of chronic muscular rheumatism rests, therefore, mainly upon the rational signs. Hence it is often impossible to avoid the suspicion of malingering, particularly where certain applicants for hospital care are concerned. We should not, however, be too uncharitable, since without doubt there are cases where quite severe pain is felt, now in one set of muscles and now in another, without any anatomical basis for such pain being discoverable. Nor should we ever forget that other diseases may have pain for their first symptom. It is not at all exceptional for the lancinating pains of locomotor ataxia to be for a long time regarded as "lumbago." Lumbago may be confounded with insidiously developing diseases of the vertebræ, or with various hypogastric disorders (particularly in women). We should therefore never omit to make a careful physical examination.

Treatment.—Acute muscular rheumatism has this in common with acute articular rheumatism, that it is usually very favorably affected by salicylic acid. In cases of genuine acute rheumatic myositis the employment of this remedy in the manner already described, for twelve to twenty-four hours, will often give surprising relief. We also see good results from antipyrine. Local treatment of the affected muscles may also be followed by great and speedy improvement. Massage is particularly valuable. It is not infrequently the case that a single properly conducted massage will cause a violent lumbago or omalgia to disappear almost completely, and like favorable results are witnessed where there is traumatic pain of the muscles. Most of the external applications which are so frequently prescribed for rheumatism—such as spirits of camphor or chloroform liniment—accomplish less through the cutaneous irritation they produce than by the massage incident to their employment. Next in value comes electricity. Both the constant and the faradic current may be employed. Simple counterirritation by means of mustard poultices or hot compresses will often prove palliative, but it is less effective than the remedies previously mentioned. Great benefit often follows excessive perspiration. The best means to this end is a steam bath. This is so favorite a remedy that patients often take it of their own accord.

In chronic muscular rheumatism the benefit of salicylic acid and antipyrine is merely temporary, and therefore is to be sought, if at all, only when there is an acute exacerbation. Massage and electricity are more effective, and, if persevered in for some time, will often accomplish good results even in obstinate cases. Treatment by baths is frequently prescribed with advantage. Steam baths are often beneficial, but their use requires great caution where the patient is corpulent and has a tendency to congestion or cardiac failure. There is also value in mud baths, pine-needle baths, and in the baths given at Teplitz, Wiesbaden, and other places.

In many cases of chronic muscular rheumatism constitutional treatment is of great importance. Particularly where the patient is overfed, and intemperate in the use of alcohol, much benefit will often be accomplished by a proper regulation of the ingesta and the prescription of a sufficient amount of muscular exercise. Such patients may also be helped by a cautiously conducted cold-water treatment.

[In acute cases with localized pain I have found a thick flaxseed poultice, applied as hot as it can be borne, renewed once or twice, and followed by the application of a thick layer of cotton, useful.

A dry cup or two is also often productive of great relief. In chronic cases, plasters and the iodide of potash are often of benefit.

Muscular rheumatism is a common and often very troublesome affection in those whose occupation calls for decided muscular exertion. A muscle is strained, pain settles in and is apt to recur in the part; and, while the general health is sufficiently good, the man is compelled to remain idle. Quack advertisements dwell so much upon pain in the back as a symptom of Bright's disease, that we are frequently consulted by those who, suffering from muscular pain and soreness, think themselves the subjects of serious disease of the kidneys.]

APPENDIX.

ACUTE POLYMYOSITIS.

From some very recent observations (E. Wagner, Unverricht, and others) we have obtained knowledge of a disease which consists essentially of an acute inflammation of most, or even of all, the muscles of the body. The disease occurs chiefly in young or middle-aged persons. Without special cause they begin to have pains in the arms, legs, and trunk, which more or less rapidly result in a considerable disturbance of motion. The general disturbance is at first slight, but it increases later, especially if, as usually happens, there be fever. A marked œdematous swelling is soon very noticeable; it is first seen on the extensor side of the extremities, and later in the face and trunk. The œdema is stiff and painful, and it may be very considerable. As soon as the muscles of deglutition and respiration are affected the whole condition becomes distinctly worse. It becomes harder and harder to take food, and there is severe dyspnœa. Bronchitis and lobular pneumonia soon develop, which become the more distressing, since expectoration is more and more impaired, and finally becomes impossible. The spleen as a rule seems swollen. There is usually a marked tendency to sweating.

In the cases thus far published death always ensues after a few weeks, with symptoms of extreme dyspnœa and cyanosis. We do not yet know certainly whether there are also milder cases which recover.

In the cases observed thus far there was on examination a true acute inflammation of the muscles. Not only do the muscular fibers show all forms of degeneration and destruction, but also in the interstitial connective tissue we find true inflammatory foci (accumulation of nuclei about the vessels, etc.). In a case which we recently examined these changes were apparently to be found in all the muscles, even in the tongue, the ocular muscles, etc. In genuine polymyositis the peripheral nerves are found to be perfectly normal, but it is, of course, possible that on future investigation relations may be established between polymyositis and multiple neuritis (*vide supra*).

The diagnosis of polymyositis will probably soon be comparatively easy, when the disease becomes better known. The distinction from trichinosis must be the most difficult; here we must first of all consider the ætiology. Polymyositis is to be distinguished by the pronounced œdema from multiple neuritis, which in other respects closely resembles it.

Experience is still limited in regard to the treatment of the disease. We would first recommend the exhibition of salicylic preparations, antipyrine, and similar remedies. Toward the close of the disease narcotics usually become unavoidable.

CHAPTER IV.

RACHITIS.

(*Rickets.*)

Ætiology.—The first accurate description and the now universal name of "rachitis" (from ῥάχις, the spinal column) is to be ascribed to the Englishman Glisson, who published a comprehensive monograph upon this disease in 1650. It was his opinion that it first appeared in England in the beginning of the seventeenth century; and for this reason rachitis is still often called by Germans "the English disease."

Although the clinical and anatomical phenomena of rachitis have been often and accurately investigated since that time, its true cause still remains entirely unknown. It is certain only that its development is promoted by all unfavorable external circumstances affecting the nourishment and health of the child. It is therefore more frequent among the poor than the wealthy, in the damp and crowded quarters of large cities than in the country, and among artificially fed, and therefore weakly and anæmic children, than such as receive the mother's milk. Nevertheless, the essential cause of the disease is not to be sought among these various influences, for rickets undoubtedly does occur, although rarely, in children whose circumstances seem in every respect most favorable.

Guerin, Friedleben, E. Voit, Wagner, Baginsky, and many others have made very exhaustive experimental researches with regard to the development of rachitis. It has been found possible to produce certain changes in the bones of growing animals by giving them as little lime as possible in their ingesta, or by administering very large amounts of lactic acid, with the purpose of dissolving the calcium salts, or by giving small quantities of phosphorus. The changes thus caused have been, with more or less correctness, regarded as analogous to those of rachitis. These investigations are of great interest with regard to the physiology of bony structures in general, but in our opinion they throw little light upon the clinical question which here concerns us. It is indeed natural enough to suppose that rachitis in childhood may be due to an insufficient proportion of lime in the food; or to a defective absorption of the lime-salts, on account of intestinal catarrh; or to an abnormally abundant production of lactic acid, or even of carbonic acid, which may dissolve the lime-salts in the system; but every one of these theories is contradicted by the facts of experience; for it is wholly improper to assume that the food of children with rachitis contains less lime than the food of healthy children, and the hypothesis of a mere poverty in lime of the bony tissues is by no means adequate to explain the whole complicated rachitic process. In our opinion everything seems to indicate that some special, specific, ætiological factor is requisite for the development of rachitis. This factor, however, is as yet entirely unknown to us. The thought had occurred to many that the disease bears some relation to congenital syphilis; but this assumption has long since been proven to be entirely without foundation. It is also claimed that heredity plays an important part in rachitis. The proof of this is lacking. It is, however noteworthy that quite often several children of the same family are attacked by the disease.

Rickets is most common in children of two or three years old. According to Kassowitz, the disease usually begins in the first months of life, while the severe symptoms, of course, appear only at the age of two or three years. Congenital rachitic changes (fœtal rachitis) have been repeatedly observed, but the cases of so-called *rachitis tarda*, where the disease is said to develop in children of eight or ten years of age and over, are at least extremely rare.

Sex exercises no great influence upon the occurrence of the disease.

Pathology.—Rachitis consists in a peculiar disturbance of the processes connected with the growth of the bones. As a result of an increased absorption of the already formed bony tissue, and especially as a result of an insufficient or an almost wholly deficient deposition of lime-salts, the bones become or remain abnormally flexible and soft, so that they can easily be cut with a knife.

Upon minute examination, we find both the periosteum and the marrow much reddened and congested. If we try to detach the thickened periosteum from the bone, not infrequently a few bits of bone adhere to the membrane. The most striking changes, however, are exposed upon making a longitudinal section of the bone. They are located at the base of the epiphyses, because here is the place where the normal, and therefore the abnormal, processes of ossification are most active. Under normal circumstances, the epiphyseal cartilage of the bones in childhood is separated from the main shaft by two narrow layers: first, an outer one, nearest the epiphyseal cartilage, of a bluish color, and one or two millimetres thick; this is the proliferative layer, or hyperplastic zone, where the cartilage-cells become divided and arrange themselves in rows. Secondly, an inner, dull yellow layer, only about half a millimetre thick, known as the ossific layer, or zone of calcification, in which the real process of ossification takes place. That is, blood-vessels grow into it, osteoblasts develop, lime is deposited, and medullary spaces are hollowed out. In healthy bone these two layers are parallel to each other, and are limited by perfectly straight lines. In rachitic bone, on the other hand, they are both much enlarged, and their naturally sharp boundaries are replaced by an irregular serrated edge, so that the two zones encroach mutually upon each other. These changes affect both layers, but are most marked in the proliferative layer. Upon microscopical examination, the details of which can not be given here, we can see most plainly the complete confusion, if we may be permitted to use the expression, into which the growth of the bone has fallen. The proliferation of the cartilage-cells has increased beyond all bounds, and the scanty matrix of the cartilage displays a fibrous character. In the bony layer are seen irregularly scattered foci, which are already undergoing incomplete calcification, or marrow formation, breaking down the cartilage. The latter is due to an invasion of the vessels, which are always affected in an active new growth, pierce the cartilage like lacunar spaces, and are surrounded by a so-called osteoid tissue.

The periosteum presents analogous changes. The innermost osteoblastic layer of the periosteum is thickened; but the newly formed tissue does not become completely calcified, but remains in large part soft and spongy. Finally an increased absorption of bone takes place inside the bones. The bony partitions disappear, and the cortical layer of bone often becomes much smaller.

These various processes furnish a direct explanation of the macroscopic changes presented by rachitic bones. The proliferative process causes marked swelling of the epiphyses of the long bones, and thickening of the flat bones of the skull. The abnormal softness of the bones is due to the increased absorption of bone, their insufficient calcification, and it in turn causes various deformities, which are, for the most part, very characteristic (*vide infra*). If recovery takes place, the whole bone becomes firm at last, but often remains permanently deformed.

The deficient development of rachitic bones can also be recognized upon chemical examination. While normal bones in a dry state contain about sixty-three to sixty-five per cent. of lime, rachitic bones have only about twenty to thirty per cent.

Clinical History.—Rachitis often begins so insidiously that it can hardly be detected. Attention is not called to the disease until the deformity of the bones becomes very obvious, or it is noticed that the child does not learn to walk as early as other children, or, having already learned, is no longer able to do so. At last the anxiety of the parents is excited; and, on seeking medical advice, they find their fears only too well grounded.

In other cases, certain prodromata precede the development of the characteristic changes in the bones, which are especially mentioned by Oppenheimer. There is often a peculiar form of diarrhœa, which is said to occur only in the first half of the day, being entirely absent at other times. The discharges are scanty and almost colorless. Not infrequently the diarrhœa is attended by fever, and it is said that the spleen is almost invariably swollen. The children are pale, but not emaciated. The first characteristic changes in the cartilages of the ribs and elsewhere are said to appear within two or three weeks of these first symptoms. In other cases, Oppenheimer observed that the development of rachitis was preceded by attacks of screaming at night, likewise associated with intermittent elevations of temperature and splenic tumor; or, again, there were simple febrile attacks at night, which passed away in the morning with profuse perspiration.

These facts indicate that the entire organism is considerably affected by rachitis. It would seem reasonable to suppose that the disorder is caused by some specific infection. That this infection is malarial, as Oppenheimer thought, is more than improbable.

The diagnosis of rachitis can not be definitely established until the characteristic changes in the bones have been developed. These anomalies vary, of course, in their severity and extent in different cases. We append a list of the most important:

The head is often noticeable for its great size and somewhat square shape; the fontanelles remain open until the second or third year of life; their edges seem soft and yielding; the thinness and softness of the occiput is sometimes very striking, so that it can be pressed in like parchment. This phenomenon (the *craniotabes* of Elsässer) appears to be due to the pressure exerted upon the occiput when the child is lying on its back. There is often a peculiar change in the shape of the jaws, particularly of the lower jaw. This is not rounded, but angular, being sharply bent in the neighborhood of the canine teeth; so that the incisors stand in a perfectly straight line, besides being somewhat inclined inward. Fleischmann was the first to describe this condition, and referred it to the action of the mylohyoid and masseter muscles upon the soft bone. Dentition in rachitic children is usually tardy and tedious.

The thorax presents, even in the mildest cases, very characteristic and noticeable changes. There is a swelling at the junction of the cartilages with the ribs, which can be felt and seen through the skin, and produces what is called the "rosary of rickets." In severe cases the lateral portions of the thorax are often drawn inward, particularly at the parts which correspond with the insertion of the diaphragm. This change is due mainly to the action of the diaphragm during inspiration upon the abnormally soft and therefore yielding ribs. The changes are greatest when the respiratory efforts, and particularly abdominal respiration, are exaggerated because of bronchitis, pneumonia, or some other disease of the air-passages. In such cases the entrance of air into the lungs is impeded, so that it is possible that the external atmospheric pressure also contributes to produce the deformity of the thorax. Deep hollows may finally be developed on each side of the chest, while the sternum becomes unusually prominent, giving the whole chest that shape which has been termed pigeon-breast, or *pectus carinatum.* When once this deformity has been developed, of course it in turn contributes to render respiration difficult.

The clavicles are sometimes distorted, and may even be partially fractured (*vide infra*). The spinal column is usually unaffected if the child remains quiet in bed; but if it sits up, or is carried about, or tries to walk, the traction and pressure thus exerted often produce curvature of the spinal column (rachitic scoliosis and kyphosis). These deformities may become extreme. Changes in the bones of the pelvis are of no special clinical importance at this period of the patient's life; but in later life the consequent shortening of the antero-posterior diameter of the pelvis may, as is well known, prove a great obstacle to childbirth.

The extremities not only present swelling of the epiphyses, but are liable to curvature. This latter change is most marked in the lower limbs, inasmuch as they have to support the weight of the body. The swelling is especially well developed at the lower ends of the bones of the forearm and of the tibia and fibula. The curvature is almost invariably greatest, and therefore most easily recognized, in the tibia, which becomes convex outward, giving the rachitic child its "bow-legs." Similar curvature of the femur is less often seen, although it may be obvious enough in severe cases. The same is true of the humerus. The deformity of the lower limbs causes that waddling gait which can be so often seen on the streets of any large city. Sometimes the limbs present a sharp bend, the result of partial fracture. These "green-stick fractures of rachitis" are invariably referable to some slight traumatism, and are most often seen in the lower third of the tibia, although sometimes visible in the clavicles, ribs, and bones of the lower arm. The infraction usually takes place upon one—generally the concave—side, so that it is often compared to the partial fracture of a quill or an osier rod.

Symptoms in Other Parts of the Body.—Apart from the changes in the bones, a rachitic child may seem to be perfectly well. Even the general nutrition may be unimpaired. As a rule, however, rickets is associated with anæmia and impaired nutrition. The child seems pale, thin, and feeble, and may present swollen lymph-glands and other "scrofulous" symptoms. Sometimes there is a tendency to profuse perspiration, especially from the scalp. Very frequently there is chronic intestinal catarrh, and sometimes there is chronic bronchitis or lobular pneumonia. The liver and spleen are often, but not invariably, enlarged. It should also be stated that spasm of the glottis and convulsions are frequently observed in rachitic children. Possibly they are due to the effect of the disease upon the skull.

The fæces and urine have been repeatedly subjected to careful chemical examination, in the hope of gaining some information as to the pathogenesis of the disease. The results have thus far been rather contradictory. Much emphasis has been laid upon the fact that the fæces contain a comparatively large amount of lime. This has been ascribed to a deficient absorption of the lime-salts from the intestinal canal. The amount of lime in the urine, on the other hand, seems to be diminished rather than increased.

The disease almost invariably runs a chronic course. Usually months, or even years, pass before the process ends. Its termination is to be recognized by closure of the fontanelles, increase in the length of the bones, and, above all, by the fact that the patient becomes stronger and makes attempts to walk. Unfortunately, many results of the disease persist through life. The legs are crooked, the thorax deformed, the spinal column curved, and the pelvis narrowed. Even in the most favorable cases persons who have once had rachitis usually remain somewhat smaller than those who are perfectly healthy.

Some authorities describe an "acute rachitis," in which painful swelling of the epiphyses is said to be developed in the course of a few weeks. At the same time the child becomes emaciated, and may also suffer from diarrhœa or ulcerative stomatitis. Recovery takes place in a few months. How far cases of this sort are related to genuine rickets has not yet been determined.

Rachitis does not involve direct danger to life, although many rachitic children fall victims to the attendant intestinal catarrh, or to such complications as catarrhal pneumonia or tuberculosis. The prognosis is, therefore, not unfavorable where the outward circumstances of the child permit of good care and nourishment. The remote influences of the thoracic, spinal, and pelvic deformities can be readily inferred.

The diagnosis of rachitis is but seldom difficult if the characteristic changes in the bones exist. In case cranial changes exist, we should guard against confounding rickets with hydrocephalus, but we can usually avoid error. The rachitic child holds its head erect, and is free from mental or other functional nervous disturbances.

Treatment.—The most experienced specialists agree that the first aim in treating most cases of rachitis is to improve the general nutrition. It is often possible to bring about recovery simply by means of proper diet (milk, the yolk of eggs, and perhaps meat), good air (in the country), and baths (brine, malt, and medicated baths). Digestive disturbances should be corrected by such remedies as hydrochloric acid or tincture of rhubarb; and iron should be administered if the patient be anæmic.

It is very important that the child should be placed upon a good mattress, and should neither attempt to walk too early, nor be needlessly taken up and carried about. The best way to avoid the development of deformities in the bones is to avoid all such unfavorable mechanical influences.

Attempts have also been made to check the disease by specific remedies. Upon theoretical grounds, lime has been very frequently prescribed in the form of phosphate of calcium, of which fifteen to forty-five grains may be given in powder several times a day; or in the form of lime-water, of which one or two teaspoonfuls are added to the milk which the child drinks. The benefit of these remedies is seldom very obvious. Kassowitz has given a fresh impetus to the employment of phosphorus. To support his belief he brings forward numerous clinical observations as well as facts obtained from experiment. We may either dissolve the phosphorus in cod-liver oil (0·01–100), giving one or two small teaspoonfuls of this solution every day, or we may write for the following mixture, which is more elegant, but is also more apt to spoil:

℞ Phosphori.................................. 0·01;
Olei amygdalæ expressi.......................... 10·0.
Misce, deinde adde:
Pulv. acaciæ,
Syrupi simplicis.................................āā 5·0;
Aquæ destillatæ.................................. 80·0.
M. Sig.: Two to four small teaspoonfuls a day.

We can state from our own experience that the remedy is usually very well borne, and, in fact, often shows its beneficial action after a few weeks, the fontanelles growing smaller and the bones becoming firm.

It may be eventually necessary to resort to orthopædic or surgical treatment in order to correct the deformities of the bones.

[The comparative rarity of rickets, especially in its extreme degrees, in this country strikes all observers who have studied in Germany. With a fairly extensive experience among the poorer classes of the city, the writer can recall scarcely half a dozen cases of craniotabes. The colored race furnishes a large contingent of cases of rickets, although, as is shown by Haven, in attention to diet and fresh air its members are superior to the Irish laboring classes, as a rule. The more pure the negro blood, the greater does the liability to rickets seem to be in this latitude—an indication, perhaps, that a northern climate is unsuitable to the African race.]

CHAPTER V.

OSTEOMALACIA.

Ætiology and Pathology.—As a rule, osteomalacia does not, like rachitis, consist in a disturbance of development. The growing bones are not prevented from ossifying, but, having already undergone normal development and acquired normal firmness, they afterward become soft. It is mainly a disease of adults, say between thirty and forty years* of age. The female sex is noticeably predisposed to the disease, although occasionally it has been observed in men.

The true cause of osteomalacia has not yet been ascertained. It is a remarkable fact that the disease is much more frequent in certain regions than in others. It is very common along the Rhine, and in Westphalia, in eastern Flanders, and northern Italy. This suggests that there is some specific cause for the disease, endemic in certain localities. Among exciting causes, child-bearing is certainly the most important, for both the first signs of osteomalacia, and also fresh exacerbations of the disease, usually date from a pregnancy. Another factor said to promote the development of the disease is found in unfavorable hygienic surroundings, such as damp dwellings and the like.

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* Rehn maintains that genuine osteomalacia may occur in children; but his statement has not yet been fully corroborated.

The anatomical process of osteomalacia consists in a disappearance of the earthy salts of the bone, which begins interiorly and spreads outward, and causes a corresponding softening of the bony structure. The marrow is at first extremely hyperæmic; and extravasations of blood are not infrequently found here and there. The bony substance surrounding the myeloid spaces and the Haversian canals becomes transformed into a soft fibrous tissue, while the irregularly arranged bone-corpuscles are either destroyed or lose their characteristic shape. The softening process gradually extends over the spongy substances outward to the cortex. The central cavity grows larger and larger, so that finally the cortical substance is as thin as paper, and the whole bone like an "inflated and dried coil of intestine." At this stage the original hyperæmia of the marrow has vanished. The marrow acquires a yellow color, and may finally be entirely transformed into a yellow, viscid fluid. The affected bones are now flexible and soft, they can be easily cut, and they are of less specific gravity than normal. The periosteum is also at first thickened and hyperæmic, as if inflamed. When it is removed, the surface of the bone beneath it is found to be rough and uneven. The attendant alterations in the shape of the bones will be mentioned below.

Upon chemical examination of the bones in osteomalacia, we naturally find a marked diminution in the proportion of lime-salts. It is also stated that lactic acid has been discovered in the bones. This is an interesting fact, as it may be that the acid plays an important chemical part in the process of decalcification.

Clinical History.—Osteomalacia begins very gradually in most cases. Usually the first thing noticed is an ill-defined, deep-seated pain, most often felt in the sacral region, the nape of the neck, and the back and thighs. The affected parts are also sensitive upon pressure.

The pain is persistent. While it still keeps on, motion becomes gradually impaired. The patient experiences more and more difficulty in walking, partly because of the pain and partly because of muscular weakness. The gait is either uncertain and tottering, or characterized by short painful steps, the lower limb and the pelvis being jerked forward as if in one piece. Sooner or later it becomes impossible to walk, and the patient is permanently bedridden. Even now the pains usually persist in great severity. They are not actually spontaneous, but the mere pressure of the mattress and the bedclothes is sufficient to excite them.

In the meanwhile many of the bones will probably have become distorted. Usually the deformity of the spinal column is the first change which attracts attention. This is generally kyphotic; less often the curvature is in the opposite direction. At the same time the head approaches the sternum more and more, and the patient is thus made to appear much shorter than she really is. In most cases, also, the thorax is much distorted. It is compressed laterally, while the sternum becomes very prominent, and is sharply bent. The change in the shape of the pelvis in osteomalacia is less obvious externally, but it can be detected on internal examination. It is, of course, of great importance from an obstetrical point of view. The pelvis, like the chest, is compressed laterally, while the symphysis is made to project forward like a beak. The sacrum and its promontory are also pushed forward, and the superior strait thus acquires somewhat of a heart shape.

The extremities are less often distorted, particularly if the patient becomes bedridden at an early period. Manifold changes are, however, possible. Sometimes there is also fracture. In a few reported cases the softness of the bones of the extremities was so extreme that one could bend the limbs at will, like wax, and give them the most extraordinary positions. In cases so far advanced as these, the pain in the bones seems finally to cease.

The bones of the head and face seldom undergo noticeable change, but we often find the teeth carious or lost. In the muscles, several observers have noticed trembling and fibrillary contractions. It is also said that sometimes even a slight irritation of the skin suffices to excite painful contractions of the underlying muscles. These phenomena have not yet been thoroughly investigated.

The general condition of the patient is often unimpaired for a long while, except for the pain and the impairment of motion. The internal organs perform their functions in a normal manner, and the appetite is good. Fever is observed only when the disease is undergoing some temporary exacerbation. With regard to changes in the urine, there have been a good many statements made, but their significance is extremely doubtful. It is said that the amount of phosphoric acid excreted is diminished. With regard to the amount of lime, no definite statement can be made. Lactic acid has been repeatedly detected in the urine, as has also albumen. Concretions of lime have been found in the bladder and the kidneys.

The disease runs a chronic course, occupying seldom less than two or three years, and sometimes even five or ten. Apparent arrest of the disease is not infrequently observed, but this is followed by fresh exacerbations. The most frequent termination is in death. This results either from general debility, or, more often still, from the dyspnœa caused by the compression of the lung, or by some such disease as lobular pneumonia. Recovery is exceptional, although not impossible.

Diagnosis.—In well-developed cases it is not difficult to recognize the disease, but at first a correct diagnosis is often impossible, unless the endemic frequency of osteomalacia suggests it. In the onset of the disease we may often be led to the false belief that there is incipient disease of the cord on the vertebræ. An examination of the pelvis is then of distinct diagnostic importance, as its peculiar deformity can be recognized early. The very peculiar hobbling gait of osteomalacia is also unlike anything that is seen in spinal disease. As the disease is almost entirely confined to adults, we are seldom in danger of confounding it with rachitis. Besides, osteomalacia does not produce swelling of the epiphyses, nor changes in the bones of the skull. It is said that osteomalacia is occasionally confounded with diffuse carcinosis of the bones, as this may produce similar symptoms and deformities.

Treatment.—As has been already implied, therapeutic efforts have thus far proved in severe cases almost unavailing in this disease. In the onset of the disease we can obtain a distinct improvement by the use of hygienic remedies, good air, proper food, etc. Internally we give cod-liver oil and iron. Many patients praise warm baths, with or without salt.—The exhibition of lime seems to be of no special use, but, from our own experience, we must urgently advise trying small doses of phosphorus in the form given above (page 925).

The changes in the bony pelvis produced by osteomalacia may eventually demand obstetrical interference, but we need not discuss such procedures here. We should invariably warn women who suffer from the disease of the dangers of becoming pregnant.

DISEASES AFFECTING THE BLOOD AND TISSUE-METAMORPHOSIS.

(*CONSTITUTIONAL DISEASES.*)

CHAPTER I.

ANÆMIA AND CHLOROSIS.

Definition and Ætiology.—The word "anæmia" might properly be taken to signify diminution of the total volume of the blood, such as, for example, is directly brought about by a severe hæmorrhage. Usually, however, the word is employed to signify not so much diminution in quantity as deterioration in quality. The total volume of the blood is not liable to nearly so great variation as is the number of its most important constituents—the red corpuscles—inasmuch as the total volume is dependent merely upon the amount of watery constituents, and even after large hæmorrhages the water is quite rapidly replaced by absorption. This is undoubtedly the case in most instances of sudden loss of blood. Even in chronic anæmia there is usually no reason to assume that the total amount of blood is diminished, although it may be where there is general emaciation, or diminished supply of liquids (persistent vomiting, dysphagia), or large watery discharges (as in cholera). The essential element in anæmia is, therefore, a diminution in the number of the red blood-corpuscles, or so-called oligocythæmia. Changes in the character of the red blood-corpuscles, however important, are not taken into consideration here; nor is there usually any stress laid upon any incidental variations in the proportion of albumen, especially as oligocythæmia is not invariably accompanied by a diminution in the amount of serum albumen ("hypalbuminosis").

The circumstances under which anæmia is observed are manifold. They admit, however, of our distinguishing the two great classes of anæmia—primary and secondary. Primary anæmia is developed as an apparently primary and idiopathic disease in people previously healthy, while secondary anæmia is merely a symptom of some already existing disease. However simple this theoretical distinction, yet in actual practice it is often quite difficult to determine whether the particular case before us should be regarded as primary or secondary. A secondary anæmia may occur where the true primary cause can not be at all readily determined. There are, nevertheless, quite a large number of cases which would seem to deserve the name of primary or essential, in which we feel compelled to assume that some pathogenetic influence acts directly upon the blood and the hæmatopoietic processes.

In the first place, we would class as primary, cases which may best be described as "simple constitutional anæmia." These often stand close to the borderland between health and disease. There are not a few individuals who present a striking pallor for a large part, if not all, of their lives. These persons may feel so well and vigorous that we scarcely have a right to regard the existent anæmia as an actual disease. Sometimes, however, such individuals do betray some diminution of energy, are easily fatigued, and are subject to headache. We may then certainly regard the condition as pathological. In many instances the cause of this simple anæmia is found in the general hygienic surroundings of the patient, for such cases are most often met with among the poorer classes. Deficient nutrition, bad air, unhealthy occupation in factories or the like, not only affect the general health, but more especially interfere with the processes of normal blood-making. Other cases of constitutional anæmia, apparently primary, occur in individuals who are entirely beyond the reach of such influences as have just been mentioned, in whom the anæmia has developed and persists despite the best of food and air. Here we are forced to the conclusion that the organs engaged in the manufacture of the blood are in some way prevented from performing their proper functions. The trouble often seems to be congenital, for such individuals may present the symptoms of anæmia in their earliest infancy. There are persons who have always been pale and feeble. Or, again, anæmia does not develop until later on, in which case it not infrequently associates itself with certain phases of physiological development, as when growth is particularly rapid, or when adolescence occurs. Virchow has directed attention to another factor, which he regards as potent in many of these cases of congenital anæmia. He has found that the arteries may be congenitally small, or that the whole arterial system may be imperfectly developed. The condition may be associated with a congenitally weak and small heart. The importance of this factor has not yet been fully determined. Possibly the condition of the circulatory system just mentioned may be the result rather than the cause of the anæmia.

A second division of primary anæmia includes cases which present a far more definite and distinct group of symptoms. They quite often appear in persons previously healthy, last for a certain length of time, and then end in complete recovery. The typical form of this variety is chlorosis (χλωρὸς = greenish yellow), or "green sickness." This well-known disease is especially frequent in young girls fourteen to twenty years of age—that is, at puberty. It often comes on quite rapidly without any ascertainable cause. There are not infrequently predisposing influences in the outward circumstances of the patient. Thus it is promoted by an unhealthy sedentary mode of life, as in seamstresses; bad air, as in factory operatives; mental and physical over-exertion, as in teachers, governesses, and students; and, finally, by mental influences. It is, nevertheless, true that chlorosis also appears in girls who have lived under the most favorable hygienic conditions possible. Sometimes the disease seems to be merely a temporary exacerbation of a simple constitutional anæmia which has existed a long while; but it may also appear in young women who were previously healthy, and even robust.

The true cause of chlorosis is unknown. In all probability it is a disease of the blood itself, or a process interfering with its normal manufacture. Its pathological physiology is, however, as yet entirely beyond our grasp. The old view, that chlorosis was referable mainly to sexual derangement, such as disturbance of menstruation, or defective development of the genital organs, must be regarded as a confusion of cause and effect, although it is true that such disturbances are often seen in the disease. Furthermore, cases of marked temporary anæmia, precisely like ordinary chlorosis in their symptoms and behavior, occur in men and in elderly individuals.

A third variety of primary essential anæmia is the so-called progressive pernicious anæmia. This is likewise an idiopathic disease, distinguished from chlorosis mainly by its continuous progress and fatal termination. We must confess that in our opinion it is not possible, at least from a clinical point of view, to draw a sharp dividing line between "ordinary chlorosis" and "pernicious anæmia." Some future investigator may discover ætiological as well as anatomical differences between the two diseases, which will separate them widely. In the meanwhile we have only the clinical phenomena to guide us, and must acknowledge our inability to make any sharp distinction. There are "severe cases of chlorosis" which resemble "pernicious anæmia" in every respect, except that they finally get well ; so that the only point which would enable us to distinguish them from the fatal disease is the mode of termination. To take this for a criterion is evidently unscientific. Cases of "severe essential anæmia" also have many points in common with certain other diseases, such as pseudo-leukæmia and splenic anæmia. These will be discussed later.

The forms of secondary anæmia offer a contrast to the forms of primary or essential anæmia just described in the much greater number of their causes. Under this head come cases of anæmia which do not occur idiopathically, but as a result of other abnormal processes. The simplest variety is anæmia from hæmorrhage. This is produced by profuse loss of blood, whether from the stomach, lungs, uterus, intestines, kidneys, or some wound. Repeated small hæmorrhages finally produce the same result as a single large one. Thus the most profound anæmia may be observed where there is a very frequent epistaxis (hæmorrhagic diathesis), or where cancer of the womb gives rise to a constant oozing of blood.

Other cases of secondary anæmia may be divided into two great groups. In one class the anæmia is a symptom of impaired nutrition. This is seen in almost every severe disease, acute or chronic, and is usually associated with more or less emaciation and loss of strength. The bad appetite, the lack of fresh air and exercise, and perhaps an impairment of digestion, or fever, or some abnormal drain upon the system, as in suppuration—these injure the entire body. It is not surprising that the blood shares in the universal misfortune. This is why most chronic invalids seem pale, particularly if they suffer from disease of the stomach, kidneys, chest, or nervous system. In the second class, the anæmia is secondary to some other disease, but assumes especial prominence as a symptom, independently of any general impairment of nutrition. Of course, it is often associated with emaciation, but nevertheless its extraordinary intensity offers a striking contrast to the condition of the rest of the body. This "specific secondary anæmia" must, like essential anæmia, be due to a special lesion of the blood itself, and is, therefore, to be regarded as in a certain sense a special complication or localization of the primary disease. General malnutrition never directly produces anæmia of this grade. This fact is illustrated in stenosis of the œsophagus. Here the ingestion of food is very greatly, if not completely, impaired, and there is the greatest emaciation, with a subnormal temperature and slow pulse. Of course, such a patient appears pale and wretched, but he does not present that peculiar waxy pallor which is the infallible sign of genuine anæmia.

The exact mode of origin of specific secondary anæmia is often obscure. We have already reported, in a preceding chapter, a very instructive example. In cancer of the stomach we find usually emaciation and pallor. This is natural enough; but sometimes the carcinoma is complicated by an extraordinarily profound anæmia, comparable only to the pernicious variety. In one such case we found, at the autopsy, an extensive secondary carcinosis of the bone-marrow. Here, therefore, the anæmia certainly was not the result of the general impairment of nutrition occasioned by the gastric carcinoma, but of the disease of the marrow, which tissue undoubtedly plays an important part in the manufacture of the blood.

Some cases of specific secondary anæmia deserve special mention, although it is seldom possible to demonstrate their precise cause. In the first place come such cases as develop after certain acute diseases, usually of an infectious character. For example, there may be great anæmia after typhoid fever, or less often after acute articular rheumatism. A peculiar form of anæmia is often observed during the secondary stage of syphilis, although nutrition seems to be otherwise well maintained. This is known as "syphilitic chlorosis." Tuberculosis, chronic malarial poisoning, and other cases of chronic poisoning (lead), as well as amyloid disease, renal disease, etc., may also be attended by anæmia of such intensity as to justify the conclusion that there is some special disturbance of secondary origin affecting the manufacture of the blood or the blood itself. [Chronic arsenical poisoning from wall papers, dress fabrics, furniture covering, and the like, is a not infrequent cause of anæmia and debility, which resists treatment as long as the exposure to the toxic influence lasts. The classical symptoms of chronic arsenical poisoning are not necessarily present. The number of cases in which prompt recovery has taken place after the removal of the poison which was proved to exist in the surroundings as well as in the urine of the patient is too large and too striking to explain by simple coincidence. Recovery may, however, be slow in those whose toleration of arsenic is slight, or in whom the cause of the symptoms has remained long undetected.]

We shall now proceed to describe the symptoms common to all forms of anæmia. Upon this will follow a sketch of chlorosis. A special chapter is assigned to the grave form of essential anæmia known as progressive pernicious anæmia; and in the same connection will be set forth the little that is known with regard to the relations of anæmia to pathological changes in the hæmatopoietic organs.

Symptomatology of Anæmia.—The first symptom which attracts the attention of the physician in any case of anæmia is the altered appearance, the pallor of the skin and visible mucous membranes. This is especially striking in the face, but it is sufficiently evident everywhere. Special value is usually assigned to pallor of the mucous membranes, for example, of the lips and conjunctivæ, inasmuch as their color is not liable to be interfered with by pigmentation or opacity of the epidermis. The degree of pallor, of course, varies greatly. The whole body may present a waxy appearance. Such pallor, of course, indicates a very decided diminution in the number of the red blood-corpuscles, which elements impart to the blood its normal color. More information about this and other changes in the blood will be found below, under chlorosis and pernicious anæmia.

Besides the alteration in complexion, there is always a group of symptoms ultimately referable to an impairment of the normal processes of innervation resulting from a lack of arterial blood. First among these phenomena comes general weakness of the motor system. The voluntary muscles are easily fatigued, and the patient suffers from constant languor. When the anæmia is very great, as after severe hæmorrhage, this weakness may be so pronounced that the patient can neither walk nor stand.

This diminished nervous energy is also shown by the mental condition. There is no intellectual vigor. The patient is incapable of any great mental exertion, and experiences a constant feeling of weariness and sleepiness. Whether the special senses are blunted in anæmia has not yet been determined. It is very probable that a careful investigation would reveal an impairment of the perceptive powers, corresponding to the muscular weakness. If the anæmia reaches a certain degree, the patient becomes unconscious. This explains the frequent fainting attacks (compare page 708), which are referable to a temporary aggravation of the cerebral anæmia, and are therefore apt to come on after the patient has been standing for some time, or when he rises from a horizontal position. It is an extremely interesting fact that a circumscribed portion of the nervous system may alone suffer; thus we may have an anæmic amaurosis after profuse hæmorrhage. There is no doubt that this blindness is due to anæmia of the optical nervous apparatus. The only question is whether the anæmia affects chiefly the retina or the central portion of the optic tract (cortex of the occipital lobe).

If the anæmia be at all marked, many other organs exhibit functional derangement. In particular, the secretory organs are disturbed. The mouth and tongue are frequently dry. This is in part due to the diminished secretion of mucus and saliva. Of course, where there is a sudden great loss of blood, the condition is also in part a result of the compensatory abstraction of water from the tissues. Other and still more important glands belonging to the digestive system are affected. Our knowledge in this regard is as yet very far from complete; but Manasseïn has called attention to the interesting fact that the amount of hydrochloric acid in the gastric juice is considerably less than normal, and that the dyspepsia so often seen in anæmic patients must in part be referable to this condition. Analogous disturbances in the functions of other digestive organs are presumably present, but they have not yet been actually proven to exist. We would add only that the constipation to which anæmic persons are very liable is usually due to diminished energy of peristaltic action.

The symptoms thus far described are all referable to diminished functional activity. On the other hand, anæmia gives rise to certain symptoms of irritation in the nervous system. It would, of course, be illogical to say that these symptoms are the direct result of a lack of oxygen-carrying blood. They are, in all probability, expressive of the irritation excited in certain portions of the nervous system by the products of abnormal tissue-metamorphosis. It may be that these products are themselves the result of a deficiency in the supply of oxygen.

The symptoms of cerebral irritation observed in anæmia include vertigo, spots before the eyes, and tinnitus aurium. This last is an almost constant symptom, if the anæmia be at all severe, and it may cause the patient great discomfort. It is usually aggravated if the patient lies upon his side.* Another irritative symptom is eructations. We also have anæmic vomiting, and no doubt this is usually of central origin. It may be very troublesome. Sometimes violent hiccoughs are observed, as well as frequent yawning of a convulsive character. Anæmic headache may be very severe. It is usually referred either to the entire head or the forehead; there is a painful feeling of pressure, which may attain great severity.

Other important evidences of irritation are to be found in the behavior of the pulse and the respiration. The changes here are apparently in part of a compensatory character. The pulse is accelerated in most cases of any severity, reaching 80 to 100 beats per minute, or even more. It is also very excitable, so that comparatively slight external influences suffice to increase its rapidity for the time being. This increase in frequency would not, of course, necessitate an increase of intravascular tension, or of the rapidity of the circulation; but it may exert a favorable influence in this direction, and so be teleologic. Respiration is also usually accelerated in anæmia. In cases of great severity the breathing may be so deep and noisy as to justify the term "anæmic dyspnœa." This is the direct expression of the body's hunger for oxygen. It is obvious that this increase in the number of respirations favors, to a certain extent, the absorption of that gas.

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* We may also state that the tinnitus of the anæmic is sometimes nothing but the jugular venous hum (*bruit de diable*) heard by the patient.

There are still other symptoms referable to the circulatory system. It has been already stated that the total volume of the blood is not diminished in anæmia unless, of course, there has just been an actual hæmorrhage. This explains why the pulse in anæmia is often comparatively full and strong. Quite frequently there is a peculiar quickness of the pulse. This seems to be due to a vigorous cardiac systole, in conjunction with a low intra-arterial tension. This supposition explains the fact, which we have often observed, that there may be in profound anæmia a loud sound in the femoral artery similar to that heard in aortic regurgitation.

It has long been known that anæmia may produce functional cardiac murmurs, often called "anæmic murmurs." Their mode of origin is not yet fully explained. They are usually heard loudest over the base of the heart, in the neighborhood of the pulmonary valves, although sometimes at the apex of the heart. As a rule, they are purely systolic in time, but we certainly heard in one case of pernicious anæmia a loud diastolic murmur of anæmic origin. The murmurs are of a blowing character. Sometimes they are so rough as to simulate pericardial friction-murmurs; it has therefore been suggested that many anæmic murmurs are actually due to the rubbing upon each other of the abnormally dry folds of the pericardial sac. Another explanation of their occurrence is, that the movements of the cardiac valves are interfered with, as a result perhaps of fatty degeneration of the myocardium (*vide infra*). It is also possible that they are due in some cases to relative insufficiency, resulting, for instance, from dilatation of the heart or imperfect action of the papillary muscles.

Murmurs in the large veins of the neck are very often heard in anæmia, either with or without cardiac murmurs. They are often called *bruit de diable*. No less an authority than A. Weil has maintained that murmurs in the jugular veins are just as frequent in healthy persons as in the anæmic; but our experience obliges us to differ from this view. We believe that the loud venous murmurs are more frequent in the anæmic than in other persons. We can not claim, however, that they are of any great diagnostic value.

The processes of tissue metamorphosis in profound anæmia are of great interest, but, unfortunately, they have not yet been thoroughly investigated. It is extremely probable that the absorption of oxygen in severe anæmia is less than normal, and that the body must, therefore, suffer from a diminished supply of oxygen. It has been demonstrated by the experiments of A. Fränkel that there is an increased destruction of albuminoids within the body and a correspondingly increased excretion of nitrogen through the kidneys. This experimental deduction we were the first to confirm in a case of very severe essential anæmia; and later various observers have in other cases arrived at similar conclusions. Of course, the excretion of nitrogen is influenced by many different factors, so that the truth of the above statement is not easily established. There can be no doubt, however, that in many instances of profound anæmia a larger amount of nitrogen is excreted than is ingested. This fact acquires a special significance when taken in connection with certain anatomical lesions produced by anæmia: there is almost invariably a well-marked fatty degeneration of many organs, particularly of the heart and kidneys. This fatty degeneration is the direct result of the abnormal destruction of albuminoid structures. The fat represents the non-nitrogenous remnants of the decomposed albuminoids. The reason why the fat itself does not undergo oxidation is the same that leads to the destruction of the albuminoids—namely, a lack of oxygen. This explains why the panniculus adiposus is for a long while preserved in many cases of anæmia.

It is evident that the fatty degeneration once produced must, in its turn, lead to unfavorable results. It has already been suggested that the fatty degeneration of the heart may be the cause of certain irregularities in its functional activity; but it should be said that this is not invariably the case, for often the heart exhibits a surprising energy despite marked fatty degeneration of its muscular tissue. The corresponding changes in the walls of the blood-vessels are, however, of great importance, as they frequently occasion disturbance, above all by hæmorrhage. In many instances (for example, in leukæmia, *vide infra*) an actual hæmorrhagic diathesis is developed; but we shall find later (see the chapter on pernicious anæmia) that the condition of hæmoglobinæmia and the chronic fibrine-ferment intoxication caused by it play a part in producing capillary hæmorrhages in severe anæmia. There is also strong evidence in many cases that there is an abnormal permeability of the vascular walls. From this results the mild grade of œdema frequently seen in anæmic patients, although in some few cases this œdema may be ascribed to passive congestion, resulting from the cardiac debility (*vide supra*). The increased permeability of the renal blood-vessels is sometimes shown by polyuria.

The urine is usually rather light-colored, if the anæmia be at all marked. It is evident that there is a diminished production of urinary pigment, the material for which is the coloring matter of the blood. Another reason for the light color of the secretion is the polyuria above mentioned. There may be fifty to seventy ounces (1,500–2,000 c. c.), or more, secreted in twenty-four hours. The specific gravity may nevertheless be comparatively high, and higher than one would expect from the appearance of the urine, not infrequently ranging between 1015 and 1021. This indicates, of course, that the amount of solid constituents is comparatively large; and in fact we find a corresponding amount of urea, say four or five hundred grains (grm. 25–32) in twenty-four hours. This is a large figure when we consider the amount of ingesta. As to the other constituents of the urine, we possess as yet little definite knowledge. The amount of phosphoric acid is sometimes surprisingly small, compared with the amount of nitrogen. Albuminuria is exceptional in cases of simple anæmia.

The bodily temperature is very often affected in anæmia. An "anæmic fever" is very frequently associated with pernicious anæmia, and even with cases of profound secondary anæmia, as after a large hæmorrhage from the stomach. There are irregular elevations of temperature, usually occurring at evening, and attaining 101°–102° (38·5°–39° C.), or even still higher figures. This fever is not due to inflammatory organic changes, but, in all probability, is rather the result of the presence of fibrine ferment in the blood.

Clinical History of Chlorosis.—Chlorosis, or "green-sickness," as already explained, is a term applied to the mild forms of essential anæmia, such as are most often seen in females at the time of puberty. The disease sometimes develops in previously healthy girls with considerable rapidity, and may completely vanish again at the end of a few weeks or months. In other cases it runs a more tedious course, without sharp limits, so that the condition resembles that of constitutional anæmia, or it may be described as an habitual chlorosis. In many instances the disease may be said to undergo repeated relapses.

The various symptoms of chlorosis are almost all direct results of the anæmia, so that they have already been described. Their intensity and variety are, however, very different in different cases. There are mild cases where the patient can hardly be called ill. She feels perfectly well, but is "a little pale." From these cases there is a gradual and unbroken transition to the other extreme of severity.

A constant and essential symptom is the greater or less pallor of the face as well as of the rest of the surface of the body, and of the mucous membranes so far as visible. There is also almost invariably general languor; the patient is easily fatigued, and has neither the desire nor the ability to make any great bodily or mental exertion. There is also a tendency to headache and vertigo. Other nervous or "hysterical" symptoms, if they occur, are not directly ascribable to the disease itself, but are merely complications. Chlorotic patients often complain of dyspepsia. The appetite is usually diminished, and there is often a sense of pressure in the epigastrium after meals. There may also be severe cardialgia. This is usually of a purely nervous origin, but is sometimes produced by a gastric ulcer existing as a complication of the chlorosis. There is not infrequently constipation, as might be expected from the small amount of food taken, and from the diminished activity of intestinal peristalsis. Over the veins in the neck we often hear a loud murmur, above referred to, and called *bruit de diable.* Sometimes the heart is found to be slightly dilated. This is probably a result of the diminished power of resistance of the cardiac tissues. Anæmic cardiac murmurs are not infrequent; the pulse is accelerated and easily excited. Otherwise physical examination does not reveal any abnormal condition of the internal organs. It is a very great exception to find any indications of disturbance in the spleen or lymph-glands, or the bone-marrow. Fever is rare in cases of simple chlorosis, but in severe cases we sometimes see, especially at night, a slight rise of temperature, up to 101° F. (38·5° C.). The urine is usually pale, but seldom differs greatly, either in amount or constituents, from the normal character. It is noteworthy that chlorotic girls are very apt to suffer from disorders of menstruation. The menses may not appear until late, or, if they occur, they are very scanty. It is only in exceptional instances that chlorotic patients have menorrhagia.

Examinations of the blood have been made, in the hope of gaining a clearer insight into the true character of chlorosis. The blood is usually pale. Upon microscopic examination, we notice that the red blood-corpuscles do not tend to form rouleaux so much as normally, and sometimes we can perceive that the globules are comparatively light-colored, and are not all of the same size. There are some of the normal dimensions, others are notably small (microcytes), while there are a few which are unusually large (macrocytes). Here and there we see corpuscles of irregular shape (poikilocytes). Often there is some increase in the number of white blood-corpuscles—that is, there is a slight degree of leucocytosis (*vide infra*). In occasional instances there are considerable numbers of "granular bodies" in the blood. These are usually regarded as products of the disintegration of white blood-corpuscles. Malassez, Hayem, Thoma, and others have endeavored to determine the number of blood-corpuscles in chlorosis and allied diseases by means of special methods of counting. In general, we may say that in most cases of chlorosis the number of red blood-corpuscles is decidedly diminished. In a cubic millimetre of blood we find perhaps only 3,000,000 to 3,500,000 red blood-corpuscles. instead of the normal number of 5,000,000. The diminution may be even greater than this. On the other hand, it should be noted that Duncan, Hayem, and Laache have met with cases where the number of corpuscles was not diminished. Probably here the amount of hæmoglobin in the blood-corpuscles was diminished. An intelligent and satisfactory explanation of all these isolated facts has not yet been given. There are certain hypotheses which have been brought forward, and these we shall discuss in the following chapter, where also a more extensive description will be found of the changes presented by the blood.

We have already intimated that there is great variety in the general course of chlorosis. Many cases which seem severe at first terminate in complete recovery by the end of four to six weeks, or a few months. Other cases are much more obstinate, resist all modes of treatment, and undergo frequent relapses. The prognosis may therefore be regarded as favorable on the whole, but it should always be given with a certain degree of reserve. It is true that ordinary chlorosis never involves direct danger to life. We shall, however, find that there is no sharp dividing-line between "simple chlorosis" and "pernicious anæmia"; and in any individual case it may not at first be possible to decide which variety of anæmia is before us.

Diagnosis.—The diagnosis of chlorosis may be regarded, therefore, either as extremely easy or extremely difficult to make, according to the point of view. It is easy in that we can readily perceive the characteristic symptoms of pallor and the like; but it is difficult in that the term chlorosis should be applied to those cases only where the anæmia is primary and essential. We therefore have no right to declare the diagnosis of chlorosis until we have made a careful and thorough physical examination, and have found that no factors are present which might produce a secondary anæmia. We should, above all, bear in mind the possibility of incipient tuberculosis, and examine the lungs, the expectoration, and the general condition of the patient, and also consider hereditary and other predisposing influences. We should also bear in mind the possibility of some organic disease of the stomach, such as ulcer, catarrh, or dilatation, or chronic renal disease, or possibly constitutional syphilis, giving rise to the syphilitic chlorosis before mentioned. In many cases it is easy to exclude all these secondary forms of anæmia, but occasionally the task is a very difficult one.

Treatment of Anæmia and Chlorosis.—The first indication in treating chlorosis, as well as every other form of anæmia, is to promote the regeneration of the blood. There are two ways in which we can endeavor to fulfill this indication: first, by general hygienic and dietetic measures, and, secondly, by employing certain internal remedies.

First among general measures comes a care for pure air and proper food. Many a pale city girl regains her ruddy cheeks after a few weeks spent either in the country, or on the mountains, or at the seashore. The choice of a place must, of course, depend mainly upon the circumstances of the patient. In many instances, boarding in any suitable country place answers as well as a long and expensive journey. If the seashore be chosen, some bathing resort on the North Sea will usually be preferable. There are other places where the patient can combine the advantages of pure forest air and ferruginous mineral water; these will be mentioned below.

The diet of anæmic patients should be easily digestible and rich in albumen; carbo-hydrates and fat should be given in but limited amounts to patients in whom the panniculus adiposus is already well developed. Lean patients, on the other hand, require such ingredients in their diet, and should be recommended to take simple puddings, extract of malt, butter, and cod-liver oil. If milk be well borne it is an excellent article of diet for the anæmic; but what is called the "milk cure"—that is, an almost exclusively milk diet—is not advisable, as we have already had occasion to observe (see the chapter on tuberculosis). Some authors ascribe an exaggerated value to alcoholic beverages. They may be allowed in moderate amount if the patient asks for them and finds the appetite improve under their use. The best to choose for an emaciated patient are porter and other varieties of beer rich in extractive principles, but wine is often ill borne by chlorotic girls.

Many physicians also insist upon "abundant exercise in the open air." This can, however, be carried too far. A chlorotic girl is often made to take long walks in spite of her own reluctance to do so, with the result of becoming more languid and exhausted than she was before. We even believe that a case of profound anæmia will be benefited by a certain degree of bodily rest. Thus any needless tax upon the muscles is avoided. The most brilliant results we have ever observed in the treatment of chlorosis have been within the hospital. The patients, who are usually factory operatives and shop-girls, are often kept in bed for the first week. We admit that pure country air may be very beneficial, but we would strongly advise moderation in bodily exercise. If the patient begins to feel more vigorous, she will of herself take more exercise, and may safely indulge in long walks or extended excursions on foot.

Of internal remedies, the preparations of iron have long occupied the first place in the treatment of all varieties of anæmia. The manner in which their good effects are exerted was until recently unknown, for careful investigation had established the fact that the salts of iron are absorbed by the intestines only in an extremely small amount, and that the system needs so little iron that the iron in ordinary food ought to be entirely sufficient. Bunge has recently enabled us to understand why iron possesses therapeutic value, at least in some cases. He has shown that the iron contained in ordinary food does not exist in an inorganic form, but in a very complex organic combination, to which he has given the name of hæmatogen. The inorganic salts of iron given as a medicine protect the hæmatogen from being decomposed, a danger to which it is liable from the sulphides which are generated in the intestinal canal.

Hence it follows that iron can not act in the same way in all cases of anæmia, and, in fact, we often see that iron may be taken by anæmic patients for a long time without any effect; but, on the other hand, the action of iron is often apparently so beneficial that, in spite of our unsatisfactory theoretical knowledge, we still give one of the many iron preparations, together with general dietetic prescriptions, in severe anæmia and especially in chlorosis. It may be regarded as a general rule to give not too small doses, and to give these two or three times a day, directly after meals. Ferrum redactum is perfectly pure iron, in a state of very fine subdivision, and may be given either as a powder or in pills, several times a day, in the dose of one to three grains (grm. 0·05–0·20). A very good preparation for children consists of pastiles made of chocolate containing reduced iron. Two other powders are contained in the list of the German pharmacopœia, ferrum carbonicum saccharatum, and ferrum oxydatum saccharatum, the dose of each being half a teaspoonful to a teaspoonful in water three times a day. The second of these preparations, ferrum oxydatum, has the advantage of not blackening the teeth. An easily digestible preparation is ferri lactas, which may be given either as a powder or in pills, the dose being five to twelve grains (grm. 0·3–0·75). Finally, the sulphate of iron should be mentioned. This is one constituent of the well-known and very valuable Blaud's pills (ferri sulphatis exsiccat., potassii carbonat., āā grm. 15; pulv. tragacanth, *q. s.* Misce et div. in pil. No. 100. Sig.: Three to five pills three times a day). The German pharmacopœia contains three tinctures of iron—viz., tinctura ferri acetici ætherea, tinctura ferri chlorati ætherea [resembling tinctura ferri chloridi, U. S. P.], and tinctura ferri pomata. We must also praise the liquor ferri albuminati, a teaspoonful three times a day, which often has a good effect on the appetite. Repeated trials have lately been made with successful results, in giving hæmoglobine internally, especially Pfeufer's hæmoglobine pastiles, made of ox blood, giving six of them a day. Often iron is given in combination with other remedies, of which quinine is chief. This is said not only to increase the tonic action, but to lessen the headaches to which anæmic patients are subject. Stomachic tonics, such as gentian, or laxatives, such as aloes, are also combined with iron.

Exceptionally iron is not well borne, and causes digestive disturbances or diarrhœa. We must then try another preparation or diminish the dose. It is common to forbid the ingestion of sour articles of diet while taking iron, but the injunction is due mainly to prejudice.

The use of mineral waters containing iron is quite common, although the amount of iron thus introduced into the system is so small that it is hard to understand their therapeutic value. Of those artifically produced, that which contains the pyrophosphate of iron is the best, and does not irritate even a delicate stomach. The natural chalybeate waters are also bottled and sent to distant places. They are often more effective if drunk upon the spot, but merely because, in that case, the general hygienic surroundings of the patient are better than they would be at home. The best-known and most popular ferruginous springs in Germany and Switzerland are situated at Cudowa, Rippoldsau, Homburg, Eister, Schwalbach, Pyrmont, Driburg, Liebenstein, St. Moritz, and Tarasp. "Steel baths" are also much employed; but the effect is not due to the iron contained in the water, but to the temperature of the water and the carbonic-acid gas it contains. Baths generally seem to be of distinct service in chlorosis, and hence we often prescribe simple baths or salt baths, giving a bath of fifteen or twenty minutes duration, at a temperature of 90°–95° F., two or three times a week, adding five or six pounds of salt to the bath.

No other remedies for chlorosis approach iron in popularity. Arsenic is the only one which needs to be mentioned. Its effects are often very beneficial, and it deserves a trial, especially in severe cases. It may be given alone or in combination with iron. Hydrochloric acid also deserves special mention, and often does good service in cases with digestive troubles, pressure in the stomach, etc. We give ten or fifteen drops of the dilute acid in a quarter of a tumbler of water, half an hour after meals. Scholz and Strübing recommend the use of sulphur—one part of purified sulphur to two parts of milk sugar—giving as much as will go on the point of a knife, three times a day. We must refer to the following chapter for the experiments with subcutaneous injections of blood.

If there be constipation, it is desirable to remedy it rather by suitable diet than by laxatives. Thus we may prescribe fruit, Graham bread, and other foods which promote peristaltic action by the mechanical irritation which they exert.

CHAPTER II.

PROGRESSIVE PERNICIOUS ANÆMIA.

(Grave Form of Essential Anæmia.)

We term pernicious anæmia that variety of essential anæmia which does not, like chlorosis, terminate in recovery, or maintain a low degree of severity, but which goes on uninterruptedly from bad to worse, in many instances leading to a degree of anæmia which of itself proves the immediate cause of death. The word "essential" is here used to signify that the anæmia is a primary one, the result of some pathogenic cause acting upon the blood itself, or the hæmatopoietic system. We must hold fast to this conception of the disease, especially as the attempt has been repeatedly made of late to degrade pernicious anæmia from the rank of an idiopathic disease, and regard it as merely a grave form of secondary anæmia which may result from any one of the most various causes.

Of course, the diagnosis of primary anæmia may be wrongly made in cases where a more careful search will detect some special cause for the condition. Such a mistake may become evident at the autopsy. Thus, in repeated instances, cases which have been during life regarded as "pernicious anæmia" have been shown to be cancer of the stomach (*vide* page 391). In other cases, however, it is much more difficult to discover the primary cause of the anæmia. Thus the profound anæmia which attacked the men at work upon the St. Gothard Tunnel was at first regarded as idiopathic, but more careful investigation showed that the disease was really due to anchylostoma (*vide* page 448), the ravages of which parasite have in other cases also been confounded with the symptoms of pernicious anæmia. Reyher and Runeberg have lately called attention to the fact that the presence of bothryocephalus latus in the intestine may produce an affection resembling severe and apparently primary anæmia. Of late there have been a number of cases reported where the post-mortem examination showed that the progressive marasmus and anæmia of the patient had resulted from an extensive atrophy of the coats of the stomach and intestine, associated in some instances with well-marked lesions of the sympathetic plexus. These cases are very interesting, but have nothing whatever to do with pernicious anæmia, except in so far as the changes are of a secondary nature, analogous to the fatty degeneration seen in other organs (*vide infra*). Often the symptoms even during life differ not a little from those of pernicious anæmia, so that we can not think it right to establish what is called a "gastro-intestinal variety of pernicious anæmia." It is proper to group and classify the forms of secondary anæmia according to their various causes, but not the cases of primary idiopathic anæmia, which are all essentially alike.

The credit of having been the first to study pernicious anæmia as a special form of disease belongs to Biermer (1868), although occasional instances of the disease had long before been noticed. Its occurrence during pregnancy was first pointed out by Gusserow.

[The puerperal form of pernicious anæmia was described by Walter Channing in 1842.]

With regard to the special ætiology of progressive pernicious anæmia, it must be confessed that we have as little definite information as about the ætiology of chlorosis. Klebs, and more lately Frankenhäuser, have discovered micro-organisms in the blood, to which they have given the names "*cercomonas globulus*" and "*cercomonas navicula*"; and they ascribe to them a pathogenic influence. These discoveries, however, have not yet been satisfactorily confirmed. It is a noteworthy fact that pernicious anæmia is much more frequent in some countries than in others; thus, it is seen far more frequently in Switzerland than in North Germany. This would be one argument for assuming that the disease is an infectious one. In Leipsic, pernicious anæmia was comparatively frequent several years ago, while of late it seems to have become decidedly less common. There do not usually seem to be any special exciting causes. It has been maintained that unfavorable hygienic surroundings and insufficient nourishment promote the development of the disease; and this would apply perhaps to some cases, but certainly it does not to all. We are inclined to regard it as characteristic of this disease, as well as of chlorosis, that the anæmia may develop despite the most favorable outward circumstances. There is, however, one factor which seems to have a decided influence, and that is the condition associated with pregnancy and childbirth. The first symptoms of pernicious anæmia in women are very apt to date from this period. It is a very interesting but somewhat puzzling fact, that the disease may appear as a sequel to a great loss of blood, whether from one or several hæmorrhages. It seems as if the body were sometimes unable to recover from the effects of large hæmorrhages of this sort, so that the acute merges into a chronic and progressive anæmia, which, despite nursing and treatment, advances to a fatal termination. It is quite doubtful, however, whether such cases should be classed as genuine pernicious anæmia.

Most of the cases occur during middle life, between twenty-five and forty years of age. Both sexes are equally liable to the disease, except for those cases already referred to which seem to be especially connected with the sexual functions.

Pathology.—Pernicious anæmia so often terminates fatally that there has been abundant opportunity to make accurate anatomical investigations of the lesions it produces. We shall not here discuss the changes in the blood itself, inasmuch as they will more properly be described among the clinical phenomena. The changes in the internal organs are divisible into two groups: First, such as are secondary and the result of the anæmia; and, secondly, such as may perhaps be primary and pathognomonic. All the internal organs are, of course, anæmic. Another striking change is fatty degeneration. This is usually best marked in the heart, but it also affects the kidneys, liver, and the walls of the stomach and intestines, as well as the intima of the blood-vessels (compare page 934). We have already pointed out that this fatty change is to be regarded as the direct result of the anæmia, or more particularly of the diminished supply of oxygen in the tissues; and we have also explained that the destruction of albuminoids indicated by this fatty degeneration maintains a direct ratio to the increase of nitrogen in the urinary secretion.

The second important lesion found at the autopsies of those who die of pernicious anæmia is the frequent hæmorrhages, usually small, but occasionally large; these are found in all sorts of locations. The most important of these hæmorrhages are into the retina, because they can be demonstrated by means of the ophthalmoscope during life. They are very frequent. We may also find minute hæmorrhages in the serous membranes, such as the pleura or pericardium, in the brain and in the mucous membranes. Cutaneous ecchymoses are also occasionally found. As we shall note later, the hæmorrhages are probably due to little capillary embolisms caused by the formation of fibrine ferment in the blood.

Still another secondary symptom is an abundant deposit of iron in the cells of many organs. This can usually be determined only by the aid of the microscope or micro-chemical tests. It is most abundant in the peripheral zone of the hepatic lobules, but it also occurs in the kidneys, pancreas, and other organs. Quincke has studied this point very carefully, and finds that the total amount of iron contained in the liver in pernicious anæmia is much larger than normal. The most natural and most probable interpretation of this fact is that the iron originates from the destruction of large numbers of red blood-corpuscles.

The changes thus far described are the result of anæmia, and hence are the same in all severe anæmias, whatever their cause. On the other hand, search has been made for specific changes which could be regarded as the primary disease, and thus, of course, our attention has been especially directed to the organs engaged in the formation of the blood. The lymph-glands do not usually present any special changes in pernicious anæmia. If they are much altered, it is probable that the disease is quite a different one, which we shall later have an opportunity to study (*vide* pseudo-leukæmia). The spleen is in many cases normal. In exceptional instances, however, it is decidedly enlarged, although never enormous. Even when the organ is enlarged there are no important histological changes to be detected. Cases of pernicious anæmia with marked splenic tumor are often termed splenic anæmia (*vide infra*), but we do not ourselves see how such cases differ essentially from others in which the spleen is not enlarged. The changes in the bone-marrow are by far the most constant. This structure plays a much more important part in the formation of the blood than does the spleen. C. Wood, and after him Cohnheim, called attention to the fact that the bone-marrow is almost invariably affected in pernicious anæmia. Instead of its normal yellow color, it has a reddish-purple appearance. This is due mainly to the fact that the numerous fat-cells of the marrow are all, or nearly all, destroyed. This is the more surprising as the fatty tissues in other parts of the body are often very little affected in pernicious anæmia. The specific cellular elements of the marrow also exhibit certain changes. There is a decided hyperplasia, and often, although not always, there are found large numbers of nucleated red blood-corpuscles. Cohnheim is inclined to regard the disease of the bone-marrow as specific and primary, but it must be confessed that there are many reasons which strongly oppose this view, and suggest the possibility of this change in the marrow being merely a secondary phenomenon, a sign of the extremely active formation and regeneration of the red blood-corpuscles. Neumann's investigations would lead to the belief that the nucleated red blood-corpuscles are young blood-cells in the process of development. Furthermore, these vigorous processes of regeneration, and the corresponding changes in the bone-marrow, are often found in cases of profound anæmia which are beyond a doubt of secondary origin.

We can not, therefore, feel certain that the lesion of the bone-marrow is the primary anatomical disturbance, and we are therefore obliged to assume that essential anæmia may be a disease of the blood itself—that is, some process which does direct injury to the red blood-corpuscles, possibly of an infectious character (?).

Symptoms.—As already stated, the symptoms of pernicious anæmia usually begin, independently of any demonstrable cause, in individuals previously healthy, and develop so slowly and gradually that it is hardly ever possible to determine the precise date of the commencement of the disease. This is still more likely to be the case if the trouble occurs, as indeed it may, in individuals who were previously feeble and pale without being actually ill. Occasionally a more acute onset is observed in pregnant women.

The first symptoms are almost invariably traceable to the incipient anæmia. They include the subjective disturbances and the objective changes which are seen in ordinary chlorosis. The patient feels languid and is easily fatigued, is liable to headache, vertigo, palpitation, and tinnitus aurium; there are anorexia, frequent nausea, and, above all, a striking pallor of the skin and mucous membranes. While these symptoms, however, usually remain of slight or moderate severity in chlorosis, they attain, in pernicious anæmia, the greatest conceivable intensity. Of course, it would be impossible to describe all the stages through which the patient passes, but we append a sketch of those symptoms which are almost sure to be present, in greater or less completeness, in any well-marked case of pernicious anæmia.

If the anæmia be profound, the patient may be so weak as to be confined to the bed. If he sits up for any length of time, symptoms of increasing cerebral anæmia develop, and he is apt to faint. Usually the patient lies upon his back, with his head rather low, and presents a countenance of waxy pallor. Very frequently a slight but distinct yellowish hue may be detected. Occasionally there are cutaneous ecchymoses, but those are exceptional. The mucous membrane of the lips, the gums, and the conjunctivæ are likewise pale and colorless. The intellect is unimpaired, but all answers to questions are slow, apathetic, and delivered in a low and feeble tone of voice. The patient is usually incapable of any great mental exertion. If the body be moved, and especially if an upright position be substituted for a horizontal one, there is great liability to syncope, as already mentioned. This may result from other slight physical exertions, and is often accompanied by a peculiar spasmodic rigidity of the body. The main subjective symptoms are weakness, and more especially intense headache; this often assumes a pulsating character, and is located mainly in the temples or forehead. There is also an annoying ringing in the ears, described as a rushing or roaring sound. There are certain other subjective sensations, namely, nausea, a sense of thoracic oppression, and pain in the bones, which will be further considered below.

If we now proceed to a systematic physical examination, we are struck, in the first place, by the condition of the eyes. The pupils are often somewhat enlarged, but react in a normal manner. Vision is often disturbed by spots before the eyes. The anæmic amaurosis seen after a sudden large hæmorrhage has not yet been observed in pernicious anæmia. The ophthalmoscopic examination of the fundus has very great diagnostic importance. We find in a majority of cases, although not in all, retinal hæmorrhage. There may be either one or many hæmorrhages. If they are extensive enough to involve the macula lutea or the disk, they may disturb vision greatly. Retinal hæmorrhages invariably signify that the anæmia is profound, and are, with considerable justice, regarded as distinguishing pernicious anæmia from chlorosis.

Respiratory Symptoms.—Breathing is usually accelerated, and, in the most advanced cases, is often remarkably deep and noisy (anæmic dyspnœa, *vide supra*). Sometimes there is a very annoying and almost painful sense of thoracic oppression, which is evidently connected with the dyspnœa. There is a "hunger for air." Physical examination of the lungs gives negative results. Sometimes there is a little cough, and there may be sufficient hæmorrhage into the mucous membrane of the air-passages to give rise to a slight hæmoptysis. Even in this case no anatomical changes can be detected during life. We may also mention in this connection that epistaxis is not very infrequent.

Phenomena referable to the circulatory system are of still greater clinical importance. The area of cardiac dullness is usually normal, although sometimes slightly increased. Upon palpation, we often find that the heart's action is exaggerated, and that its beat is felt over a larger area than normal. The pulse is usually decidedly rapid (100–120), but it is regular, and sometimes, but by no means invariably, small. It is often surprisingly strong. The loud "anæmic murmurs" are very characteristic. They can be appreciated at the apex of the heart, but are usually still better heard at its base. We generally hear a loud *bruit de diable* in the veins of the neck.

Digestive Organs.—The tongue is usually pale, smooth, and dry. The appetite is often very poor. The most prominent disturbances, however, are not due directly to the condition of the stomach, but they result from the cerebral anæmia —that is, they are symptoms of the irritation of the nervous centers. We refer to eructations and vomiting, which may be very frequent and distressing. There is usually a tendency to constipation. Occasionally there is diarrhœa.

The liver is usually normal; as is also the spleen in many instances, while in others it is shown by percussion and palpation to be enlarged. It is sometimes possible, as we can ourselves bear witness, to observe an enlargement of the spleen increasing as the anæmia grows more profound, and again decreasing if improvement occurs (*vide infra*). On ordinary examination, the urine does not usually differ essentially from normal. With few exceptions it is free from albumen, and it never contains sugar. As already set forth, however, accurate quantitative analysis often furnishes important evidence of changes in tissue-metamorphosis resultant upon the anæmia (cf. page 934). We will merely mention once more the comparative increase in the amount of urea excreted, and the occasional excess of uric acid. Sometimes the urine gives an unusually vivid reaction for indican.

In regard to what has already been said about the bones, it is an interesting fact that they are very sensitive to pressure in many cases of pernicious anæmia.

The sternum in particular is painful upon light percussion; and sometimes pressure will cause pain in the bones of the extremities. In rare instances swelling of the knee and other joints has been observed.

The blood has been made the subject of numerous and careful investigations; nevertheless, no characteristic change has been discovered. The changes presented in pernicious anæmia likewise occur in cases of profound secondary anæmia. This seems the more readily intelligible to us because of the view which we have already expressed on page 930 with regard to the origin of secondary anæmia. To the naked eye the blood seems extremely pale and watery. The number of red blood-corpuscles is sometimes so diminished that it seems incredible that life should persist. It is not at all unusual, in the last stages of the disease, to find less than 500,000 red blood-corpuscles per cubic millimetre—that is, about one tenth of the normal amount. The red blood-corpuscles are found to present striking varieties in size and form (*vide* Fig. 118). While some corpuscles have a normal appearance, others may be of an unusually large size (macrocytes). These "giant corpuscles" appear normal except in dimensions, and some observers (Laache) have thought that they were even possessed of an unusual amount of hæmoglobin. It is therefore surmised that they represent an effort on the part of nature toward compensation. In contrast with these large cells are found a varying number of minute red cells of a spherical shape; these were first described by Vanlair and Masius, and called microcytes. Their mode of origin and significance are not known. Finally, there are numerous red blood-corpuscles of abnormal shape. These Quincke was the first to notice. They present remarkable forms, being biscuit-shaped, hammer-shaped, or anvil-shaped, and so on, as illustrated in the accompanying cut. These "poikilocytes" are found in perfectly fresh undiluted blood, so that there is no reason to suppose that they are artificial products. Both the microcytes and poikilocytes are at present generally regarded as blood-corpuscles which have undergone imperfect or abnormal development, or suffered changes due to disease. Nucleated red blood-corpuscles have also been seen by Ehrlich a number of times during life. The white blood-corpuscles are not usually increased in number; in occasional instances, however, a temporary leukocytosis has been found. "Granule-masses" are often found in considerable abundance. Chemical examination of the blood has not as yet brought to light any facts of great importance. Of course there is a great diminution in the total amount of hæmoglobin. The amount of albumen in the blood-serum remains nearly normal.

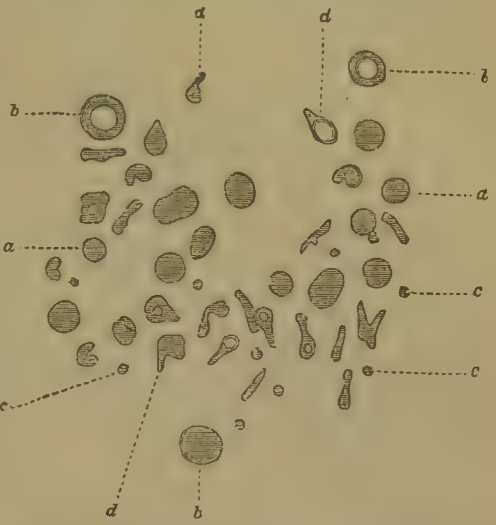

FIG. 118.—Changes in the red blood-corpuscles in pernicious anæmia. (From QUINCKE.) *a.* Normal blood-corpuscles. *b.* Macrocytes. *c.* Microcytes. *d.* Poikilocytes.

We observe in pernicious anæmia the tendency to fever common to all varieties of profound anæmia. In many cases the evening temperature will for weeks reach 100°–101° (38°–38·5° C.), or even higher. Previous to death, however, the temperature may become subnormal, falling to 86° (30° C.), or even lower.

As to the origin of all these symptoms we must, as we have said, regard the impairment of the blood, especially its poverty in red blood-corpuscles, as the essential morbid process. How this impairment is produced, whether by injury to the blood-corpuscles themselves or to the place where they are formed, we do not know. A large part of the other morbid symptoms—the pallor of the skin, the weakness, the "anæmic cerebral symptoms," tinnitus, vertigo, nausea, faintness, etc., are due directly to the anæmia. In regard to certain other important symptoms, however, another factor is probably of great significance, namely, an auto-intoxication of the body with fibrine ferment.

By the destruction of many red blood-corpuscles, probably a certain amount of hæmoglobin is always set free, and enters the blood plasma. There is then a hæmoglobinæmia. From many experiments, however (Dorpat school of Alexander Schmidt, Ponfick, Silbermann, etc.), we know that hæmoglobin dissolved in the blood destroys the white blood-corpuscles, and in some way produces from them fibrine ferment. Chronic "ferment intoxication" of the body causes certain symptoms, which are almost never absent in pernicious anæmia—capillary hæmorrhages and fever. The hæmorrhages are usually associated with embolism of the smaller vessels, seldom with local thrombi. Many severe nervous symptoms may perhaps also be referred to this intoxication.

General Course, Duration, and Prognosis.—As the very name "pernicious" indicates, the disease generally terminates unfavorably. Death usually seems to be the direct result of the extreme anæmia; special complications are exceptional. The disease often maintains a slow but gradual progress to the end. Its duration, reckoning from the appearance of the first symptoms, may not exceed three to six months. It may even run its course in a still shorter time. It seldom lasts more than a year. Sometimes its course is interrupted; there may be an arrest of the process, or improvement, or even apparent recovery. Usually, however, there are fresh relapses. In a certain class of cases the disease lasts two or three years, and is marked by a number of "attacks of anæmia" so intense that the subsequent improvement of the patient seems simply marvelous. It is in cases of this sort that splenic tumor has been made out at the time the anæmia was at its height; yet we do not perceive the necessity of establishing "splenic anæmia" as an affection essentially different from progressive pernicious anæmia. It is merely a clinical variety of the disease under discussion. Apparently, it likewise has an invariably fatal termination.

Permanent recovery may occur in cases of idiopathic anæmia so profound that we are at first inclined to regard them as pernicious. These cases are unfortunately very rare, and even where there is a marked improvement the danger of a relapse is to be borne in mind. The prognosis is therefore always very grave, if not absolutely unfavorable. Of course, general hygienic surroundings and good care may exert some influence upon the course of the disease. It is noteworthy that if pregnancy be complicated by profound anæmia there is a great liability to premature delivery, after which there is often a rapid change for the worse. There are exceptions to this rule.

Diagnosis.—It is seldom difficult to make out the existence of a profound anæmia, or to determine the degree of danger which the consequent symptoms indicate. We have, however, the same difficulty here as in chlorosis in proving that the anæmia is primary and idiopathic. The factors essential to this diagnosis have been already indicated. We should bear in mind the possibility of incipient tuberculosis, organic diseases of the stomach, or such parasites as the anchylostoma and tapeworm.

Treatment.—For treating progressive pernicious anæmia we have only the same remedies as for the benign variety of anæmia. Abundant and suitable nourishment is requisite, and all hygienic influences should be carefully regulated. Internally, our main reliance is to be put upon the preparations of iron. With us, a favorite remedy in pernicious anæmia is the tinctura ferri chlorati ætherea (analogous to tinctura ferri chloridi, U. S. P.), of which ten drops in sweetened water may be given several times a day. It would also be extremely advisable to try the effect of arsenic. This remedy sometimes accomplishes striking results in the whole group of blood diseases, including leukæmia and pseudoleukæmia as well as anæmia. It is much better administered in pill form than in Fowler's solution. Iron may be given at the same time with arsenic. Some authorities recommend phosphorus.

If the case be not too far advanced, baths may prove useful adjuvants to the internal treatment. Salt baths or artificial carbonic-acid baths may be employed. Symptomatic treatment is often indicated: the dyspepsia may call for dilute hydrochloric acid, or the troublesome vomiting may require bits of ice, bromide of potassium, or opium.

The transfusion of blood has been employed in pernicious anæmia. Sometimes the effect seems to be favorable. Experience thus far, however, would not lead one to expect very great benefit from it. Von Ziemssen has lately recommended in all severe forms of anæmia the subcutaneous injection of blood. It is done by injecting under the skin, by thoroughly disinfected instruments, fifty cubic centimetres of defibrinated human blood, divided into two parts, twenty-five cubic centimetres being injected into each thigh. The point of injection is vigorously rubbed, and thus the injected blood is forced into the lymph-channels. In severe cases this procedure should be frequently repeated. Experiments on this method are thus far very favorable. Not only the subjective symptoms, but the objective condition of the blood, the number of red blood-corpuscles, sometimes show a striking improvement after the injection.

[In the experience of American and English clinicians, arsenic, long continued and in as large doses as are tolerated, is of far greater value than iron.

The inhalation of oxygen is expensive, but seems to be of distinct service in some cases of grave anæmia of all forms.]

CHAPTER III.

LEUKÆMIA.

(Leucocythæmia.)

Definition and Ætiology.—Virchow, in 1845, was the first to obtain an insight into the disease leukæmia (" white blood "). He detected the great increase of white corpuscles occasioned by it, and from this time these constituents of the blood were subjected to observation in all sorts of diseases. It was soon found that there may be a temporary increase of the white corpuscles in various primary diseases, and that in certain instances this increase may actually constitute the essential symptom. In this latter case the increase is due to a depraved condition of certain internal organs. In the temporary cases, where the increase of white blood-corpuscles is usually not very great, we find one white corpuscle to one hundred red, or even one to fifty—the normal ratio being one to six hundred or more. This is usually termed leukocytosis, in distinction from leukæmia proper. Leukocytosis is most frequent in acute infectious diseases, such as typhoid fever, recurrent fever, intermittent fever, and pyæmia, and is also often seen in anæmia.

Genuine leukæmia is a rather rare disease. Its characteristics are well marked in most cases, but of its true nature we remain entirely ignorant. In a majority of the cases the change in the blood is associated with marked changes in the spleen and the bone-marrow, and often also in the lymph-glands. The organs just enumerated being concerned in the manufacture of the blood, it is very reasonable to suppose that leukæmia is a disease which primarily affects these organs, and that the increase in white corpuscles results from the disturbance thus occasioned. The cause of the disease in the organs mentioned is as yet unknown. Various authors have suggested that there may be some specific infection, but they have not been able thus far to produce any evidence of the truth of their surmise. In few cases can we discover even any exciting cause. The illness seems to develop spontaneously in perfectly healthy persons. In some cases, on the other hand, leukæmia does seem to be a sequel of some other disease. Thus it is occasionally preceded by a tedious attack of intermittent fever. It has also been asserted that syphilis and other infectious diseases, such as typhoid, may give rise to leukæmia, but this is not very probable. Finally, trauma of the spleen or bones has in repeated instances been regarded as the occasion of the disease.

The hygienic surroundings of the patient have also been regarded as causes of leukæmia. The disease is more frequent among the poorer classes than among the wealthy; but there are numerous exceptions to this rule. Ætiological importance has also been ascribed to anxiety, trouble, and mental depression in general, but with how much justice is doubtful.

Leukæmia is most common in middle life. between thirty and forty-five years of age, but well-marked cases have been observed repeatedly even in childhood, as also, though less frequently, in old age. Men are somewhat more liable to the disease than women. It has been repeatedly stated that in female patients the disease is sometimes referable to sexual derangement, but this has not been proven.

Pathological Anatomy.—The pathognomonic change in leukæmia is an increase in the number of white blood-corpuscles; this may be so considerable that there may be one white to three red, or even one white to two red corpuscles. The characteristics of the blood can be studied during the life of the patient, and upon them the diagnosis is mainly based. They will therefore be discussed under symptomatology, while we shall here confine ourselves to the lesions presented by the spleen, bone-marrow, and lymph-glands.

Of the organs just enumerated, the spleen is the one most frequently affected (splenic leukæmia). It is often greatly increased in size, not seldom attaining a weight of six to thirteen pounds (3–6 kilogrammes) and a length of a foot (30 ctm.). There is a true hyperplasia of the whole organ: all the histological constituents are increased. The cut surface is usually a rather vivid red in early cases, but later on it often has a lighter, yellowish color. The consistence is usually diminished, but in the later stages it may be greater than normal. Upon microscopic examination, we find enlargement of the blood-vessels and a great increase in the cells of the pulp and of the follicles. Sometimes the hyperplasia of the follicles predominates, giving the spleen a spotted appearance, like marble. In such cases the pulp usually presents retrograde metamorphosis, with atrophy and fatty degeneration of its cells and deposits of pigment. In advanced cases a considerable amount of firm connective tissue may be present. There are often hæmorrhagic infarctions, presenting the appearance of circumscribed spots, dark red, or in the later stages brownish yellow, in color.

Lesions of the bone-marrow are next in frequency to those of the spleen (medullary and myelogenous variety of leukæmia). Neumann and a few other authorities regard changes in the bone-marrow as an essential lesion, and hold that they can be demonstrated in every case of leukæmia. There would certainly seem to be exceptions to this last rule, but nevertheless in a majority of cases the marrow does present a peculiar yellowish or almost puriform appearance. By means of the microscope we can detect a great increase in the lymphoid cells of the marrow and the presence of a considerable number of nucleated red blood-corpuscles.

In many cases the lymph-glands remain perfectly normal; but in others they become considerably enlarged, and form actual tumors in various parts of the body, such as the axilla, neck, and groin, and sometimes the internal lymph-glands. Such cases are examples of lymphatic leukæmia. Histologically, the change here is simple hyperplasia of the glandular tissue.

These three forms of leukæmia—splenic, myelogenous, and lymphatic—can not be regarded as distinct diseases, inasmuch as cases occur which present all sorts of combinations of these different lesions. Purely myelogenous cases are very rare, if they occur at all; and we rarely meet with cases which are purely splenic or purely lymphatic. Most cases present lesions of the spleen and marrow conjointly. In less frequent instances splenic disease is associated with that of the lymphatic glands. The fact that these combinations exist indicates that there is one common cause for the disease, which assails sometimes one, sometimes two, or sometimes all three of the organs named.

Just what the connection is between the changes in the blood and the lesions of these various organs is an unanswered question. A view which seems to us very plausible regards the disturbance in the spleen, or marrow, or lymph-glands, as the case may be, as the primary one, and the alteration in the blood as a result of this primary disturbance; there is an increase in the number of colorless corpuscles formed, and a consequent increase in the number of them introduced into the circulatory fluid. Some authorities have taken for granted that the colorless blood-corpuscles normally undergo transformation into red blood-corpuscles, and believe that in leukæmia this metamorphosis is hindered; but the assumption lacks support. It is true, however, that the number of red blood-corpuscles is diminished in leukæmia. This diminution in number might be ascribed either to scantiness of supply or to increase in the processes of destruction, but which factor is in reality the important one must remain undetermined.

Changes in Other Organs.—Leukæmia sometimes causes new growths of a lymphatic character in certain organs other than those already mentioned. The growths may either be diffuse or circumscribed and nodular. They are observed in the tonsils, Peyer's patches, and the intestinal follicles. They are also very frequent in the liver, kidneys, and retina, and more rarely in the lungs and the pleura. These various lesions may be regarded as in a certain sense analogous to the metastatic tumors of cancer or sarcoma, and suggest the possibility of the diffusion of the pathogenic poison throughout the whole body. In one or two cases a well-marked leukæmia has been found independent of any demonstrable organic lesions. It is impossible, at present, to explain such occurrences. Leube and Fleischer have reported a case of this sort, and are inclined to believe that the blood itself was diseased. There is little known as yet as to changes in the chemical characters of the blood and viscera in leukæmia. Xanthine and hypoxanthine have been found in the blood, as have also lactic acid and formic acid. It is also noteworthy that octahedral crystals are often found after death in the blood, spleen, and marrow, and in other parts. These are known as Charcot's crystals, and have already been described as occurring in the sputum in certain pulmonary diseases (compare page 169).

Symptoms.—The clinical phenomena of leukæmia are in many respects similar to those of progressive pernicious anæmia, and need not be enumerated again here. In leukæmia, however, we have, in addition, symptoms referable to the spleen or lymph-glands or bone-marrow (as the case may be), and also the characteristic alterations in the blood. The blood-changes, being pathognomonic, demand a full description.

The pallor and watery character of the blood in leukæmia are noticeable even to the naked eye in advanced stages of the disease. They can not, however, be distinguished without the aid of the microscope from the changes present in grave anæmia (*vide* Fig. 119). Looking through that instrument, we frequently perceive at once an enormous increase in the number of the white blood-corpuscles. As already stated, there may be almost as many white as red corpuscles. The size of the white corpuscles varies in different cases, and also in the same case. Virchow has called attention to the fact that the smaller cells originate mainly in the lymph-glands, and are therefore especially numerous where the leukæmia is of a lymphatic type. The larger cells are referred mainly to the spleen and the marrow. The marrow is also said to contribute certain extremely large nucleated cells, the dimensions of which considerably exceed those of the normal white blood-corpuscles. It is not always possible to determine the origin of the white cells from their size. Ehrlich has succeeded in making out various forms of white corpuscles by staining. What are called "eosinophilous cells" are especially increased in the blood of leukæmia. These are colorless cells, the granules of which take a deep stain with acid pigments, but not with basic. Coincident with this increase in the white cells in leukæmia there is almost invariably a considerable diminution in the number of red blood-corpuscles. We also find an occasional nucleated red blood-corpuscle in leukæmic blood, and sometimes also microcytes, poikilocytes, and almost always a large number of "granule-masses" interspersed between the blood-corpuscles.

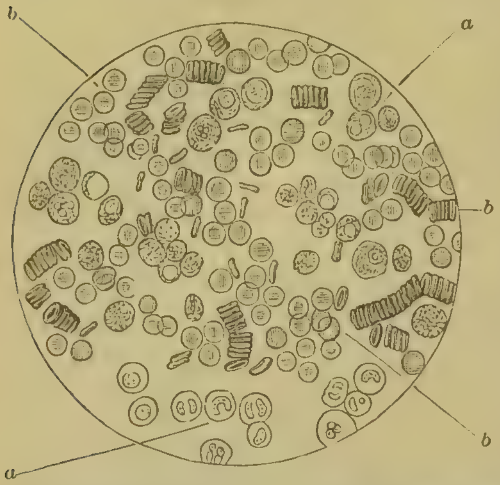

FIG. 119. — Anæmic blood. (From FUNKE.) *a.* White blood-corpuscles. *b.* Red blood-corpuscles.

Splenic tumor is the most frequent and important of the organic lesions produced by leukæmia. It is rarely possible to observe its development. In most instances the spleen is already large when the patient first comes under observation. It projects from under the ribs as a firm, hard mass, the lower and anterior extremity of which often extends to the median line of the body. The inner edge of the tumor is somewhat characteristic; it is rather sharp, and presents one or two notches. At first there is little subjective disturbance or pain referred to the spleen. Where the enlargement is very great, there is often an annoying or even distressing feeling of distention and fullness in the abdomen. Respiration may also be interfered with by the crowding up of the diaphragm.

The lesion of the bone-marrow can never be determined absolutely during life. The only symptom which renders its existence probable is pain in the bones, but even this is not an infallible symptom. There is seldom pain except upon pressure. It is usually brought out by percussion of the sternum, but there may be well-marked disease of the marrow without this "sternal pain."

As already stated, the lymph-glands often remain perfectly normal. If they are affected, the disturbance is betrayed by their increase in size. Not only may the glands in the neck, axilla, and groin be enlarged, but occasionally also those of the mesentery and retroperitoneum, as can be demonstrated upon palpation of the abdomen. The enlarged lymph-glands rarely cause severe pain, if any at all.

We have already referred to leukæmic new growths in other internal organs. These possess, for the most part, merely a scientific interest, as they cause no special symptoms. Sometimes hepatic enlargement occurs, as the result of a diffuse leukæmic infiltration. The changes in the retina associated with leukæmia are of importance, as they can be detected by means of the ophthalmoscope. The retina presents white spots or stripes running parallel with the blood-vessels. They are due to collections of lymphoid cells, or to actual lymphoid growths. These lesions have been inappropriately called leukæmic retinitis. Retinal hæmorrhages occur also in leukæmia, as in grave cases of idiopathic anæmia.

All the other clinical phenomena of leukæmia result from the abnormal condition of the blood, meaning thereby the anæmia. The ability of the blood to perform its normal functions is impaired mainly through the loss of red blood-corpuscles; the resulting symptoms are therefore precisely the same as in essential anæmia, and we do not need to describe them again. They usually are the most prominent symptoms of the disease, and include noticeable pallor of the skin, equal to that sometimes seen in anæmia; anæmic murmurs over the heart and the veins of the neck; general debility; anorexia, and digestive disturbances; palpitation and dyspnœa; and, finally, the whole group of the "cerebral symptoms of anæmia," that is, headache, vertigo, syncope, and tinnitus aurium. Sometimes there is a troublesome pruritus. We would also call attention once more to the frequent hæmorrhages. These must be due to impaired nutrition of the vascular walls, and sometimes justify our speaking of a "hæmorrhagic diathesis." Obstinate epistaxis is particularly frequent. Less often we have hæmorrhage from the intestine, stomach, kidneys, or into the skin or muscles, etc. We may have cerebral hæmorrhage, with hemiplegia, or sometimes immediate death, consequent upon it. Severe cases may present a slight œdema of the skin and serous effusions into the various cavities of the body.

The urine in leukæmia is essentially like that excreted in pernicious anæmia. Fleischer and Penzoldt have shown that in leukæmia as well as in pernicious anæmia there is increased destruction of albuminoids, and a consequent increase in the excretion of nitrogen. There is often also a considerable increase of uric acid.

The temperature is apt to undergo slight elevations, as in severe cases of anæmia. In advanced stages there may be quite high fever of an intermittent character, reaching 103°–104° (39·5°–40° C.). The fever is sometimes accompanied by chills, and when the temperature falls there is often a profuse and debilitating perspiration.

Complications are, on the whole, rare. Sometimes we observe pulmonary tuberculosis, or some acute intercurrent disease such as pneumonia. We saw one case end in death from hæmorrhagic angina and œdema of the glottis.

In regard to the pathogenesis of the different symptoms the conditions are manifestly very like those of simple severe anæmia. Poverty of the blood in red blood-corpuscles and chronic ferment intoxication are the most important factors. We may therefore refer to what was said on page 944.

Clinical History.—Leukæmia is almost always chronic in its course. The disease begins insidiously and progresses gradually; the patient grows pale, feels languid, and has slight and apparently insignificant symptoms, which gradually give place to alarming phenomena. The patient may himself notice the organic lesions incident to the disease. If the leukæmia be of the lymphatic type, he is apt to be struck by the swelling of the lymph-glands, while in splenic leukæmia his attention is attracted by the feeling of tension and pressure in the abdomen, the increasing prominence of the left side, and the unusual sense of resistance present in that part of the abdomen. Sometimes it is obstinate epistaxis, or hæmorrhage from some other source, which first attracts attention and leads to a careful examination of the blood and spleen.

The disease usually lasts several years. Many cases are rather mild and gradual in their progress, while in others there is a rapid development of all the symptoms. Some cases go on so rapidly, occupying but a few months, that they might almost be termed "acute leukæmia." Instances of this form are most frequently seen in children. Apparent arrest of the disease is frequent, as is also temporary improvement, followed by fresh exacerbations. The final termination is almost invariably unfavorable. Death is usually preceded by symptoms of the most profound anæmia, and is caused directly by the general debility. It is sometimes hastened by the occurrence of hæmorrhages, for instance, obstinate epistaxis or cerebral hæmorrhage.

Recovery is not absolutely impossible, but still it is very rare, and it is out of the question after the disease has passed its first stages. In an advanced case, therefore, the prognosis must be regarded as hopeless.

Diagnosis.—Leukæmia can be easily and unmistakably recognized by a microscopic examination of the blood. In a very early stage of the disease the increase of white blood-corpuscles may be so slight that a definite decision can not be made; but the later developments will afford absolute certainty in any typical case.

We can not fail to recognize a case of leukæmia if the blood be examined. Such an examination is therefore demanded in every case of obstinate anæmia, and, above all, in such patients as have a chronic enlargement of the spleen, or swelling of the lymph-glands in various parts of the body. The enlarged lymph-glands are readily recognizable. The splenic tumor can usually be diagnosticated from its characteristic position and shape, and especially from its internal edge, with the notches already spoken of. It may be simulated by hydronephrosis and other diseases causing enlargement of the kidneys, and in women by ovarian tumors. In cases of doubt the blood should be examined, and if the result be a positive one, we may feel certain of our diagnosis. If there is a chronic enlargement of the spleen without a leukæmic change in the blood, we must consider all the possible causes of such an enlargement; thus there may be a passive congestion of the spleen, with enlargement, as the result of hepatic disease, portal thrombosis, or disease of the heart, or a splenic tumor from malarial poisoning. Again, there are cases where we have the signs of a gradually progressive anæmia, apparently idiopathic, and a chronic enlargement of the spleen, or still more frequently enlargement of the lymph-glands in various parts of the body, without increase in the number of white blood-corpuscles. Such cases are termed pseudoleukæmia, and are described in the next chapter.

Treatment.—Nearly the same remedies are employed in leukæmia as in idiopathic anæmia. Of course the greatest attention should be paid to general nutrition. Of internal remedies, the preparations of iron have been mainly employed. They seldom produce any brilliant or permanent effects. We have much more confidence in the administration of arsenic, and this remedy should certainly be tried in large doses. It may be given either in the shape of pills, or, possibly with still more advantage, subcutaneously. Of course no permanent benefit is to be hoped for even from this, except in the early stages of the disease.

What are called "splenic remedies" have been often employed, but do not seem very effectual in leukæmia. Mosler obtained good results from the long-continued use of quinine (5–8 grains = 0·30–0·50 grm., or more, in twenty-four hours). He also recommends a trial of piperin and oil of eucalyptus:

℞ Olei eucalypti............................ gtt. 100;
Piperini,
Ceræ albæ.............................. āā ʒ j (grm. 4·0);
Pulv. altheæ........................... ʒ ij (grm. 7·5).
M. et fiant pilulæ no. c.
S. Three to five pills three times a day.

Local treatment of the spleen has also been attempted. If an ice-bag be constantly kept on the splenic region, it will sometimes diminish the size of the tumor, and may also relieve pain. Botkin has recommended faradization of the spleen, but we can scarcely expect any great benefit from such a procedure. Injections of quinine, arsenic, and other remedies, have been made into the substance of the spleen. We do not believe that this is advisable. The splenic tumor of leukæmia has actually been extirpated surgically; but the proceeding is so ineffectual and so fatal that it is now universally abandoned. The transfusion of healthy human blood has also been tried, but without satisfactory results. Subcutaneous injections of blood (*vide supra*) have been tried in only a few cases, but they may, perhaps, have good results. In some recent cases inhalations of oxygen have acted favorably.

Many other particulars of treatment have been mentioned under anæmia.

CHAPTER IV.

PSEUDO-LEUKÆMIA.

(*Hodgkin's Disease. Adenia. Malignant Lympho-sarcoma. Pseudo-leucocythæmia.*)

It was mentioned in the preceding chapter that there are cases in which the organic lesions are apparently the same as in genuine leukæmia, and yet there is little if any increase in the number of white corpuscles in the blood. There is generally, however, a diminution in the number of red corpuscles. These cases usually receive the name which Cohnheim gave them of pseudo-leukæmia. It is nevertheless doubtful whether they are to be regarded as a special form of disease, and there are various facts which indicate that they are at least very closely allied to genuine leukæmia. There is a great similarity in most of the symptoms of the two diseases and in their general course, as well as in the organic changes they produce. Furthermore, a case of pseudo-leukæmia may finally assume the character of genuine leukæmia, with its characteristic blood changes.

The purely splenic type of pseudo-leukæmia is the least frequent one. As yet very few such cases have been reported. There is a gradually increasing anæmia with the usual symptoms, and associated with these increasing enlargement of the spleen. It is impossible to draw any sharp dividing line between such cases and cases of pernicious anæmia attended with moderate enlargement of the same organ (splenic anæmia). It may be said to be a matter of taste which name the physician shall give to a case of this sort. The bone-marrow seems to present the same characteristics in splenic pseudo-leukæmia as in pernicious anæmia.

Pseudo-leukæmia Lymphatica.—Pseudo-leukæmia of a lymphatic type is a much more frequent and well-defined disease. It was first described in 1832 by the Englishman Hodgkin, and is sometimes called Hodgkin's disease. Wunderlich was the first in Germany to study the disease thoroughly; he described it in 1858 under the name of "progressive multiple hypertrophy of the lymph-glands"; and later Billroth termed it "multiple malignant lymphoma." Trousseau gave it the name of "adenia."

Little is known about the ætiology of lymphatic pseudo-leukæmia. It is said to be connected with malarial poisoning, syphilis, and some other diseases; but such statements are without a satisfactory basis. The tendency of late is to assign adenia to the group of infectious tumors, although the reasons for this belief are as yet purely theoretical. Pseudo-leukæmia is most common in young and middle-aged people, and is apparently rather more frequent in men than in women.

Pathological Anatomy.—The hyperplasia of the lymph-glands may be very great, producing large tumors of varying consistency. These have been called lymphoma, lymphadenoma, and lymphosarcoma. On section, the tumors display a white or grayish-red surface, and are seen to be made up of a number of swollen glands fused into nodular masses. Upon microscopic examination, we find a very abundant proliferation of lymph-cells, sufficient to obscure the reticulum of the gland completely. The new growth may even escape beyond the capsule of the gland and invade surrounding structures. Inflammatory adhesions often take place between the tumor and the overlying skin. There does not seem to be any essential difference between the harder and the softer varieties of these tumors.

These changes in the lymph-glands are often, though not invariably, associated with a swelling of the spleen. Its increase in size is usually slight. Lymphomata may also develop in the tonsils, the intestinal lymphatics, liver, kidneys, and other organs. Whether there are changes in the bone-marrow has not yet been determined.

The symptoms are very gradually developed. It is almost invariably the swelling of the lymph-glands which first attracts the attention of the patient or his physician. The glands upon one or both sides of the neck are usually the first to be enlarged, and they may finally grow to tumors the size of the fist, or even larger, producing great disfigurement. To the changes in the neck succeed swelling of the axillary, inguinal, and perhaps also the internal lymph-glands. The changes are gradual, and vary in their rapidity and extent.

At first the general health is hardly at all affected, but as the disease progresses its constitutional effects become more and more marked. The patient grows pale and languid, and finally presents all the symptoms of profound anæmia. We may also have certain symptoms due to mechanical compression occasioned by the growth of the lymphomata. The tumors in the neck may cause dysphagia, from compression of the pharynx and œsophagus; dyspnœa, from compression of the larynx and trachea; and perhaps alarming cardiac disturbance, from interference with the vagus. Hypertrophy of the bronchial glands sometimes occasions great difficulty in respiration; enlargement of the abdominal glands may produce ascites or jaundice; and enlargement of the glands in the groin may give rise to œdema in the lower extremities. In advanced stages we may have "cerebral symptoms of anæmia" precisely similar to those seen in genuine leukæmia or in pernicious anæmia. There may be a tendency to hæmorrhage and pruritus, and the urinary secretion and temperature may be abnormal. For particulars the reader is referred to the preceding chapters.

Upon examination of the blood, there are usually found the changes characteristic of ordinary anæmia, without increase in the number of white blood-corpuscles. Sometimes, however, there may be a slight leukocytosis; and sometimes, as already said, lymphatic pseudo-leukæmia may merge into genuine leukæmia. The examination of the blood must therefore be repeated from time to time. The spleen should also be examined. It is usually somewhat enlarged, and in some cases the enlargement may be considerable. Such cases might be properly denominated splenic-lymphatic pseudo-leukæmia. We may also discover a tenderness of the sternum or other bones.

The disease often lasts but a few months; it may, in rare instances, extend over two or three years or more. Recovery is not absolutely impossible in the early stages of the disease (*vide infra*), but at a later period the prognosis is absolutely unfavorable. The fatal termination results either from increasing debility and anæmia, or from the effects of mechanical compression, or from hæmorrhage, or from some intercurrent disease.

The diagnosis of pseudo-leukæmia is usually easy. It is to be based upon the objective signs and the condition of the blood. The disease is most apt to be confounded with swelling of the lymph-glands occasioned by tubercular infection; but in this latter case the changes are seldom seen in so many parts of the body, and the patient usually presents other indubitable evidences of tuberculosis.

Treatment.—We possess only one remedy capable of promoting absorption of the lymphomata, namely, arsenic. We have ourselves, in common with a great number of observers, had the most convincing evidence of the favorable influence of arsenic. It must, however, be given in sufficient doses: for example, a pill containing one fifteenth of a grain (0·004 grm.), or even a larger amount of arsenious acid, three times a day; and its use must be persisted in for a long time. It might also be tried subcutaneously. We have also seen apparent benefit from associating with the arsenic inunctions of iodoform (iodoform one part, vaseline fifteen parts) over the tumors.

In early stages decided benefit may be expected from this mode of treatment. At a later period we may obtain a decrease in the size of the tumors, but we can hardly hope for any permanent improvement. Operative interference is out of the question, except at the very beginning of the disease. Later on, it would be perfectly useless, and could seldom be carried out.

Other suggestions with regard to treatment may be obtained from the chapters on anæmia and leukæmia.

CHAPTER V.

HÆMOGLOBINÆMIA AND HÆMOGLOBINURIA.

Definition and General Ætiological Considerations.—If any cause produces a solution of the red blood-corpuscles in the blood-serum, hæmoglobin is excreted through the kidneys. The hæmoglobinæmia—the presence of free hæmoglobin in solution in the blood—excites hæmoglobinuria, i. e., the excretion of hæmoglobin in the urine. The causes of hæmoglobinæmia and its correlative hæmoglobinuria are manifold. In the first place, there are a whole series of poisons which, if introduced into the blood in sufficient amount, exercise a directly destructive influence upon the red blood-corpuscles, and thus excite hæmoglobinuria. To this list belong chlorate of potash (Marchand), pyrogallic acid and naphthol (Neisser), glycerine, toluyleudiamine, and many other substances. Distilled water is also in this sense a poison. Boström has discovered a fact of practical importance which deserves mention in this connection. It is, that a certain kind of mushroom (*Helvella esculenta*), when fresh, contains a poison which is capable of producing intense hæmoglobinuria, and such grave symptoms as jaundice, delirium, drowsiness, and tetanic convulsions, with perhaps a fatal termination. This poison is, however, so evanescent and so readily soluble in hot water that the mushroom becomes perfectly harmless if thoroughly soaked and then boiled, or if it has been dried.

Secondly, hæmoglobinuria may be developed in connection with infectious diseases. In this case, also, it may be referable to the action of poisons created within the system. Thus hæmoglobinuria has been observed in the course of a severe attack of scarlet fever or typhoid fever. Possibly malarial poisoning and syphilis may give rise to paroxysmal hæmoglobinuria. This question will be discussed later on.

There is a third mode of origin which also possesses practical importance. If blood from one animal be injected into another of a different species, hæmoglobinuria is almost sure to result. Not only do the injected blood-corpuscles undergo solution, but also the injected serum acts as a poison upon the original blood-corpuscles, destroying and dissolving them. This transfusion-hæmoglobinuria has been described by Prévost, Dumas, Ponfick, and Landois. It can be produced in human beings, as there was only too good opportunity to observe during the brief period when the transfusion of lamb's blood was in vogue. The practical deduction is evident, that we should not use for injection into the circulatory system of a patient anything but an unirritating salt solution, or human blood.

A fourth and very important ætiological factor is exposure to extremes of temperature. Hæmoglobinuria is a very frequent result of severe burns. The blood-corpuscles in that region of the periphery exposed to the heat are destroyed. Cold is capable of producing precisely analogous results. This is particularly evident in the cases of so-called paroxysmal hæmoglobinuria described by Wickham Legg, Lichtheim, and Küssner. [Raynaud's disease (see page 588) and hæmoglobinuria are associated in a sufficiently large proportion of cases to show some relationship between the two.]

Pathology and Symptoms of Hæmoglobinæmia, particularly the Paroxysmal Variety.—In most of the cases above enumerated, hæmoglobinuria is the result of an obvious or easily demonstrable cause. There is, however, another variety which appears paroxysmally in individuals who are otherwise perfectly well. Its symptoms are extremely characteristic. Although not a very frequent disease, there has been abundant opportunity to study it.

As just intimated, the disease is paroxysmal. Very often an attack is ushered in by frequent and persistent yawning. To this symptom are soon added pain in the limbs, headache, nausea, vomiting, and coolness of the periphery. The temperature speedily rises to 102° (39° C.) or more. With this is often associated a decided chill. Sometimes there is a violent, cramp-like pain in the hepatic region. Then the temperature falls again, perspiration appears, and the patient, although languid and depressed, soon recovers. A slight icteric hue can almost invariably be detected toward the end of the attack, the ordinary duration of which is from two to twelve hours. An eruption of urticaria is noticeably frequent in connection with the attacks.

The most interesting phenomenon of all remains to be described. We refer to the condition of the urine during and directly after the paroxysm. This secretion presents a dark brownish-red color resembling blood; it may even appear almost black. Its reaction is almost invariably acid; its specific gravity is usually rather low, say 1008–1012. On boiling the urine, the hæmoglobin is decomposed and a brown coagulum of albumen formed. If the excretion be examined through a spectroscope, we find the stripes characteristic of hæmoglobin, and sometimes also the narrow stripes indicative of methæmoglobin. It is therefore impossible to doubt the existence of hæmoglobin in the urine; and yet, upon microscopic examination, we find no red blood-corpuscles in the urine, or, in other words, no "hæmaturia." Frequently there are great numbers of opaque red granules in the urine, the shape of which is extremely irregular. These are, doubtless, granules of hæmoglobin. Some of them are free in the urine, some are attached to casts. Of the latter, we find hyaline and a few epithelial casts present. Sometimes masses of hæmoglobin assume the appearance of casts. The sediment may also contain a few cells of renal epithelium. The presence of this and of hyaline casts indicates that the kidneys have been slightly irritated by the excretion of hæmoglobin.

If now we examine the blood during a paroxysm, we shall find that hæmoglobinæmia is associated with hæmoglobinuria of the paroxysmal type, as well as with that occasioned by the action of various poisons. Küssner obtained blood from a patient during a paroxysm, and found that its serum had a ruby-red color, and contained hæmoglobin in solution. This proves that the destruction of blood-corpuscles takes place within the circulatory system. Indubitable tokens of this destructive process are to be seen upon microscopic examination of the blood during a paroxysm, especially when the paroxysm has been produced artificially in the manner described below. The red blood-corpuscles have little tendency to form rouleaux. They are pale, and many of them are irregular in shape (poikilocytes). Irregularly shaped flakes of hæmoglobin are also present, and often large numbers of decolorized red blood-corpuscles are to be seen. To these latter Ponfick has given the name of "shadows." Hæmoglobinæmia is also of the greatest clinical importance, because it probably must lead to a ferment intoxication of the body (*vide supra*, p. 944), to which a great part of the symptoms of the hæmoglobinuric attack, such as chill, fever, and nervous disturbances, must be referred. The origin of jaundice will be considered below.

In many cases the exciting cause of each separate paroxysm is perfectly evident. There is a cooling off of peripheral portions of the body—that is, the temperature of the blood in those parts is lowered (or the cold acts on the walls of the vessels ?) and this leads to a destruction of red corpuscles. Patients are free from attacks unless they have been out in cold or stormy weather, or have been wet through in a cold rain. As we might expect, the paroxysms are extremely rare in summer; they may, however, as Rosenbach showed by experiment, be artificially produced at any time, by exposing the surface of the body to severe cold, as by giving the patient foot-baths of ice-cold water. Ehrlich and Boas have both performed experiments which show that the action of cold is a purely local one. They have separated a finger of the patient from the general circulation by means of an elastic ligature, and then immersed it for a quarter of an hour in iced water. Blood taken from this finger invariably exhibited the above-mentioned changes, while the condition of the rest of the blood in the patient's body remained almost perfectly normal.

There can, therefore, be no doubt that in certain cases cold affects any portion of the peripheral circulation exposed to it in such a way as to excite a paroxysm of hæmoglobinuria. The paroxysms cause headache, fever, nausea, and other symptoms, the immediate origin of which is not perfectly understood. Several authorities believe that these phenomena are uræmic; and it has indeed been proven, both from the post-mortem appearances in such patients as have from some coincidence come to autopsy, and from experiments upon animals, that the kidneys may be plugged up with granules of hæmoglobin in sufficient amount to give them a dark-brown hue. The granules collect chiefly in the straight tubules, and also in the convoluted tubes and the glomeruli. Such a condition may present a considerable obstacle to the excretion of the urine, and more especially to the elimination of its solid constituents. In point of fact, the urine is usually of low specific gravity. This retention of urinary constituents in the blood certainly might produce some of the symptoms observed; but it is not certain whether we must not consider other influences.

No explanation has yet been offered for the liability of certain unfortunate individuals to paroxysms of this sort, from which most men are exempt. It may be mentioned that most patients have had a syphilitic history; so that a connection between these two affections is in many cases very probable (Murri). Paroxysmal hæmoglobinuria has also been observed in hereditary syphilis. It is doubtful whether hæmoglobinuria can have, as is claimed, any relation to malaria. We must note, however, that the individual attacks may be due not only to cold but sometimes to other influences, especially great physical exertion, long walks, etc. In conclusion, certain pathological facts should be mentioned. The kidneys are not the only receptacles of the *débris* resulting from the destruction and solution of the blood-corpuscles. Ponfick has been led by the result of certain experiments to believe that the spleen and liver are also affected. The spleen appropriates the undissolved remnants of the corpuscles, and, as a consequence, may undergo considerable enlargement. The liver absorbs a large part of that portion of the hæmoglobin which has undergone solution, and converts it into bile. As a result there seems to be an increased secretion of bile. Jaundice is probably due to local biliary stasis and biliary absorption in the liver itself ("hæmo-hepatogenous jaundice" of Afanassiew). It is not yet established whether a part of the hæmoglobin dissolved in the blood can be changed into bile pigment (true "hæmatogenous jaundice").

Prognosis.—When hæmoglobinuria is merely a symptom of other abnormal processes caused by poisoning or by some specific infection, the future of the patient depends entirely upon the severity of the primary disease. An attack of paroxysmal hæmoglobinuria would not seem to involve any direct danger to life. Recurrence of the paroxysms is always to be feared if the patient be exposed to those influences which produce it. There are no certain means of decreasing the patient's liability to attacks. In a few cases, however, where there had been syphilis, the paroxysms are said to have been permanently banished by mercurial inunctions. Likewise, if we suspect malarial influences, quinine should be tried.

No special treatment is required during the paroxysm itself. The patient must escape as soon as possible from the exciting cause; it is then advisable for him to lie quietly in bed, and to drink a large amount of fluid, so as to wash out the masses of hæmoglobin from the kidneys.

CHAPTER VI.

SCURVY.

(*Scorbutus.*)

Prefatory Remarks.—Scurvy is one of a group of diseases which may be termed "hæmorrhagic." They all have one predominant symptom, namely, a decided hæmorrhagic diathesis, respectively associated in the different diseases with various other more or less pronounced disturbances. This tendency to spontaneous hæmorrhage is in many cases, particularly the milder ones, more or less exclusively confined to the skin, but in numerous other instances hæmorrhage also takes place into the underlying tissues, such as the muscles or joints, as well as into the mucous membranes.

The distinctions between these various diseases are founded upon the manner in which the hæmorrhages occur and the symptoms which attend them. We may mention scorbutus, purpura simplex, purpura hæmorrhagica, and peliosis. It should, however, be stated that, although it is possible to distinguish several varieties of disease, each one of which presents a tolerably characteristic picture, there are innumerable transitional forms. It may, indeed, be almost a matter of taste in any particular case what name shall be applied to it. The existence of so many intermediate forms renders it evident that the various members of this group of diseases are at least closely related if not actually identical. We shall even find, upon careful consideration, that certain other diseases not usually regarded as hæmorrhagic are nearly akin to the group now under consideration. We refer to certain skin diseases, which are characterized mainly by inflammatory and exudative lesions of the skin. Chief among these should be mentioned erythema exsudativum multiforme, which not very infrequently exhibits some tendency to hæmorrhages, and thus presents external appearances closely simulating the forms of purpura.

In order to understand the underlying connection between these various disorders, a precise knowledge of their ætiology is requisite. Already considerable evidence has been gathered pointing to the importance of infectious influences in their production (*vide infra*), but no absolute proof has yet been obtained. In the meanwhile, we must be guided mainly by the purely clinical phenomena. These, again, indicate that sharp distinctions between the various hæmorrhagic diseases would be purely artificial. In this and the following chapters we shall discuss the two main types of hæmorrhagic disease.

Ætiology of Scurvy.—Scurvy is sometimes sporadic, and sometimes epidemic and endemic in its occurrence. There were formerly very extensive and fatal epidemics of the disease, at a time when the laws of health with regard to large aggregations of human beings were little regarded. The disease was prone to attack armies, or the inhabitants of besieged cities, or, especially, seamen. It was, and to a certain extent is still, one of the diseases most dreaded by the mariner. It has often swept away an entire ship's crew. To-day endemics of scurvy are by no means infrequent, although not so extensive as formerly. They are most apt to occur in prisons and similar institutions, and in barracks.

These facts, under the light of our present views in regard to such matters, would almost force us to seek some organic infectious poison as the origin of the disease. Formerly men were inclined to direct their sole attention to such circumstances as the character of the food, the dwelling, the climate, and similar conditions; nor can it indeed be denied that these hygienic factors do exert a decided influence upon the spread of the disease. It is, however, evident that they can not be its proper cause, for, beyond a doubt, scurvy may occur independently of any of the factors usually regarded as essential to its development. These causes must therefore be regarded as simply predisposing influences.

Great ætiological importance has long been ascribed to certain errors in diet. These include the use of bad or insufficient food, the undue predominance of certain kinds of food, and in particular of the salt meats so much employed on shipboard; or, again, the deficiency of certain varieties of food, in particular the lack of vegetable food, and still more of fresh vegetables. Much industry and acuteness have been expended in defending the theory that the lack of vegetable food is injurious because of the deficient supply of potassium salts under such circumstances (Garrod). Nevertheless, this view does not reach the heart of the matter, for in numerous epidemics of scurvy there has been no such lack of vegetable nourishment; and in some instances the diet employed has contained an unusual abundance of potassium compounds.

A like predisposing but not specific influence is exerted by the other factors to which ætiological importance has been assigned. They are indeed often present in epidemic as well as in sporadic cases, but, as previously stated, they may not exist at all. To this class belong damp and unfavorable quarters, cold, moisture, persistent heat, and excessive muscular exertion.

Age and sex exert no great influence upon the disease. Weakly persons seem to be somewhat more liable to be attacked than are the vigorous. The possibility of contagion has been maintained repeatedly, but contagion has not been proven to exist, and unprejudiced observation would incline one to doubt its existence.

[It seems probable that the dietetic causes of scurvy lie rather in a want of variety in the food than in the absence of any one class or order of foods. Wales says (Pepper, "System of Medicine"): "No single natural order contains plants that supply all the elements essential to the nutrition of the body and the right composition of the blood. The graminaceous and leguminous articles of food, for instance, are numerous but not various; they all afford the same or analogous albuminous elements, which have about the same nutrient value as the corresponding substances in animal food, and hence health and vigor can not be sustained on a diet of flesh combined with wheat, rice, and oatmeal, or with beans and peas, or with all of them together. Outbreaks of scurvy have occurred on shipboard where the ration is made up principally of these articles—as in Anson's ship when supplied with an abundance of fresh animal, farinaceous, and leguminous foods. It is clear, therefore, that, in order to obtain a variety of materials required in nutrition, we must resort to several of the natural groups, those particularly which comprise the succulent vegetables and fruits."

It is certain that scurvy is a disease which we can produce artificially, and that it is preventable in the vast majority of cases. It is now as rare among seamen as it was formerly common—a change which is the result chiefly of care to vary the diet, especially on long voyages. The United States law requires that lime- or lemon-juice, sugar, and vinegar shall be carried by all sailing vessels bound on ocean voyages or engaged in the fisheries, specifies the circumstances and minimum doses under which the antiscorbutics are to be given, and provides penalties for violation or neglect.]

Clinical History.—The disease does not usually begin suddenly. There is a gradual onset, marked by certain constitutional symptoms. The chief of these are languor and debility, a sense of thoracic oppression, palpitation, and usually a "rheumatic, dragging pain" in the loins and extremities, especially in the lower extremities. The patient is obliged to take to his bed if the case be at all severe; he is very sensitive to cold, and often is drowsy and apathetic. These somewhat indefinite premonitory symptoms last for a few days or a week, when other and more characteristic phenomena appear.

Among these new appearances are spontaneous hæmorrhages, occurring chiefly in the lower extremities. There are cutaneous hæmorrhages, producing dark-red macules of varying size, most of them with a hair-follicle in their center, and there are almost invariably hæmorrhages into the deeper tissues also. The subcutaneous connective tissue and muscles, and sometimes the periosteum, are affected. These deeper extravasations are a peculiarity of scurvy. They can sometimes be felt as hard, painful swellings in the parts affected, and are sometimes discernible from the discoloration of the skin, which soon results from the solution and diffusion of the blood-pigment. The patches present a diffuse bluish color, merging into greenish or yellowish at the periphery, and they are often quite large. They have a precisely similar appearance to "black-and-blue" spots resulting from injury. Of course, the more abundant and the more superficial the extravasation, the more extensive and darker is the macule. Similar appearances may sometimes be observed in the upper extremities and trunk, mainly in severe cases. The face and scalp rarely present ecchymoses.

Sometimes a hæmorrhage results in the necrosis and sloughing away of a portion of the skin. The necrosis is succeeded by ulceration ("scorbutic ulcers"). Under unfavorable hygienic influences this process may assume a grave significance. It should also be stated that we may observe other cutaneous disturbances, such as erythema, wheals, vesicles (the contents of which may be tinged with blood—"scorbutic pemphigus"), papules, and pustules. These eruptions are more frequent in some epidemics than in others; they may either be associated with or replace the cutaneous ecchymoses.

In the ordinary sporadic cases of scurvy which occur among us, hæmorrhages into the mucous membrane, except of the gums, are very rarely seen. The same is true of hæmorrhages from the stomach and other internal organs. In severe cases, during epidemics and under bad hygienic surroundings, it is otherwise; hæmorrhage may take place from the nose, stomach, intestines, bronchi, and kidneys, and blood may be effused into the serous membranes.

Next in importance to hæmorrhage is another peculiar symptom, presented by the mucous membrane of the mouth, and particularly that of the gums. In order to establish a diagnosis of scurvy in sporadic cases, we must demonstrate the existence of these two main symptoms—namely, the hæmorrhage into the skin or muscles, and the changes in the gums now to be described.

The scorbutic changes in the gums usually appear quite early in the course of the disease, being in many cases simultaneous with the hæmorrhages, although they may either precede or follow the latter. The gums assume a bluish hue, become swollen and spongy, are painful, and have a tendency to bleed. The changes are usually most pronounced in the salient parts of the gums between the teeth. It is a remarkable fact that they are hardly visible at all at places where there are no teeth; and the gums of very young children and of aged patients remain almost intact. In severe cases the gums are not only swollen but necrosed; the change is at first a superficial one, but it may extend inward and produce dirty-looking ulcers. Other parts of the mouth are liable to become involved in the ulceration, producing a diffuse ulcerative stomatitis, and giving the breath a most offensive odor.

Certain other local and constitutional phenomena are not infrequent, though less characteristic than the hæmorrhages and the alterations in the gums. Chief among the general disturbances is scorbutic anæmia. This is often in part referable to the unfavorable hygienic influences surrounding the patient, but the disease seems itself to impair the general nutrition. The patient looks pale, has a dry skin, and loses flesh rapidly. The temperature is often normal. Sometimes there may be an occasional rise of temperature, either in the beginning or in the further course of the disease. If complications occur, they are not infrequently accompanied by considerable fever.

Among more localized symptoms should be mentioned the premonitory sore throat which sometimes occurs. It is usually of the ordinary catarrhal variety, but it may assume a hæmorrhagic character. Bronchitis may also occur. Lobular pneumonia and genuine lobar pneumonia have been repeatedly seen in severe cases. Pleurisy, pericarditis, and inflammations of other serous membranes occasionally complicate the disease. They may likewise display a hæmorrhagic tendency in the exudations to which they give rise. Disturbances in the joints are sometimes seen, and are characterized by an effusion of liquid into the articular cavities, which effusion may be either serous or hæmorrhagic. This is a favorable opportunity to call attention to a peculiarity common to all the hæmorrhagic diseases and allied affections (*vide supra*)—they are apt to be associated with articular swelling.

The pulse may be somewhat accelerated, or may be slower than normal. It is usually small and compressible. Endocarditis may occur, but it is very exceptional. The blood does not present any constant and characteristic alterations in scurvy. The spleen may be decidedly enlarged, particularly in severe cases. Albuminuria has been repeatedly observed, but it is almost wholly confined to severe cases, in which, indeed, a typical acute nephritis may be developed.

Varieties of Scurvy. Prognosis.—The sporadic cases usually met with in this region almost invariably pursue a favorable course. The symptoms are mainly confined to constitutional disturbance, ecchymoses in the lower extremities, and the affection of the gums, the grave complications above mentioned being rarely met with. The average duration of the disease is, nevertheless, some weeks. Recovery is deferred in proportion as the hygienic surroundings are unfavorable, but even then the termination is almost sure to be favorable.

The prognosis in grave cases, occurring under unfavorable hygienic influences, and aggravated by the lack of proper food and attention, is far otherwise. Here death is not infrequent, sometimes as a result of progressive cachexia, sometimes because of pneumonia, pericarditis, cerebral hæmorrhage, or a similar intercurrent disease.

Anomalous or rudimentary cases of scorbutus may occur. They are most apt to be seen when the disease is epidemic or endemic. As a rule, the symptoms are mild. We find, for example, a scorbutic gingivitis and stomatitis without hæmorrhage, or, on the other hand, hæmorrhage into the skin and mucous membranes unattended by alteration in the gums. There have even been cases reported of simple scorbutic anæmia without any local symptoms.

[The experience of army surgeons during our late civil war deserves mention in this connection. Hammond, Woodward, and others state that many cases classed in the sick reports as "general debility" were cases of incipient or imperfectly developed scurvy, hæmorrhage from mucous membranes or into the skin being absent.]

Diagnosis.—The diagnosis of scorbutus is almost self-evident when the two chief symptoms of hæmorrhage and alteration in the gums are both present. If, however, one or the other of these symptoms is suppressed or imperfectly developed, it may be difficult to determine what disease we have before us, or to exclude ordinary stomatitis, rheumatic peliosis, and similar diseases. If we remember the statements made at the beginning of this chapter, and bear in mind that these various diseases are probably ætiologically related to one another, we shall be less disturbed by these uncertainties. It may be mentioned, in conclusion, that various septic disorders, and also acute ulcerative endocarditis, may occasion the appearance of numerous hæmorrhages, and thus simulate scurvy.

Treatment.—The essential requisites in the treatment of scurvy are proper hygiene and diet. Fresh air, suitable nourishment, and good nursing, if promptly supplied, are usually of themselves sufficient to induce recovery, while the physician possesses no remedies which compensate for their absence.

The belief that a main cause of scurvy lies in a deficiency of fresh vegetables has given rise to a practice, still in vogue, of prescribing a great abundance of fresh vegetables, such as lettuce, spinach, and sorrel, fruit, lemonade, and other drinks prepared from fruit syrups. There is no reason to deviate from a course to which experience has given its sanction, although we have repeatedly had opportunity to see that the administration of fresh vegetables is by no means essential to rapid recovery. Patients supplied with any other proper nourishment thrive equally well. Certain varieties of plants have attained a special reputation as "antiscorbutics," such as the spoonwort (*Cochlearia officinalis*), so frequently mentioned in accounts of early polar expeditions. None of these plants, however, possess the specific properties assigned to them. The administration of vegetable acids and the salts of potassium (bitartrate and nitrate of potassium), in a chemically pure form, has also been repeatedly tried, but it has not gained popularity.

The drugs most employed are the bitters and tonics. They have no specific value, but are perhaps as good remedies to prescribe as any. We may give a decoction of cinchona, to which may be added a small amount of dilute sulphuric acid and sirup, or some preparation of gentian or a similar bitter. It was once believed that the internal administration of the mineral acids exerted a specially favorable influence upon the hæmorrhagic diathesis; but this is very doubtful.

Certain symptoms may demand attention; in particular, the affection of the mouth and gums. It is of great importance to cleanse the mouth frequently with disinfectants and mild astringents, such as chlorate of potassium or sage tea. It is also advisable to paint the inflamed and spongy gums at short intervals with tincture of myrrh or tincture of rhatany. The absorption of the ecchymoses in the lower extremities will be promoted by cautious massage. Inunctions of linimentum chloroformi and the like give great relief from the pain caused by the extravasations into the deeper tissues. In severe cases stimulants are often demanded, such as camphor, ether, and alcohol. Such complications as appear may also demand special treatment.

Convalescence is promoted by continued attention to diet, bathing, and the administration of iron and quinine.

CHAPTER VII.

PURPURA. MORBUS MACULOSUS WERLHOFII. PELIOSIS.

As already stated in the preceding chapter, the various "hæmorrhagic diseases" are so intimately related to one another that it is quite impossible to make a rigid categorical division of them. The numerous names which have been introduced into the literature of this subject certainly contribute more to obscure than to elucidate the attendant phenomena.

From a clinical standpoint this fact is the all-important one—namely, that there are cases in which the foremost symptom is the spontaneous occurrence of hæmorrhage. There are cutaneous ecchymoses, and there may be at the same time hæmorrhages in the internal organs and into the mucous membranes. In the milder cases of this sort these hæmorrhages constitute almost the only symptom of disease; but they may be associated with considerable general disturbance, indicated by fever and weakness, or with certain local complications. The true cause of these diseases has not yet been discovered. There is seldom any evidence of an exciting cause, and the disorder may attack either the well-nourished or the poorly nourished, the old or the young, men or women. There is, however, an indisputable relationship between these diseases and certain others—namely, scurvy, erythema exsudativum, and perhaps acute rheumatism and endocarditis. This similarity indicates that the process is of an infectious, or toxic, character. Such an assumption promotes greatly a proper understanding of the phenomena under consideration. It is also said that subcutaneous injections of blood from patients with morbus maculosus may cause a like affection in rabbits (Petrone and others). In some few cases the weight of evidence would seem, however, to point to an antecedent impairment of nutrition in the walls of the blood-vessels. A good example of this is seen where cutaneous ecchymoses occur in old and marantic individuals (*peliosis senilis*). There is some doubt whether these exceptional cases belong with the others.

The mildest forms of the diseases under discussion are termed **purpura**. The hæmorrhages are seen mainly in the skin of the lower extremities, and are apt to take place into the follicles. There may also be ecchymoses upon the trunk and upper extremities, but the mucous membranes and the deeper tissues remain intact. A means of distinguishing purpura from scorbutus lies in the fact that in purpura there are no hæmorrhages into the muscles and no lesions of the gums, although it should be confessed that transitional forms between the two occur. The disorder is called *purpura simplex* if the cutaneous ecchymoses constitute the only symptom, or, at any rate, the only important one. These cases almost invariably terminate in recovery, and are over at the end of ten days or three weeks. Sometimes elevations of the skin are formed resembling wheals, and hæmorrhages take place here and there into them. This subvariety has been called by some *purpura urticans.* It forms a connecting link between purpura simplex and those cases of erythema exsudativum which are associated with hæmorrhage. Further particulars may be found in special works upon dermatology.

Quite often the hæmorrhages are attended by "dragging rheumatic pains": such cases are termed **purpura rheumatica** or **rheumatic peliosis** (Schönlein). There may also be constitutional disturbance, slight fever, anorexia, and indisposition to either bodily or mental exertion. There may sometimes be actual arthritis, with an inflammatory effusion into the joints. The knee and other joints of the lower extremities are most apt to suffer in this way. The gums are usually normal; nor is there, as a rule, hæmorrhage into the mucous membranes or the viscera. These cases may last but two or three weeks. Often, however, they are more tedious, being marked by the recurrence of the ecchymoses and articular pain. Most of them get well at last.

No sharp dividing line can be drawn between the forms of purpura thus far described and certain graver cases. These latter are most of them grouped under the name of *purpura hæmorrhagica*, or its preferable because more distinctive synonym—every purpura being hæmorrhagic—**morbus maculosus Werlhofii**. The cutaneous ecchymoses in this class of cases are usually extensive; and, furthermore, we have hæmorrhages into the mucous membranes of the nose, mouth, soft palate, stomach, and intestinal canal, as well into internal organs (the brain and kidneys), and also into the serous membranes. The constitutional disturbance is apt to be severe. The condition may be distinctly "typhoidal." Fever may be entirely absent, even in grave cases, although sometimes there is a considerable rise in temperature.

There are usually no local symptoms beyond those already mentioned. In typical cases the gums remain intact. Swelling of one or more joints has been repeatedly observed, as have also endocarditis and acute hæmorrhagic nephritis. If marked cerebral symptoms are developed, suggesting an apoplectic shock, we may surmise that a cerebral hæmorrhage has taken place. It should also be stated that marked gastro-intestinal disturbance may occur. Cases of this sort have been observed by Henoch in children. They may also occur in adults. In rare instances there may be intestinal ulceration, with perforation and consequent peritonitis. The spleen may undergo acute enlargement.

The prognosis in purpura hæmorrhagica should always be a guarded one; the patient is in danger both from the general depression and anæmia, and from certain special lesions. Even a severe case may, however, recover. The disease sometimes proves very tedious; it may occupy several months.

Treatment.—The general regimen to be prescribed is similar to that directed in scurvy. The physician must strive to support his patient's strength by means of proper nourishment. A great many internal remedies have been recommended, most of them on purely theoretic grounds. It is difficult to say whether they actually exert a favorable influence upon the course of the disease. The following drugs are chiefly employed: Ergotine, perchloride of iron, dilute sulphuric acid, and cinchona. If there be swelling of the joints or endocarditis, we should advise a trial of salicylic acid or antipyrine. Such special symptoms as demand attention should be treated according to general principles.

CHAPTER VIII.

HÆMOPHILIA.

Definition and Ætiology.—Hæmophilia is the term used to denote a peculiar constitutional anomaly, exhibited in a remarkable tendency to spontaneous and traumatic hæmorrhage. The condition is probably in every instance congenital, and is usually hereditary; the existence of families of "bleeders" has long been known. Generation after generation displays frequent cases of hæmophilia, both among the direct descendants and the lateral branches. Bleeders are very apt to have a numerous progeny. Not all of the children, however, fall victims to the disease. Grandidier has pointed out two facts which are of interest in this connection, as they might aid in deciding as to the marriageability of certain persons.

If a man belonging to a family of bleeders marries a healthy women, neither a bleeder herself nor inheriting a predisposition to hæmophilia, his children are almost certain to be healthy, even though the father himself be a bleeder. On the other hand, a woman belonging to a family of bleeders, even though she herself be healthy, will almost always have some children who are subject to hæmophilia. In other words, hereditary predisposition is transmitted much oftener through the female than through the male members of the family. Hæmophilia itself, is, on the contrary, much more frequent in the male sex than in the female; at least this is true of the pronounced cases. Hössli, who has lately published a very careful family tree of bleeders of Tenna (Canton Graubünden), comes to the following conclusions: "The inheritance of hæmophilia is often from the father, through the daughter, to the grandson, also from the mother, through the daughter, to the grandson, and most rarely directly from the father to the son." It is doubtful whether race and place of residence are of ætiological importance. So far as is known, hæmophilia appears to occur in all countries, although it is fortunately rare anywhere.

[A similar transmission through the females, who themselves usually escape, is seen in color-blindness and in pseudo-hypertrophic paralysis.]

The real causes of hæmophilia are entirely unknown to us. We can make one or two steps toward finding the source of the hæmorrhage, but we are unable to proceed further. It would seem that the bleeding must depend, in the first place, upon an abnormal delicacy of the walls of the vessels predisposing them to rupture, and, secondly, upon deficient coagulability of the blood. This latter abnormality is evident from the fact that in hæmophilia it is difficult to check even the most insignificant hæmorrhage. Thus far all attempts to discover any anatomical or chemical explanation of this imperfect coagulability have been vain. It has not been possible to detect any variation in the saline constituents of the blood, or in the amount of albuminoids, such as fibrinogen, that it contains, or in its morphological elements. And likewise no anatomical change in the vascular walls or the heart has yet been reported which throws light upon the character of the disease. Various authorities have laid stress upon the small diameter of the arteries and the thinness of the intima, but these conditions may occur independently of hæmophilia. Fatty degeneration of the intima is, to be sure, often found in connection with this disease, but it is doubtless rather a result of the coincident anæmia than the cause of the hæmophilia. The observations with regard to the heart are very contradictory; sometimes it is found to be very small, sometimes of normal size, and again actually hypertrophied.

The subjects of hæmophilia do not present any distinctive constitutional peculiarities. It has been stated that they are very apt to be blondes with a delicate white skin, and superficial and abnormally distended cutaneous veins; but the exceptions to this rule are not a few.

Clinical History.—Hæmophilia does not display equal malignity in all cases. If we have opportunity to obtain thorough information with regard to families of bleeders, we shall find that quite often rudimentary varieties occur side by side with typical and severe cases. There is, to be sure, a striking tendency to hæmorrhage even in them; but the hæmorrhage never assumes threatening proportions. By perseverance and industry it is possible to collect an almost unbroken series of cases, varying in degree from extreme mildness to extreme severity. The following sketch applies mainly to typical and severe cases.

That hæmophilia is a hereditary constitutional disease is shown by the fact that it sometimes appears in earliest infancy. Many cases of umbilical hæmorrhage in the newborn are referable to hæmophilia. Of course this does not apply to all cases. In Jewish children the disease may betray itself for the first time when the rite of circumcision is performed. In many cases the disease does not betray itself at so early a period; but this is no proof that the disease is not already developed, inasmuch as the young child is not much exposed to traumatism and other causes which naturally occasion hæmorrhage.

The most striking symptom in a fully developed case of hæmophilia is the occurrence of severe hæmorrhage as a result of the most insignificant causes. A slight blow produces a " black-and-blue spot " such as is ordinarily seen only after a very violent injury. The prick of a pin, a slight cut on the finger, or the extraction of a tooth, may give rise in hæmophilia to an obstinate and alarming hæmorrhage. Epistaxis may be caused by blowing the nose, and hæmorrhage from the gums by brushing the teeth, and so on. Whether there is ever a perfectly spontaneous hæmorrhage is uncertain. It is true that in severe cases hæmorrhages take place independently of any visible cause. This may be seen in the skin and mucous membranes (nose and gums); and in rare instances we may even have free hæmorrhage from the stomach, intestines, or urinary passages. Yet it may be doubted whether these occurrences are not the result of comparatively insignificant mechanical injuries which escape our notice. At any rate, we scarcely ever find hæmorrhage taking place into the parenchyma of the viscera, in places where injury from external sources is entirely out of the question. This fact constitutes an important point of distinction between hæmophilia and the acquired hæmorrhagic diathesis.

The second important symptom of hæmophilia has been already referred to: it is extremely difficult, and may even be impossible, to check by artificial means any free hæmorrhage which may occur. It is this which makes the disease so dangerous, and prevents most patients from reaching old age. It has frequently happened that an apparently trifling wound of the skin, or some insignificant operation, or a leech-bite, or in women childbirth, has started up a hæmorrhage, which eventually became fatal. In other cases the hæmorrhage is finally checked, but not until it has caused profound anæmia. Bleeders are apt to recover with remarkable rapidity from the effects of excessive hæmorrhage; yet continually repeated hæmorrhages may lead to a persistent and profound anæmia, attended by all the symptoms described in preceding chapters.

We see, therefore, that the general condition in hæmophilia varies with the severity of the individual case, and with the more or less fortuitous circumstances which develop its dormant characteristics. If no special accident occurs, the patient may maintain the appearance of perfect health for years. In the worst cases, however, such a state is very temporary, if it exists at all, because the hæmorrhages can be so easily excited. As a consequence, the skin almost always presents a greater or less number of ecchymoses, while hæmorrhages from the internal organs contribute from time to time to the general debility and anæmia. Certain complications may occur in hæmophilia, but they are little characteristic. There is a noticeable tendency to "rheumatic" inflammation of the muscles and swelling of the joints, wherein is seen a striking analogy to the "hæmorrhagic diseases." Often there is an actual effusion of blood into the joints. This may cause considerable functional disturbance of the joints, and eventuate in anchylosis. Various writers have also called attention to the comparative frequency of neuralgia, especially in the trigeminus.

Prognosis.—In only too many instances the victims of hæmophilia die in childhood; in other cases the patient attains an advanced age. A fact of great practical importance is that often, although not invariably, hæmophilia grows gradually milder with advancing years. If, therefore, the patient have survived the period of adolescence, we may believe that his prospects are gradually improving. The prognosis of hæmophilia is obvious. The amount of danger at any given time depends upon the severity of the hæmorrhage and the consequent anæmia. The comparative severity of the case must be judged from its previous history; as has just been said, the prognosis grows more favorable as the patient grows older.

Treatment.—Prophylaxis assumes a very important place in the treatment of hæmophilia. First, children who inherit a tendency to the disease, or who have given evidence of its existence, should be treated with a view to improve their general constitutional condition, so as to check the development of the disease as far as possible. The means to this end need not be described at length. They comprise good nourishment, fresh air, cautious endeavors to harden the system, baths, and tonics. Secondly, when hæmophilia already exists, the patient should be guarded as much as possible from any mechanical injury, such as might excite hæmorrhage. Thus caution is demanded in performing vaccination and other apparently trifling operations.

As regards direct treatment of the disease, no effectual remedy is known. The general tonic treatment already referred to should not be neglected; but the administration of ergotine, acetate of lead, and similar drugs is indicated, if at all, only when hæmorrhage is actually taking place, and even then it is very apt to fail. The only way to stop the hæmorrhage is by surgical methods, and these need not be described here. They do not differ essentially from those employed when hæmorrhage occurs independently of hæmophilia. If mechanical efforts to check the bleeding fail, we can expect nothing from the remedies above mentioned, nor from sulphate of sodium and the other laxatives which have been recommended. For the symptomatic treatment of the anæmia and of its results, we may refer to the first chapter of this section.

CHAPTER IX.

DIABETES MELLITUS.

Definition and Ætiology.—Under normal circumstances the blood always contains a slight amount of sugar; but this ingredient does not usually pass over in appreciable quantities into the urinary excretion. If, however, the amount of sugar in the blood exceeds certain limits—that is, if there exists an abnormal "glykæmia"—then sugar is excreted in the urine, and we have glycosuria. This is seen as a more or less temporary phenomenon under the most varied conditions. The amount of sugar in the urine is usually comparatively slight, and it soon disappears again. Its presence does not imply any persistent abnormal condition. This phenomenon has been termed glycosuria or melituria, in contrast with the peculiar disease which has for its chief symptom a persistence of sugar in the urine, and has therefore received the name of diabetes mellitus.

The causes of glycosuria need not be discussed here at any length. We will briefly state that sugar may appear temporarily in the urine as a result of poisoning from various substances, chief among which are carbonic-acid gas, morphine, hydrocyanic acid, mercury, nitrite of amyl, and curare. Von Mehring has lately discovered in phloridzine, a glucoside in the bark of the roots of apple and cherry trees a substance which causes a very large amount of sugar in the urine if given to dogs, rabbits, etc. A marked glycosuria may also be produced in man by giving phloroglucine, or by other disturbances of the general condition. Temporary glycosuria has also been seen in connection with the acute infectious diseases—for example, malignant pustule, cholera, typhus or typhoid fever, scarlet fever, diphtheria, and malarial poisoning. A far more frequent cause is disturbance of the nervous system. Thus, glycosuria may result from severe concussion of the brain, fracture of the skull, cerebral hæmorrhage, cerebro-spinal meningitis, and after epileptic fits. It is especially apt to occur when there is disease of the medulla; and we need hardly point out how close is the connection between this clinical fact and the famous discovery of Claude Bernard, who found by experiment that, when certain injuries are inflicted upon the floor of the fourth ventricle, glycosuria inevitably follows. It has been thought that diseases of the stomach and liver may occasion glycosuria; but this is doubtful. With regard to the theory of diabetes (*vide infra*), it is an interesting fact that extensive disease of the liver—as seen in phosphorus poisoning or cirrhosis—occasions no glycosuria, even when the patient's diet contains large amounts of sugar (Frerichs); but atrophy of the pancreas seems to have a definite relation to diabetes (*vide infra*). As Minkowski and Von Mehring have found, we can produce a severe diabetes in dogs by extirpation of the pancreas. Ligature of the duct of the gland does not produce this condition. There must therefore be some special function of the pancreas not yet known, and after this function ceases to be performed the consumption of sugar in the animal economy ceases to be complete.

Diabetes mellitus is a disease in which a considerable amount of sugar is constantly present in the blood, and consequently in the urine. The immediate cause and the true nature of this strange disease are entirely unknown. It is therefore difficult to determine whether all cases of "diabetes mellitus" are essentially identical. In most of the typical cases this question may, indeed, be answered affirmatively with little hesitation; but other cases, particularly many of what are called the "milder varieties" of diabetes, afford more room for doubt. We must not forget that diabetes mellitus is at present known to us only through its symptoms, and that the individual cases do not exhibit any uniformity either as to causation or as to anatomical changes.

As has been said, we know practically nothing of the true causes of diabetes. All that the physician can do in any particular case is to search for certain exciting or predisposing causes, the significance of which has been learned from clinical experience. It must, however, be borne in mind that in many cases of diabetes, and some of them most severe, no cause whatever can be made out; the disease seems to have developed of itself in persons who were previously perfectly well. We append a list of such exciting influences as seem to be most frequent and important. First, heredity: diabetes has been repeatedly observed to occur in several generations of the same family, or in several brothers and sisters. It is noteworthy that the disease may also occur in families where there is a hereditary predisposition to nervous diseases. Second, improper mode of life: by this is meant chiefly unsuitable diet, especially the persistent overindulgence in starchy foods and sugar; sedentary habits are also considered harmful, especially if associated with overeating. This is said to be the reason why diabetes is more frequent among the wealthy classes, and why it is quite common in corpulent persons. Third, taking cold and getting wet seem, in occasional rare instances, to determine the appearance of diabetes. Fourth, emotional disturbances, excessive mental exertion, anxiety, and passion, are sometimes thought to occasion the disease. Fifth, it is very remarkable that sometimes the same factors which we have already seen to be possible causes of temporary melituria, may also occasion a chronic diabetes mellitus; thus, cases of diabetes have been known to follow injuries to the head, and such acute infectious diseases as typhus, typhoid, or scarlet fever, cholera, and malarial poisoning. Sixth, certain chronic constitutional and infectious diseases, particularly gout and syphilis, may perhaps promote the development of diabetes. The disease also appears in connection with certain organic diseases; this list includes cerebral diseases, such as hæmorrhage, tumor, or sclerosis, particularly when in the region of the fourth ventricle; other nervous disorders, such as organic disease of the peripheral nerves, and functional diseases; and, in rare instances, disease of the pancreas, such as suppuration and cancer. It is obvious that such cases of diabetes should be regarded as "accidental," in distinction from the true idiopathic disease.

Diabetes occurs everywhere, but certain countries and districts seem to be particularly liable to it—for example, India, Ceylon, and Italy. In Germany, Würtemberg and Thüringen are said to present the largest relative number of cases. Jews are very liable to the disease. Most cases occur in patients between thirty-five and fifty years of age. Next in liability to the disease come younger individuals, under thirty-five and over twenty years old. After the fiftieth year diabetes is not very exceptional; but, in the other direction, children under ten are very seldom attacked by it, although they are not absolutely exempt. With regard to sex, males are much more often attacked than females.

Clinical History.—With few exceptions, the symptoms of diabetes mellitus come on slowly and gradually. Sometimes the symptoms are merely general and indefinite, such as languor, emaciation, weakness, and deficient endurance. Sometimes we have mild nervous disturbances, including headache, mental depression, wakefulness, and neuralgia, and in still other cases gastro-intestinal symptoms, including nausea, eructations, and irregularity of the bowels. At last the patient's attention is called to the altered character of the urine, and particularly to its increased amount. He also notices that he is very thirsty, and that, in spite of his enormous appetite, he is constantly growing weaker. Sometimes quite different symptoms first arouse suspicion of the existence of diabetes; these will be mentioned later. In order to make a diagnosis of diabetes mellitus, a knowledge of the abnormal character of the urine is indispensable. We shall therefore proceed to describe the changes produced in the urinary excretion as a result of diabetes mellitus.

1. CHARACTER OF THE URINE. DEMONSTRATION OF SUGAR.—Usually the first point that attracts attention is the increased amount of urine. There are often a hundred to a hundred and fifty ounces (three to five quarts, 3,000–5,000 c. c.) excreted in twenty-four hours, and sometimes there may even be as much as ten or twelve quarts (8,000–12,000 c. c.). Under suitable treatment and with proper diet the amount may, of course, be much smaller. In some cases the polyuria will

almost cease from time to time ("*diabetes decipiens*"). Often the amount of urine undergoes diminution, when some intercurrent disease appears, or when death is imminent.

In color the urine is light yellow, corresponding to its amount. It often has something of a greenish hue, but a small quantity of it may seem almost as colorless as water. Ordinarily the urine is clear and without sediment; but after it has stood for some time it may become cloudy, usually as a result of the abundant development of fermentation spores.

The odor may be somewhat aromatic, suggesting acetone (*vide infra*). Its taste, as determined by earlier observers, may be distinctly sweetish. The reaction is acid, and the acidity of the urine may increase on standing, because of the alcoholic and lactic-acid fermentation processes which the sugar undergoes.

The specific gravity is almost invariably greatly increased, as a result of the large amount of sugar. If a pale urine is found to have a specific gravity of more than 1025, we may feel almost certain that it contains sugar. Specimens often have a specific gravity of 1030–1045, and even higher. In exceptional instances the specific gravity may fall below 1020; this may occur, for instance, where the patient is very much debilitated.

The diagnosis requires that sugar be detected in the urine. The sugar found in both the blood and the urine of diabetic patients is grape-sugar (glucose, dextrose). The amount of sugar secreted in twenty-four hours often reaches half a pound to a pound (grm. 200–500). Of course the amount varies greatly according to the diet, mode of life, and treatment of the patient. The greatest amount ever known to be produced in twenty-four hours was more than two and one fifth pounds (grm. 1,000). The percentage of sugar in the urine varies between 0·5–1 per cent. at the lowest extreme, and 8–10 per cent. as the maximum; usually it is about 2–4 per cent. It is noteworthy that in the last weeks, or just before death, the sugar in the urine may become greatly diminished in amount, or may absolutely disappear.

The most important tests for sugar in the urine are, first, Trommer's test: To urine in a test-tube sufficient potassic or sodic hydrate is added to make the reaction strongly alkaline; then a solution of sulphate of copper (about one part of the salt to ten of water) is added, drop by drop. If the urine contain sugar, the hydrated cupric oxide, which is at first formed in large amounts, is dissolved, and usually the fluid assumes a beautiful deep-blue color. We ought, properly, to go on adding the sulphate of copper until the hydrated cupric oxide ceases to be dissolved. The urine is then heated, whereupon the cupric oxide is reduced and a yellow, or reddish-yellow, precipitate of cuprous oxide, or hydrated cuprous oxide, is formed. The application of heat should not be continued long after the precipitation begins to take place, lest the test be obscured; the reduction will go on even without heat. If the urine contains more than 0·5–1 per cent., this test is perfectly reliable. If the reaction be a doubtful one—that is, if there be no precipitate of cuprous oxide, although the urine becomes yellow—we should be cautious in making a diagnosis, as the urine may contain substances other than sugar capable of reducing the copper, such as uric acid, kreatinine, mucine, etc. Second, the bismuth test (Böttger's): Sodic hydrate or sodic carbonate is added to the urine, and then a small pinch of subnitrate of bismuth. Upon boiling, the urine, if it contains sugar, quickly assumes a perfectly black color, the oxide being reduced to the metallic state. Third, the potassium test (Moore's): Potassic hydrate is added to the urine in the test-tube and the uppermost layer cautiously heated; if it contains sugar, the urine quickly assumes a deep-brown color, as a result of the action of the potassium on the sugar; and this upper dark-colored layer contrasts strongly with the clear urine below.

[Fehling's test is justly a favorite in this country, and has the advantage of being applicable to the quantitative as well as to the qualitative analysis. The difficulty of its not keeping well can be met by having separate bottles for the copper and tartrate solutions, and making the mixture at the time of using the test. The Fehling's-test pellets put up by chemists are convenient for the qualitative analysis, but, on the whole, they are inferior to the solution.]

If the above-described tests leave us still in doubt, there can be only a small amount of sugar, if any, present. We may, however, attain certainty by employing the fermentation test (which causes a decomposition of the sugar into alcohol and carbonic dioxide), or circumpolarization (deflection of the plane of polarization to the right by the grape-sugar). Further particulars in regard to these and other tests, and also in regard to the quantitative estimation of sugar, may be found in works on medical chemistry.

Diabetic urine sometimes contains other varieties of sugar in small amounts—namely, levulose, which deflects the plane of polarization toward the left, and inosite; these are, however, of no practical importance.

The amount of urea is usually somewhat increased (*vide infra*). Uric acid, on the contrary, is excreted in small amounts. The amount of kreatine is normal or even increased (Senator). The amount of phosphoric acid and sulphuric acid usually corresponds to the amount of urea, or, in other words, to the decomposition of albuminoids. Occasionally, according to Teissier, the amount of phosphates is surprisingly great, and may either correspond with the amount of sugar simultaneously excreted, or replace the sugar in the urine. This subject has not yet been fully investigated. The amount of sodic chloride excreted depends, as in health, merely upon the amount ingested.

Hallervorden has discovered an important fact in regard to the excretion of ammonia. In many cases of diabetes, although not in all, it is much increased: forty-five to ninety grains (grm. 3–6), or even more, may be excreted in twenty-four hours. Despite this, diabetic urine has an acid reaction; and, as Stadelmann has shown, the basic elements are considerably out of proportion to the acids known to us. It is, therefore, evident that diabetic urine, since it contains a large amount of ammonium and yet has an acid reaction, must have among its constituents some unusual acid. Stadelmann was at first inclined to believe that this was crotonic acid. Minkowsky and Külz have, however, shown, by their more recent investigations, that the acid in question is really oxybutyric acid, or, more accurately, beta-oxybutyric acid. This acid is readily decomposed into crotonic acid, which explains Stadelmann's error. Another interesting fact is that, upon oxidation oxybutyric acid changes to acet-acetic acid, a substance easily decomposed into carbonic-dioxide gas and acetone. This suggests the possibility that oxybutyric acid may give rise within the system to acetone, a substance which has occupied an important place in the literature of diabetes.

Acetone was first discovered by Petters in diabetic urine. This was regarded as an important discovery, because it was believed that the accumulation of this substance in the blood produced those grave nervous disturbances (*vide infra*, diabetic coma) sometimes observed in diabetes. More recent developments have rendered this view extremely improbable, but it is an established fact that acetone is quite often present in the urine of diabetic patients.* Whether it is a primary or secondary product remains uncertain. It was formerly believed that acetone was formed from ethyldiacetic acid (acet-acetic ether), but of late the tendency is rather to regard acet-acetic acid as the source of acetone (Deichmüller and Tollens, Jaksch). Acetone is probably the cause of the reaction to which Gerhardt has called attention—namely, the development of a Burgundy-red color in the urine upon the addition of ferric chloride. This is quite often to be observed in the urine of diabetic patients. In speaking of diabetic coma we shall have occasion to revert to this ferric-chloride reaction.

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* It should be added that acetone has been found by Kaulich and Jaksch to occur frequently in the urine during many other diseases, both febrile and nonfebrile, and even in normal urine.

Albumen may be found in diabetic urine, as will be seen below under renal complications; small amounts, to be detected only after careful examination, are common.

2. TISSUE METAMORPHOSIS IN DIABETES. SOURCES OF THE SUGAR AND VARIATIONS IN ITS AMOUNT OCCASIONED BY EXTERNAL INFLUENCES.—Inasmuch as the presence of sugar in the urine is the most prominent symptom in diabetes, the question of its origin is all-important. One fact is indubitable—namely, that the secretion of sugar depends in large part upon the amount of potential sugar ingested—that is, upon the proportion of starches in the food. The amount of sugar excreted with the urine increases and diminishes with the amount of starchy food eaten. If a diabetic patient abstains totally for any length of time from such articles of food as contain starch, sugar will, in many instances, entirely disappear from the urine. In other words, the system of a diabetic patient is almost, if not quite, incapable of oxidizing sugar into carbonic-dioxide gas and water. Voit and Pettenkofer have made an experiment the result of which confirms this statement. By means of the great Munich respiration apparatus, they have demonstrated that in diabetes the amount of oxygen absorbed from the air, and of carbonic-dioxide gas and water given off from the body, is less than in health, the diet being precisely the same in both cases. Previously to this many investigators had shown that there was a diminution in the amount of the "insensible excreta."

The oxidation of sugar is not absolutely *nil* in diabetes. Küiz has proved by numerous experiments that not all the starch is excreted in the form of sugar, and he has also found that many varieties of sugar, such as mannite, fruit sugar, and inosite, are decomposed even in diabetes, so that their ingestion does not lead to an increase of sugar in the urine.

The disturbances in metamorphosis affect other substances than the carbohydrates. While they escape oxidation, on the other hand, the destruction of albuminoids is increased. We have already stated that diabetic urine contains a large amount of urea. Gäthgens and others have shown, by means of carefully conducted investigations, that this increase in the amount of urea is not merely relative, but absolute. The system of the patient destroys a larger amount of albumen than does the system of a healthy man, the ingesta in both cases being alike. Whether this is true in all cases of diabetes is quite doubtful, but the fact is established with regard to severe cases. It is also certain that in severe cases a part of the albumen is transformed into sugar, escapes oxidation, and is excreted in the urine. The proof of this is that in some cases of diabetes sugar continues to be present in the urine, although the diet is exclusively nitrogenous. Seegen accordingly divides all cases of diabetes into two varieties: a milder form, where sugar ceases to be excreted if starchy food be excluded from the diet, and a severer form, where sugar persists upon an exclusively meat diet.

Muscular exertion is one of the external influences which modify the excretion of sugar in diabetes. According to our present views, muscular activity wastes mainly non-nitrogenous substances, and, accordingly, we find in diabetes that an increase of muscular exertion, other things being equal, diminishes the amount of sugar excreted.

Emotional excitement is said to increase the amount of sugar excreted.

Intercurrent acute febrile diseases may cause a great diminution in the amount of sugar, but sometimes there is no essential change. Probably the altered diet of the patient plays an important part in this connection, although doubtless the special modifications of tissue-metamorphosis occasioned by the high temperature or by the disease itself also exert some influence.

3. CONSTITUTIONAL SYMPTOMS IN DIABETES MELLITUS.—In many of the milder cases there is for a long time little apparent disturbance of the general health. The patient is well nourished, and suffers little discomfort, except the inconvenience occasioned by the polyuria and the polydipsia. In severer cases the system is deeply affected by the drain upon it. The patient becomes emaciated, weak, and easily exhausted, and at length there may be profound marasmus. Mentally, the patient is apt to be depressed and irritable. The intellectual powers are not impaired, but there is indisposition to mental effort. The temperature is normal or subnormal. Fever invariably indicates some complication.

4. SYMPTOMS REFERABLE TO THE DIGESTIVE ORGANS.—We have already mentioned the excessive thirst experienced in diabetes. This may be a source of great discomfort, obliging the patient to drink at short intervals, even through the night. The interdependence of polyuria and polydipsia is not yet fully understood. The most natural view seems to be that the increased excretion of water by the kidneys is the primary factor, and the increased thirst secondary thereto. One cause of the polyuria is the excretion of sugar, in order to dissolve which a large amount of water is necessary, but certain nervous factors would also seem to be implicated. That they exert some influence is rendered probable by the fact that the amount of urine does not always correspond to the amount of sugar excreted. A very large amount of urine may be excreted containing little or no sugar; and, on the other hand, there are genuine cases of diabetes mellitus where the amount of urine is normal, and the patient feels no unusual thirst (*diabetes decipiens*). It has also been suggested that the sugar may irritate the nerves of the mouth and throat, and thus cause thirst. According to this idea, the polyuria would be merely the result of the excessive ingestion of liquids. The abnormally great appetite in diabetes seems in most cases to be due to defective assimilation of the food. Many patients are never able to eat enough. They have a longing, in many cases, for carbohydrates. Occasionally the hunger becomes ravenous, and is associated with headache and a general sense of weakness, all these symptoms being alleviated when food is taken. This rule has rare exceptions in which the appetite is not unusually great, even though the case be severe.

The tongue is frequently dry; it is broad and thick, with an irregular and fissured surface, sometimes coated and sometimes red. The gums may be spongy, and may exhibit a tendency to bleed. The teeth frequently decay rapidly. The saliva is invariably found to give an acid reaction. This is true of the isolated secretion of the parotic gland also, and is said to be due to the presence of lactic acid. It is only in exceptional instances that sugar can be demonstrated in the saliva. Thrush quite often appears on the soft palate.

There are no marked gastric symptoms. There is usually constipation, but sometimes there is a severe, though temporary, diarrhœa. The liver and spleen are seldom much affected; the liver rarely is somewhat enlarged. Jaundice is frequently observed, but it is always referable to some complication. As a rule, the secretion of the bile goes on as in health.

5. SYMPTOMS REFERABLE TO THE RESPIRATORY ORGANS.—In many cases the organs of respiration are unimpaired for a long while. It may be mentioned that often patients have a decidedly fruity odor to the breath (acetone odor). In the later stages of the disease pulmonary complications are very frequent. Almost one half of all diabetic patients perish from secondary disease of the lungs. Most frequently there is pulmonary tuberculosis; its course, symptoms, the presence of tubercle bacilli, and all other details are the same as in ordinary cases of tuberculosis. Next in point of frequency is pulmonary gangrene. Sometimes there is

a diffuse gangrenous process, and sometimes there are isolated foci of necrosis, which become liquid, and have an acid reaction, but they often have comparatively little odor. The expectoration in these cases may be odorless. Croupous pneumonia may also occur. It often terminates unfavorably, and may, as we have ourselves observed, result in gangrene.

6. SYMPTOMS REFERABLE TO THE CIRCULATORY SYSTEM.—In many instances the circulatory apparatus presents no special lesions. The pulse is either of a normal rate or a trifle slow. It is usually soft, although exceptionally it may exhibit increased tension. There is often distinct evidence of cardiac weakness (Schmitz); the pulse is small, intermittent, sometimes very slow (50 or even 40 beats per minute), and sometimes accelerated (100 to 120 beats per minute). There are shortness of breath, faintness, nausea, and the like. Sometimes sudden and profound cardiac disturbance occurs, and may occasion speedy death (*vide infra*, coma). Not very rarely diabetes is combined with general arterio-sclerosis. This is particularly apt to be the case in patients who have been subject to gout.

7. GENITO-URINARY SYMPTOMS.—Despite the great demands made upon them, the kidneys usually maintain a normal condition. As we shall see when we come to the pathological anatomy of diabetes, the kidneys are often very large. Sometimes a chronic nephritis is developed as a complication, usually in the later stages of the disease. The urine contains albumen, and there are œdema and other symptoms of renal disorder. It was formerly a common opinion that the nephritis results from the irritation of the sugar excreted by the kidneys, but in such cases other irritants are probably to be considered, especially as in most cases of nephritis complicating diabetes there are usually present at the same time other disorders to which the nephritis may be referable, such as pulmonary consumption or cardiac disease. We have seen well-marked suppurative pyelo-nephritis as a complication of diabetes. If the amount of albumen in the urine becomes considerable, the excretion of sugar usually undergoes marked diminution. Many physicians therefore consider the transition of diabetes into chronic renal disease (contracted kidney) as a comparatively favorable symptom.

Saccharine urine is apt, as it decomposes, to cause irritation of the skin. This is the explanation of the troublesome pruritus pudendi, which is especially marked in women. It may, indeed, be this symptom which first directs attention to the disease. Sometimes the external genitals are attacked by eczema or furunculosis. Men often suffer from balanitis, with inflammatory phimosis, or paraphimosis. A frequent and important symptom in men is impotence. This sometimes occurs very early in the disease, but it may afterward undergo improvement. The origin of it is not determined. Some authorities state that diabetes is apt to occasion atrophy of the testicles.

8. DISTURBANCES OF THE ORGANS OF SPECIAL SENSE.—An important and not infrequent result of diabetes is cataract. This may occasion almost total blindness. The cause of cataract in diabetes is not known. It was formerly supposed that the sugar in the blood absorbed water from the crystalline lens, and thus occasioned its opacity; but this has not been confirmed. Diabetic patients are also subject to disturbances of accommodation. Retinitis, atrophy of the optic nerve, and purulent choroiditis may also occur, but they are very rare, and perhaps are merely chance complications.

None of the other special senses are peculiarly affected in diabetes.

9. CUTANEOUS AFFECTIONS.—In most cases the skin is remarkably dry and rough. There may, however, be abundant perspiration. Several authorities claim to have found sugar in the perspiration, but this statement has not been confirmed by later investigators. Sometimes there is a troublesome pruritus. We often see a rapid loss of hair and a shedding of the nails. In many cases there is a great

tendency to furunculosis. This may be the first symptom to suggest the existence of diabetes. In the later stages there are sometimes extensive carbuncles, and multiple phlegmonous abscesses in the subcutaneous cellular tissue, which may prove fatal. Once we saw an eruption resembling pemphigus appear a short time previously to death. Gangrenous processes have been repeatedly observed, and in particular necrosis of one or more toes, which may take the form of perforating ulcer (*mal perforant*), or rarely of an entire extremity. This gangrene often seems to be due to arterial sclerosis; in other cases its cause is obscure.

Œdema of the subcutaneous cellular tissue may occur independently of nephritis. It is then probably occasioned by the cardiac weakness.

[Another skin affection which deserves mention is eczema. In the neighborhood of the genitals it is more common in women than in men, and is attributable to the irritating properties of sugar contained in the urine acting on a skin the nutrition of which is impaired owing to the morbid condition of the blood. Sometimes the eczema involves other portions of the integument, is generally acute, and has an angry appearance.]

10. Symptoms referable to the Nervous System.—It has already been mentioned that there is frequently in diabetes a moderate disturbance of the whole nervous system, as indicated by headache, physical and mental hebetude, and depression of spirits. Still more characteristic is neuralgia. This is most often located in the sciatic nerve, and an obstinate bilateral sciatica may be one of the first symptoms of diabetes. Occipital or trigeminal neuralgia or hemicrania, and, on the other hand, anæsthesia, have also been repeatedly observed. We once saw peripheral paralysis of the peroneus come on without any discoverable cause. Bouchard has called attention to the frequent absence of the patellar tendon reflex in diabetes. To what this is due, or what relation it bears to the primary disease, is not known. Possibly it is caused by degeneration of the peripheral nerves from toxic (?) influences.

The most important nervous symptom of all is a peculiar disturbance which occurs in a considerable proportion of all cases with more or less suddenness, and usually terminates in a surprisingly speedy death. This strange phenomenon was first thoroughly investigated by Kussmaul, although known long before. It is termed diabetic coma. The condition sometimes develops without any evident cause. In other instances it is apparently brought on by violent muscular exertion, mental excitement, or some trifling illness, such as gastric catarrh, bronchitis, or sore throat.

Frequently certain mild prodromata herald its onset. There may be nausea, headache, a sense of thoracic oppression, and general uneasiness. Soon the condition becomes aggravated. The patient is seized with a feeling of great anxiety, and becomes delirious, sometimes jumping out of bed and growing uncontrollable. Gradually, however, the excitement gives place to an ever-increasing drowsiness, usually terminating in the most profound coma. One of the most frequent and striking symptoms attending this condition is the peculiar alteration in respiration. The breathing becomes remarkably deep and noisy. Its rate may remain nearly normal; or it may be considerably increased, so as to justify the term "diabetic dyspnœa." The patient is sometimes cyanotic. The pulse is usually very rapid and small. The temperature gradually sinks, and has in repeated instances fallen to 86° (30° C.), or even lower. In most instances, also, the breath has a very noticeable odor, resembling fruit or chloroform, which may be perceived on entering the room. Even the urine may have this same odor; and it almost invariably becomes dark red on the addition of ferric chloride (*vide supra*).

Diabetic coma does not pursue the same course in all cases; sometimes the patient lingers on for several days before death, while in other instances the change is extremely rapid and death speedy. The early stage of excitement may be wanting. The patient becomes somnolent and then comatose, and never regains consciousness. Temporary improvement, and even complete cessation of the threatening symptoms, are not impossible, but they are very exceptional.

We have no definite information as to the cause of diabetic coma. Of course the cases where an autopsy discloses some marked organic lesion, such as cerebral hæmorrhage, capable of producing the nervous symptoms, are not true diabetic coma. Nor do the cases of sudden death reported by Frerichs deserve to be classed as diabetic coma, where death occurred with the symptoms of acute cardiac failure—namely, collapse, coolness of the extremities, small and rapid pulse, and unconsciousness. Besides, these patients never have the acetone odor, the exaggerated respiration, nor the ferric-chloride reaction, and usually the myocardium is found to be in an advanced state of degeneration.

In genuine diabetic coma, on the other hand, everything seems to indicate that the system has been poisoned by some noxious product of the abnormal processes of metamorphosis. Great effort has been made to discover what this product is, but in vain. Kussmaul regarded acetone as the injurious substance, and therefore called diabetic coma "acetonæmia." Other investigators believe that acet-acetic acid (Jaksch) is the cause of the phenomenon in question, or at least regard the coma as a result of poisoning from some of the unusual acids present in diabetes (Stadelmann, *vide supra*). No one view has obtained general acceptance, nor has it been possible to produce diabetic coma in animals by the employment of acetone, acet-acetic acid, crotonic acid, or similar substances (Brieger and others). It is, nevertheless, extremely probable that these substances do have some close connection with "diabetic intoxication" (Frerichs).

Pathological Anatomy and Histochemistry of Diabetes Mellitus.—The mysterious phenomena of diabetes mellitus present a problem, the solution of which has been most industriously sought post mortem; but no satisfactory result has been reached, even in this way.

If we exclude the organic diseases, such as pulmonary tuberculosis and nephritis, which are merely complications, the pathological changes in diabetes are trifling. Bernard's discovery, that an injury inflicted in a certain spot on the floor of the fourth ventricle produces glycosuria in animals, has directed the attention of investigators to the condition of the nervous system in this disease. In some instances, tumors, sclerosis, or similar troubles have been found in the medulla and cerebellum; but in these cases the diabetes was evidently not idiopathic (*vide supra*). In idiopathic cases the central nervous system presents no striking macroscopic changes. By means of the microscope, Frerichs has found lesions of the medulla oblongata in frequent instances. The minute blood-vessels are widely dilated; there are small capillary hæmorrhages, some of a more recent and others of a more remote date; and occasionally there are microscopic foci of myelitis. The nervous elements proper, the nerve-fibers and ganglion-cells, betray no alteration. The significance of these changes must be determined by further investigation.

The stomach and intestines present no constant alterations of importance.

The liver has naturally been the object of repeated and careful examinations, because of its well-known part in the manufacture of glycogen. Yet this organ seldom presents any special abnormality. It is usually of the natural size, and may contain either considerable or very little blood. The amount of glycogen in the hepatic cells can be demonstrated with iodine by a micro-chemical reaction. It appears that, other things being equal, there is less glycogen present than normal. It is usually found only in the cells situated upon the periphery of the lobules, and in small quantities. In an extremely advanced case of diabetes Ehrlich obtained, by means of a hollow needle, small amounts of the hepatic parenchyma for examination during life, and found that glycogen was almost completely absent. In other cases the liver has been examined as early as possible after death, and presented no trace of glycogen, but it sometimes has been found.

The spleen is usually of normal size. Occasionally it is atrophied, or, on the other hand, slightly enlarged. No other changes in it have been observed.

Many cases have presented a striking atrophy of the pancreas (Bouchardat), a fact which has acquired great significance from Minkowsky's discovery, already mentioned, that glycosuria may come on after extirpation of the pancreas. It is uncertain whether the cœliac plexus may also sometimes take part in the atrophy of the pancreas. We must also state, however, that atrophy of the pancreas is not a constant lesion in diabetes, so that it is not proper to regard the disease of the pancreas as the special cause of diabetes in all cases.

The kidneys are often enlarged, from functional hypertrophy. Ehrlich discovered in them a glycogenic degeneration * of the loops of Henle. This change is a constant one. The epithelial cells in the loops are enlarged, and the protoplasm in these cells, although apparently homogeneous, is found, by the addition of a solution of iodine in mucilage, to contain glycogen, in flakes and clumps of varying size. How important this glycogenic degeneration of the kidneys may be has not yet been determined. Perhaps the glycogen represents sugar which has been absorbed by the cells. That chronic nephritis may complicate diabetes has already been mentioned.

No thorough investigation of the chemical composition of the blood in diabetes has yet been made. One important and constant characteristic is the gradually increased proportion of sugar in the blood. There is usually somewhere between 0·2 and 0·45 per cent. of sugar, while in health the blood rarely contains over 0·1 per cent. The lymph, and such serous effusions as are found, contain sugar, but the saliva, perspiration, bile, gastric juice, and other secretions rarely furnish evidence of its existence.

Varieties, Course, and Prognosis of the Disease.—The study of a large number of cases of diabetes will show great variations in the course and duration of the disease. As already stated, we may in practice distinguish between mild and severe forms of diabetes. In the mild cases, sugar ceases to be excreted if the patient be put upon a diet containing no carbohydrates. Sometimes it is even possible for the patient to eat a small amount of starchy food without occasioning glycosuria, particularly if he take enough exercise (*vide supra*). In the severe form, sugar persists in the urine even upon a purely meat diet; and upon the ingestion of carbohydrates there will be, at the end of half an hour or so, a large increase of sugar. Most of the other symptoms are alike in both forms of the disease, varying only in degree. The moderate form may finally assume a severe character; and sometimes, although the secretion of sugar remains permanently small, fatal complications, such as tuberculosis of the lungs, finally develop.

The general course of diabetes varies in different cases, exclusive of the differences already referred to. In a few instances the disease occupies only a few weeks, and may almost be termed "acute diabetes." Other cases last one or two years, and still others ten or twenty years. The patient's condition may vary from time to time. We have repeatedly seen the sugar disappear temporarily from the urine, and the patient apparently completely recover; but sooner or later the disease breaks out again. These cases, which are commoner in old people, have been called "intermittent diabetes." The relapse is often brought about by emotional excitement or some grave error in diet. Again, the disease may undergo apparent arrest and the patient enjoy comparative comfort for years. As a general rule, it may be said that older patients are more apt to have the mild form of diabetes, while in young adults and children the disease is wont to pursue a more rapid and unfavorable course.

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* Ebstein and Armanni noticed and described this condition; but they regarded it as a necrosis of the epithelium.

Different cases also differ in the relative severity of particular symptoms. Thus, the clinical picture presented by diabetes may be modified by the general constitution of the patient—for instance, his corpulence or emaciation; by such complications as diseases of the lungs, kidneys, or brain, or syphilis and gout; and by many other conditions. In practice, especially in practice among the better classes, it is very important to have a knowledge of the milder forms of diabetes, in which sugar is present in the urine only at times, and not in very large amount. The more we are accustomed to examine every urine for sugar the oftener we find such cases, as to the pathological significance of which our knowledge is still very slight. The glycosuria of the corpulent (Seeger) is worthy of special consideration. It is not uncommon in patients who have formerly suffered from true gout. The tendency to the formation of furuncles and cataract is noteworthy. We must also mention the cases of diabetes in neurasthenics—that is, cases where there is sugar in the urine, and where the chief complaint is of general nervous symptoms, pressure in the head, anxiety, a dull feeling, incapacity for mental work, neuralgic pains, etc. In all such cases glycosuria has not the serious significance that it has in genuine and severe diabetes.

We would again call attention to the fact that diabetes may exist without polyuria or abnormal thirst, and so be overlooked. It is a very interesting fact that diabetes mellitus occasionally undergoes gradual transformation into diabetes insipidus (see Chapter X). Frerichs has given some striking examples of this.

The usual termination of diabetes is death. We have already seen how great a difference there may be in the length of time preceding the fatal termination, and in what various ways it may be brought about. The most frequent immediate causes of death are marasmus, diabetic coma, pulmonary consumption, nephritis, furunculosis, or the development of carbuncles.

There is no doubt that complete recovery may occur; but this is rare, and is possible only in the milder cases. It should also be borne in mind that apparent recovery does not exclude the possibility of a fresh outbreak of the disease.

[It should be clearly understood that the presence of sugar in the urine does not in itself indicate diabetes; and that diabetes is not now considered nearly as hopeless a disease as it was. The diagnosis was formerly seldom made unless one or all of the cardinal symptoms—increased thirst and appetite, with progressive emaciation and polyuria—were markedly present, a condition of things which may be roughly compared to phthisis in the stage of cavity. The prognosis is more grave in the young than in those past middle life, in thin than in stout people, and in those from whose urine the sugar does not disappear under dietetic treatment alone. If the bodily weight and the strength be well maintained, sugar may be excreted for long periods without any obvious ill effects.]

Theoretical Discussion of the Nature of Diabetes.—We have endeavored to give an approximately complete summary of the facts pertaining to diabetes. We trust we shall be excused from detailing all the theories and hypotheses which have been devised to explain the peculiar phenomena of the disease, particularly the glycosuria. It is a better way simply to confess that the true nature of diabetes mellitus as yet remains very obscure. We shall confine ourselves to a few remarks upon the present state of the question.

The essential fact which demands explanation is the excess of sugar in the blood. The source of this sugar is probably the same as of the sugar normally contained in the blood. The largest part of the sugar probably comes from the carbohydrates contained in the food. These are, for the most part, converted into sugar in the primæ viæ, which sugar thereupon enters the portal system. It may also be assumed that glycogen, so widely diffused throughout the system, is another source of sugar. The liver is the main seat of the manufacture of glycogen; but it is produced in other parts as well, and in particular in the muscles. The question next arises, From what is the glycogen formed? A part of it is probably manufactured from the carbohydrates contained in the food, but another part is certainly due to the ingested albuminoids. Accordingly, Von Mehring found that in fasting animals, who have no more glycogen stored up in the body, diabetes may be produced by giving phloridzine (*vide supra*). The sugar excreted can therefore come only from the destruction of albumen in the body. Again, the transformation of glycogen into sugar is not confined to the liver, but may take place wherever glycogen is produced. How it takes place is unknown. It is usually assumed that there is some "saccharific ferment."

It would seem, therefore, that the sources of sugar are the same in diabetes as in health. We have next to seek for the reason of its excessive accumulation in the blood. Under normal circumstances, the sugar present in that fluid rapidly undergoes decomposition into other substances. In health there is no great excess of sugar in the blood, even upon a diet extremely rich in starch; and it is possible to eat large amounts of sugar without any glycosuria. We see, therefore, that diabetes can not be explained by assuming that there is an increased production of sugar except in so far as corresponds to the increased amount of ingesta. On the other hand, everything points toward the conclusion that in diabetes the ordinary processes effecting the decomposition and destruction of sugar are suspended. The sugar is excreted by the kidneys unaltered, for the reason that it is not destroyed. It is difficult to conjecture what the circumstances may be which thus interfere with the decomposition of the sugar. Perhaps it is some special nervous influence, or perhaps some ferment may be wanting in diabetes which in health promotes the metamorphosis of the sugar (compare the experiments of Minkowsky, above mentioned, in producing diabetes by extirpation of the pancreas). Another point that is difficult to understand is, that in the milder cases of diabetes only such sugar as originates from the starch contained in the food is excreted, while the sugar manufactured from albumen seems to be completely decomposed. The cause of the excretion of sugar can not lie in the disturbed condition of the kidney, for in diabetes insipidus, as Frerichs shows, no glycosuria is produced, even when a very large amount of sugar is ingested.

Diagnosis.—For the diagnosis of diabetes mellitus it is indispensable that sugar should be demonstrated in the urine. We have also to decide whether the condition be a temporary or a permanent one, or, in other words, whether we have to deal with mere glycosuria or genuine diabetes mellitus. This point is to be determined by means of the symptoms and general course of the disease.

Diabetes often exists for some time unsuspected even by the physician. It may therefore be well to name over the symptoms which may be the first to attract the patient's attention, and which should therefore always suggest to a physician the possibility of the existence of diabetes. They are: 1, languor and debility; 2, furunculosis; 3, pruritus pudendi in women, balanitis in men; 4, cataract; 5, sciatica, especially if bilateral; 6, impotence. We should also be in the habit of examining the urine for sugar in all cases where complaint is made of indefinite symptoms, which can not be explained without further evidence, especially in cases where the patient is fat or nervous.

If symptoms similar to those just enumerated lead to an examination of the urine, and the result of this is ambiguous, it is advisable to have the patient partake of a meal rich in starchy elements, and to examine the urine thereafter. If even then no sugar is found, diabetes does not exist.

[The urine passed three or four hours after a full meal often contains sugar, when that passed on rising is quite free from it.]

Treatment.—Medical science does not possess the power to cure the disease, but it can greatly benefit the patient, both by alleviating his symptoms and by shielding him, at least for the time, from many of the secondary effects.

The first requisite is to institute a proper regimen. All the hygienic circumstances of the patient should be regulated. This is more important than any sort of medicinal treatment. We have seen that a large part of the food taken by a diabetic patient escapes from the body unutilized, and that, as a result of this, certain disturbances are produced in the tissues; thus there is a tendency to furunculosis and gangrene and cataract. Furthermore, the sugar contained in the urine gives rise to certain secondary symptoms, such as balanitis, and perhaps its presence in other secretions has analogous bad effects. We must therefore endeavor, first, to promote the conversion of the non-nitrogenous elements of the food; and, secondly, to furnish the system with a substitute for that portion of the food which it can not assimilate, and to obviate the excessive ingestion and production of sugar. It would be erroneous to conclude that this last point is the only essential one, and that we accomplish our whole duty by reducing the amount of sugar contained in the urine to a minimum. The general condition of the patient should invariably be considered. There can be no doubt that a diabetic patient whose strength is well maintained is better off than one whose urine contains only one per cent. of sugar but who yet is daily growing weaker.

Mental excitement has been suggested as a possible cause of the disease; and there can be still less doubt that it usually has a bad effect upon its course. The physician should therefore endeavor, so far as he is able, to guard the patient from excitement, whether incidental to his occupation or to his social position.

A proper diet is of the greatest importance. It has already been stated that in many instances the excretion of sugar may be entirely stopped by excluding starch from the ingesta; but such exclusion does not always result in permanent benefit to the patient. Cantani, however, believes otherwise, and has laid down a very strict dietetic regimen. He states that in not a few cases an almost exclusive meat diet may be persisted in for years, and the patient may at length experience complete recovery, or even acquire the ability to partake once more of starchy food with impunity. That such favorable results may occur we have no doubt; but we must call attention to the fact that it is frequently impossible to enforce the strict regimen of Cantani, and that many patients, while following it, feel worse than when they indulge in a moderate amount of carbohydrates. The "cure" affords them no relief, but merely distress. At present, authorities are generally inclined to recommend a diet mainly of flesh or albuminoids, but not absolutely devoid of carbohydrates. The amount of starch which can be safely ingested varies with the individual. Of course, the best way is to judge of the system's power to tolerate starch by means of daily quantitative estimations of the amount of sugar excreted. We repeat that, within certain limits, the percentage of sugar should not be the only criterion of the suitability of the diet, but that the general condition should also be taken into consideration.

If we consider the most common articles of diet with regard to the proportion of carbohydrates they contain, we shall find that they may be classified somewhat as follows: First, these articles may be allowed *ad libitum:* All sorts of fresh meat, ham, smoked meat, tongue, fish, crabs, eggs, caviare, sour milk, cheese, butter, bacon; also green vegetables, lettuce, spinach, and cucumbers. Secondly, the following may be used moderately: Bread, milk, fruit, rice, turnips, beets, asparagus, radishes, cauliflower; also light beer, claret, and other dry wines. Thirdly, if possible, the following should be entirely abandoned: Sweet dishes, cake, honey, potatoes, grits, sago, peas, beans, lentils, sweet fruit, sweet wine, and liqueurs.

The greatest trial for most patients is to give up bread. Every physician has had experience of the cunning which patients exercise in order to satisfy their invincible craving for it. Faults of this kind are not so apt to be committed if the patient is allowed a limited amount of bread, say two or three ounces a day in divided portions. There have also been many attempts to make a bread out of such carbohydrates as have been found by experience not to increase the excretion of sugar, and thus to furnish a substitute for ordinary bread; but these succedanea have not become popular, mainly because of their bad taste. They may, however, be tried. We have not space to describe in detail the various kinds of "diabetic bread" which have been recommended. The best known are bread made from bran flour (Prout), from almonds (Pavy), from inulin and lichenin (Külz). Most of these substances contain a considerable amount of starch.

Since we can not supply the system of a diabetic patient with carbohydrates in sufficient amount, it suggests itself that we should endeavor to supply the lack by other non-nitrogenous substances, and that the patient should be allowed a large proportion of fat in his diet. With regard to this, experience and theory coincide. Fatty substances are well borne in most cases, and we should not only allow, but urge patients who are at all emaciated to use butter, cream, and similar articles of diet. Cod-liver oil is also frequently employed, and is even regarded by many physicians as having a specific effect. It may be stated in this connection that for a time it was believed that glycerine might serve as a substitute for sugar (Schultzen); two to three ounces may be given in a day; but, with some apparent exceptions, its administration has not proved especially satisfactory.

Dühring claims that by long-continued boiling the carbohydrates may be so modified as to cease to affect the excretion of sugar in diabetes. Dühring gives his patients mainly rice and fruit, these substances having previously been soaked in water and boiled for several hours. His method also includes certain other dietetic and hygienic measures, but it has not yet been satisfactorily tested.

To quench thirst, we may allow the patient simple water or Seltzer water, and acidulated drinks. If the thirst be very troublesome, the patient may let little pieces of ice melt in the mouth. Tea and coffee may be taken with cream, but without sugar. In place of sugar, we may make trial of glycerine or mannite. Milk does not need to be absolutely banished, but few patients care for it. Alcoholic beverages may be allowed in moderation, particularly red wine, such as Bordeaux, and light beer. Thirst may also be moderated by weak brandy and water.

[The dietetic treatment of diabetes is so important that it is desirable to go more into detail.

There is some discrepancy between the authorities on this point, a more strict diet being laid down by some than by others. Ralfe's list is as rigid as any, and is as follows:

To avoid milk (except very small quantities for cooking purposes). The liver of all animals (as the liver of oysters and all mollusca is large, and abounds in glycogen, these animals must be forbidden), so also the interior of crabs, lobsters, etc. Bread, biscuits, rusks, toast, farinaceous vegetables, such as potatoes, Jerusalem artichokes, rice, oatmeal, corn-flour, sago, tapioca, arrowroot, etc. Saccharine vegetables, turnips, carrots, parsnips, green peas, French beans, beet-root, asparagus, tomatoes. Blanched vegetables of every sort, as celery, sea-kale, endive, radishes; also the stalks and white parts of such vegetables as cabbage, lettuce, broccoli, etc. Fruits of all kinds. Jams, sirups, sugars. Certain condiments, such as chutnee and sweet pickles, cocoa, chocolate, liqueurs, sweet wines.

May take meat, fish, poultry, game, bacon, ham, eggs. Bread and biscuits made with prepared gluten, bran, or almond-flour. Green vegetables, summer cabbage, turnip-tops, spinach, broccoli-tops, water-cresses, mustard and cress, laver, sauerkraut, the green parts of lettuce, sorrel, mushrooms. Nuts of various kinds (except chestnuts). Cheese.

Flint's list is more lenient, allowing oysters and a much larger choice of vegetables, such as asparagus, string beans, artichokes, cauliflower, tomatoes, etc. ("Journal of the American Medical Association," July 12, 1884; also "Pepper's System of Medicine," vol. ii, page 221).

Donkin's treatment by skim milk exclusively is highly approved by Tyson.

Rhenish and similar wines, moderate quantities of spirits, or a malt liquor in which the sugar has been entirely converted into carbonic acid and alcohol (Bass's ale, for instance), are permitted.

In the opinion of the writer, it is always well to begin treatment with a very stringent dietary, which may be relaxed gradually as circumstances dictate. The severity of the case is to be regarded rather than the name of the disease. The gluten and other diabetic flours are unreliable; and I agree with Flint that, if bread be allowed, it is better to give the crust of a French roll the ingredients of which are known. Saccharine may be used to sweeten tea and coffee, jellies, ice cream, and the like.]

Certain other general directions are important. The patient should take sufficient exercise. Külz has determined by means of accurate experiments that, other things being equal, the assimilation of sugar is increased upon increase of muscular activity, with a consequent diminution in the excretion of sugar. Practical experience also shows that regular exercise is extremely beneficial. A proper discretion should be employed, however; nothing would be more injudicious than to force a feeble patient to exhaustive efforts. But if the patient be vigorous and well nourished, he should be strongly urged to take a walking-trip in the mountains, or to try horseback riding and the like. Methodical massage of the muscles sometimes has a good effect.

Proper care of the skin is indispensable. Baths, cold sponging, and douching may be employed. Strict attention should also be given to the teeth, lest they become carious. Thorough ventilation should be maintained, both day and night.

Of internal remedies, opium should be named first. One good effect of this drug is that it lessens the annoying thirst. It sometimes also causes decided diminution in the amount of urine and sugar excreted. It is further indicated when there is general restlessness or sleeplessness. It is often well borne by diabetic patients, even in large doses. A patient sometimes can take four to eight grains (grm. 0·25–0·5), or even more, of opium in twenty-four hours without any bad effects. It is noteworthy that the alkaloids of opium, such as morphine and codeia, possess much less value than opium itself.

[If sugar does not disappear from the urine under diet alone, or if a strict diet be not tolerated for any reason, opium is indicated. The drug is usually well tolerated, and can be given in divided doses or in one dose at bedtime. If the latter course be adopted, one grain can be given and increased until the sugar either disappears or ceases to diminish in amount; this dose varies with different individuals, but when reached it can be maintained without increase for a long time.

The inhalation of oxygen is worthy of mention as a remedial measure.

Purdy seems to show that diabetes attains its highest mortality in those States which combine relatively high altitude with low temperature. A residence in the Gulf States or Southern California may therefore be curative or prolong life.]

Belladonna, cannabis indica, chloral, and bromide of potassium are also given, but they are less efficient than opium. Bromide of potassium would probably be the best of these, particularly if there were a condition of nervous excitement.

The alkalies, and still more the alkaline mineral waters, enjoy a reputation second only to that of opium. Hundreds of patients visit Carlsbad, Neuenahr, and Vichy every year to return much benefited. Of course it must not be forgotten that it is not merely the waters which produce these beneficial changes. Other factors are also important, in particular the strict diet, fresh air, and freedom from the cares of the household and business. Why the alkalies should act favorably we do not know. Griesinger, and later Külz, as well as others, have made careful comparisons of the amounts of sugar excreted under like circumstances, with and without the ingestion of bicarbonate of soda or of Carlsbad water and similar substances, and for the most part have not been able to perceive any benefit from these remedies. Practical experience, however, shows the value of these alkaline springs; and their use is to be recommended, although the expectations of the patient should not be wrought to too high a pitch.

From a theoretical point of view, there is interest in the fact that certain remedies which are antagonistic to fermentation have been shown by Ebstein and Müller to diminish the excretion of sugar in many cases of diabetes. Chief among these are carbolic acid and salicylate of sodium. The carbolic acid is given in the amount of ten to twenty grains (grm. 0·5–1·5) per diem. The amount of salicylate of sodium is one to two and a half drachms (grm. 5–10) daily. There is no doubt that these drugs possess the property ascribed to them; but they are not advantageous to the patient, inasmuch as the general condition is seldom improved by their use. On the contrary, very unpleasant effects are sometimes observed.

We need not enumerate all the remedies which have been recommended in diabetes. We have already mentioned those that possess any extended reputation. We may therefore merely refer to certain drugs which have been lately introduced.

Cantani has suggested the employment of lactic acid in the amount of one to two and a half drachms (grm. 5–10) per diem, dissolved in half a pint of water. This drug may perhaps serve as a physiological substitute for sugar, as glycerine is supposed to do (*vide supra*), but it has no specific virtues.

Certain salts of ammonia, such as the carbonate and acetate, are said to diminish the excretion of sugar, and have therefore long been employed in diabetes, but without special benefit.

Iodoform has been recommended by Moleschott to the amount of three to six grains (grm. 0·2–0·4) per diem. It is said not only to diminish the amount of sugar in the urine, but also to alleviate other symptoms. Arsenic, tincture of iodine, and quinine have also been employed; and we may mention that even electricity has been tried—we need hardly say, in vain.

It is evident, in brief, that the best mode of treating diabetes, at least according to our present knowledge, is by regulating the diet, and that it is well, in addition, to recommend the employment for a time of the above-mentioned mineral waters, with opium and other internal remedies to combat special symptoms. The treatment of such complications as phthisis or cutaneous eruptions need not be described here.

In diabetic coma, camphor or ether, subcutaneously, should be employed, together with lukewarm baths and douching. As it is possible that the coma may be due to poisoning from the acids in the blood (*vide supra*), we should also try bicarbonate of sodium in large doses, and in some cases use intravenous injections of a three to five per cent solution, or give it subcutaneously; but the efficiency of such treatment remains to be decided. Experience thus far is not very encouraging for further trials.

CHAPTER X.

DIABETES INSIPIDUS.

Definition and Ætiology.—In the preceding chapter a distinction was drawn between diabetes mellitus and the symptomatic condition termed glycosuria. There is a similar distinction to be made between diabetes insipidus and polyuria. Polyuria is an increase in the volume of urine, and mainly in the amount of water excreted by the kidneys. It is a symptom which may be produced in many different ways. In the first place, it is a natural consequence of the ingestion of large amounts of water, or of the absorption of serous effusions; it also occurs in certain diseases of the nervous system, especially of the medulla and cerebellum; it is also occasionally seen, as we have had opportunity to observe, in chronic hydrocephalus, and is a not very infrequent phenomenon in hysteria. Large amounts of urine are also secreted in certain renal diseases (interstitial nephritis and amyloid degeneration), and often during convalescence from acute diseases, such as typhoid fever, or after the ingestion of certain drugs, called diuretics.

Diabetes insipidus, on the other hand, is a disease which may develop idiopathically in people otherwise perfectly healthy. Its ætiology is unknown. It occasionally seems to be excited by emotional disturbance, concussion or other injury of the brain, or some previous acute disease, such as typhoid or typhus fever, malarial poisoning, and cerebro-spinal meningitis. The disease sometimes appears in the syphilitic, and may, therefore, in many instances, be due to syphilis. Patients frequently state that their symptoms began immediately after drinking a very large amount of some fluid, as on a very hot day or after a long march. In such cases the assumption is a probable one that the primary symptom is not polyuria, but an abnormally great thirst (polydipsia), the increase in urinary secretion being a result of the large amount of water ingested. Finally, the disease may be hereditary (*vide infra*). The true nature of diabetes insipidus is entirely unknown to us.

The view which seems most probable of any is, that some nervous disturbance is its direct cause. In support of this, we have the appearance of a "symptomatic diabetes insipidus" in connection with organic disease of the brain (*vide supra*), and the fact that polyuria may be artificially excited by injury to a definite spot in the floor of the fourth ventricle or by section of the vagus nerve. Diabetes insipidus presents a most striking analogy with diabetes mellitus. This is shown both by the similarity in ætiology and symptomatology, and still more by the fact that occasionally one disease merges into the other.

Diabetes insipidus is a disease of very infrequent occurrence, and, at least in Germany, is decidedly less often seen than diabetes mellitus. Most patients are in young adult or middle life. Males are somewhat more liable to the disease than females.

Clinical History.—Diabetes insipidus may be developed gradually or with considerable abruptness; the latter case is especially frequent when there is some definite cause, such as the ingestion of a large amount of liquid, or traumatism.

The essential and characteristic symptom is an increase in the volume of urine. This is usually very considerable. Often eight or ten quarts (8000 to 10,000 c. c.) are excreted in twenty-four hours, and cases have even been reported where the amount reached the almost incredible volume of thirty to forty quarts (litres). If a healthy person and a sufferer from diabetes insipidus are each given the same amount of water in food and drink, the sick man will excrete more urine than the healthy. In color the urine is very pale, and sometimes almost like water. The specific gravity is very low, being usually 1004 to 1002, or even 1001. The reaction is slightly acid, sometimes almost neutral.

The percentage of solid constituents in the urine is, of course, trifling. The total amount of solids, however, corresponds perfectly with the ingesta, or indeed is even somewhat above normal. The amount of urea in particular seems to be increased, but it has also been stated that other constituents of the urine have been excreted in abnormally large amounts—namely, phosphoric acid, sulphuric acid, lime, and kreatinine. Inosite has been found in the urine by Strauss and other observers, so that it has even been proposed to give diabetes insipidus the name of "diabetes inositus," in distinction from diabetes mellitus. Inosite is not invariably present, however, in the urine of diabetes insipidus. In cases of true diabetes insipidus, albuminuria is extremely exceptional.

Another important symptom is the excessive thirst. To make up for the great loss of water by way of the kidneys the patient is obliged to drink great quantities of liquid, and, indeed, it is always found that the amount of water ingested, in the way of drink and solid food, somewhat exceeds the total volume of urine excreted. Despite this, the tongue is usually dry, as is also the skin, the insensible perspiration being considerably below the normal amount. The furunculosis seen in diabetes mellitus is exceptional in diabetes insipidus. The same is true of pruritus and balanitis. Occasionally profuse salivation has been associated with the disease.

Symptoms referable to the various internal organs are few. Cataract has been occasionally observed, but it is much less frequent than in diabetes mellitus. The same may be said of pulmonary tuberculosis. In most cases the appetite is not excessive. The bowels are regular or slightly constipated. There is seldom much gastro-intestinal disturbance, unless from some chance complication. The sexual functions are usually unimpaired.

The general health is considerably affected in cases of any severity. The patient becomes emaciated, languid, and feeble, and has no inclination to mental or physical exertion. Sleep is often disturbed, the mind depressed. The temperature is normal, or even a trifle below normal, probably as a result of the large amount of cold water drunk.

Diabetes insipidus is a very chronic disease. If there is no serious complication it may last for decades, yet there are cases that run a more rapid and unfavorable course. Sometimes there are considerable vicissitudes in the condition of the patient, in part dependent upon external circumstances and in part apparently spontaneous. In case some intercurrent acute disease develops there may be, during its continuance, a considerable diminution in the amount of urine excreted.

The termination is usually unfavorable. Recovery is extremely rare. In the more fortunate cases the condition finally becomes stationary, and the patient attains to advanced years. Sometimes, however, death occurs more prematurely, being usually hastened by phthisis or some similar complication.

Weil has lately contributed to our knowledge of this disease the results of an accurate study of its hereditary and apparently congenital variety. Weil narrates the history of a family in which marked polyuria and corresponding polydipsia appeared in numerous members for several generations. These persons all enjoyed excellent health, with this exception; and most of them attained old age. We hardly need to emphasize the fact that this form of the disease is radically different from the ordinary acquired variety. Perhaps its true cause is an abnormal congenital permeability of the glomeruli, but we have no certain information in regard to it.

Post-mortem Appearances.—Such lesions as have been found in diabetes insipi dus are usually the result of fortuitous complications, such as tuberculosis, carcinoma, and pneumonia. There are but very few changes referable directly to the disease itself: among these are enlargement of the kidneys and dilatation of the ureters. In rare instances a possible cause for the symptoms has been found in some lesion of the central nervous system, but these were properly cases of symptomatic polyuria and not of genuine diabetes insipidus. Instances of this sort are seen in connection with tumors or inflammatory changes in the medulla or cerebellum, and exostoses at the base of the skull.

Diagnosis.—The characteristic urinary phenomena usually render the diagnosis of diabetes insipidus an easy matter. It is of course necessary to exclude such diseases as might occasion symptomatic polyuria (*vide supra*) ; but this is seldom difficult if we make a careful physical examination and carefully consider all the attendant symptoms. The differential diagnosis between diabetes insipidus and diabetes mellitus can almost invariably be made by means of the urinometer. If the specific gravity is below normal, it is scarcely necessary to test for sugar.

Treatment.—No special injunctions with regard to diet are required. It would of course be a mistake to forbid the patient to assuage his excessive thirst; but we may possibly lessen the amount of water drunk by prescribing bits of ice, or lemonade and other acid drinks. Opium sometimes lessens both the thirst and the amount of urine excreted. It is also important that the skin should be well cared for by means of baths and friction, and every effort should be made to promote the general vigor of the patent. He should have nourishing food and good air.

Numerous internal remedies have been recommended as specific, but few of these have won any great reputation. Valerian appears, on the whole, to be the most efficient drug, and may be given in powder or infusion to the amount of one to two and a half drachms (grm. 5-10) per diem. Ergotine may also be tried. Carbolic acid, salicylate of sodium, and nitric acid have been said to prove beneficial. It is also said that galvanization of the medulla and upper part of the spinal cord sometimes accomplishes good results.

Occasionally we may find an apparent cause for the disease and endeavor to remove it. If there is a suspicion of syphilis, mercurial inunctions should be tried. Sometimes, as we can confirm by our own experience, they have an excellent effect. Of course, where there is symptomatic polyuria, the primary disease, such as hysteria, demands treatment.

[Da Costa and others report very good results as following the administration of ergot.]

CHAPTER XI.

GOUT.

(*Podagra.*)

Ætiology.—Thomas Sydenham was the first to write a careful clinical description of gout. He himself suffered from the disease for about forty years, and has given a detailed description of his own case in the treatise which he published in 1683, under the title "Tractatus de podagra et hydrope." It was, however, Wollaston who, in 1797, threw the first light upon the peculiar anomaly of tissue-metamorphosis which exists in this disease. He demonstrated that the gouty deposits found in the joints and other parts of the body are mainly uric acid. From his time an all-important point with regard to the nature of the disease has been the relation between the symptoms of gout and disturbances in the manufacture and excretion of uric acid. In 1848 Garrod showed that in gout the blood contains an excess of uric acid, and that the excretion of uric acid by the kidneys is diminished. He was thus in a position to frame a theory consistent with all the clinical facts. Numerous investigations have been undertaken since his day; but we still remain with regard to gout in a position analogous to that which we hold toward diabetes. We are, it is true, in the possession of a considerable number of well-established facts relating to it, but we do not know why the normal chemical processes are disturbed, and are unable to explain the connection between the various phenomena observed.

Clinical experience has taught us certain remote causes of gout, first of which comes heredity. About fifty per cent. of all cases occur in patients whose families have already suffered from the disease, and it is sometimes possible to trace this transmitted taint through many generations. It is decidedly more apt to pass down through the male members of the family than through the female.

Next in importance to hereditary influences is the mode of life. From time immemorial this has been regarded as an exciting cause of the disease. It has been a matter of universal belief that over-feeding, and especially the ingestion of too large a quantity of albuminoids, is strongly provocative of the disease. The same opinion has been held with regard to persistent over-indulgence in alcoholic beverages. Seneca relates that at the time of the decay of the Roman Empire women practiced such excesses that they were as subject to gout as the men, and an old verse runs: "Wine is the father of gout, feasting is its mother, and Venus is the midwife." This view is, however, an exaggerated one. It can not be denied that there is some truth in it, but, on the other hand, gout is not exclusively an "arthritis of the rich." It occurs also among the poor, who have had only too little acquaintance with the pleasures of the table; and many a *bon vivant* has reached old age without ever experiencing pain in his great toe.

[Gout is a disease so much more common in England than in Germany or this country that English opinions in regard to it are deserving of especial weight. The most commonly accepted, though by no means the only, view as to its nature is that it depends on defective oxidation, which may be brought about in two ways: either by the ingestion of more food than can be properly oxidized, or by the presence of such conditions that even a moderate supply of food can not be worked up and undergo its proper transformations. This theory will account for gout in the poor as well as in the rich.

There can be no question that the use of malt liquors, especially in the stronger forms, consumed so enormously in England, is much more favorable to the development of gout than is the use of distilled spirits; so also the stronger wines—such as port, sherry, Madeira, and the heavy Burgundies—are more dangerous than are the lighter and more acid wines of France and Germany. In this country gout is becoming more common, a fact which may be fairly explained by the accumulation of wealth and the consequent growth of luxury.]

There is a noteworthy, although mysterious, connection between gout and chronic lead-poisoning. The fact is well established that persons who have to do with lead, such as type-setters and house-painters, are subject to genuine gout with deposits of uric acid in the joints.

With regard to still other alleged ætiological factors, confirmatory evidence is lacking. Possibly, however, when the foundation for the disease is already laid, an attack may be excited by certain determining causes—namely, trauma, taking cold, errors in diet, and mental emotion.

The geographical distribution of gout is remarkably unequal. The disease is much more frequent in England than in Germany, although in the latter country certain regions appear to be more affected by it than others. In Leipsic, in Saxony generally, and in Bavaria, gout is decidedly rare.

Gout is rarely seen in young persons. It is a disease of advanced life, rarely appearing previously to the fortieth year. Men are much more often attacked than are women.

Clinical History.—Gout may produce symptoms in many different organs; but its effect upon the joints is so characteristic that the arthritic disturbance has long been termed "normal or regular gout," in contradistinction from "atypical, internal gout." This distinction is of course an artificial one, for the various phenomena of gout present the most manifold gradations and transitions. It will, however, be advantageous, in attempting to gain a practical insight into the various symptoms of the disease, if we first discuss the so-called "typical attack of gout," subsequently appending a description of the other manifestations of the disease. Furthermore, the regular attack of gout is, in at least a majority of cases (*vide infra*), the first and earliest symptom of the disease.

1. The typical attack of gout is seldom abrupt. It is usually preceded for a longer or shorter period by certain premonitory symptoms, the meaning of which, though not evident to one who is about to suffer from his first attack, is sufficiently clear to more experienced patients, particularly as each individual case is apt to present a marked similarity in the prodromata of the separate paroxysms. These premonitory symptoms vary in different individuals. Sometimes they consist in dyspeptic disturbances; sometimes in a feeling of languor and mental depression; very often in dragging, muscular pains or cramps in the calves of the legs; or again in slight feverishness, with chilliness, sense of heat, and perspiration. On the other hand, it must be confessed that a patient may feel unusually well just before an attack.

The attack is noticeably apt to begin in the night-time, or very early in the morning. The patient is awakened by a sudden and very violent pain in the metatarso-phalangeal joint of one of the great toes ("podagra"). The joint becomes swollen, the skin over it red, hot, and tense, the veins in the neighborhood distended. At the same time there is chilliness and moderate fever. This condition persists till morning. Then the pain is almost sure to abate, the fever to remit at the same time that sweating begins, and the patient to feel tolerably well during the day. The joint, however, remains swollen, with inflammation and œdema. The next night the pain begins again, and there is a fresh fever; and these alternations are repeated, as a rule, for three to ten days. Even where the attack is more persistent than this, the pain is usually much less severe after the first two or three nights. After that time it gradually abates; and it is commonly said that an attack is brief in proportion to the violence of its first symptoms. When the pain ceases, swelling soon disappears, the skin undergoes a slight desquamation and resumes its normal appearance, the general health of the patient rapidly improves, and he is often found to be much better after an attack than he was before.

For theoretical purposes (*vide infra*) it would be very advantageous to possess a more accurate knowledge of the condition of the urine, and in particular of the excretion of uric acid during the attack. As yet, however, there have been few careful investigations made. Garrod has made a very important observation, which has been confirmed by Cantani: it is, that the amount of uric acid excreted diminishes several days before the commencement of an attack, and remains diminished during the attack. Subsequently to the attack the excretion of uric acid is said to be above normal, while the amount of uric acid in the blood varies in precisely the opposite way—that is, during the attack it is increased, and after, it is diminished. It has not yet been determined how far this variation in the excretion of uric acid may be referable to the altered diet of the patient, and how far to a diminished formation of uric acid or a deposition of that substance in the diseased joints (*vide infra*).

If there has been one attack of gout, there are almost sure to be others. They come sooner or later, at regular or irregular intervals, and separated by weeks, months, or even years. The attacks recur at long intervals in mild cases, more frequently and at gradually diminishing intervals in the severe. Spring and autumn are regarded as the time when attacks of gout are most apt to occur. In these subsequent attacks the great toe still remains the part most constantly and severely affected; but other joints may also suffer—for example, the wrist, the knee, or the shoulder. Sometimes traumatism or some previous affection of the joints—such as rheumatism—apparently determines the localization of the gouty disturbance. In each attack, the trouble is usually confined to a single joint. It is only in exceptional or advanced cases that several joints are simultaneously invaded.

The longer the disease has lasted, the less typical are the separate attacks. There may now be less suffering at the time of an attack; but the articular changes are more persistent. There are symptoms referable to other parts of the body; and the gout gradually passes into its second chronic or "atonic" stage. Occasionally the disease is irregular and atypical from its incipiency; and the first manifestations may not be arthritic, but referable to the kidneys (*vide infra*) or other organs.

2. *Atypical Gout. Gouty Disturbance of other Parts than the Joints.*—Gout may affect the mucous membranes. A gouty dyspepsia is very frequent. Its symptoms are more or less severe. The gouty are also subject to intestinal catarrh of varying severity, and to bronchitis and conjunctivitis, as well as to catarrh of the urinary organs. Ebstein regards "gouty gonorrhœa" as essentially a catarrh of the excretory ducts of the prostate gland. It is not an easy matter to explain why these various forms of catarrh should occur in gout. They may be, some of them, complications. In other cases they are doubtless the result of passive congestion, due to cardiac failure (*vide infra*); but in still other instances it must be confessed that they are apparently due to the toxic influence of the accumulated uric acid.

[My experience, though far more limited than that of Draper, nevertheless leads me to believe, as he does, that the irregular forms of gout are by no means uncommon in women as an inheritance from a previous generation. The manifestations in some of these cases are so ill-defined that a diagnosis may be difficult without a knowledge of the family history; in other cases, more or less deformity of the smaller joints, or slight recurrent swellings of those parts, throw much light on the condition underlying the symptoms.]

Inflammation of serous membranes—for example, of the pleura—also occurs; and there may be pneumonia. The skin not infrequently suffers from acute or chronic eczema, which sometimes appears to be referable directly to the gout. Keratitis, iritis, and other inflammatory disturbances of the eye are also said to be caused directly by gout. Cirrhosis of the liver has been repeatedly found, and is perhaps referable to the action of the uric acid upon the hepatic parenchyma. By far the most important of all these gouty manifestations are referable to the kidneys and to the circulatory system. The disorder of the latter is sometimes symptomatic of the renal trouble, but in other instances it occurs independently of it. There may be severe gouty arthritis for years without any lesion of the kidneys; but this is exceptional. In severe cases of gout it is the rule that, sooner or later, symptoms of renal disorder present themselves. The so-called "gouty kidney" is a form of chronic interstitial nephritis. However important this complication, its symptoms do not need to be discussed here, as they are precisely similar to those seen in ordinary cases of contracted kidney (*vide* page 856, *et seq.*). The distinctive symptom of this disturbance is albuminuria; and the gradually developing secondary hypertrophy of the left ventricle is the pivot on which turns the whole future course of the disease. So long as the heart remains capable of performing its functions, the condition of the patient will probably remain endurable if not actually comfortable. Finally, however, compensation is sure to become impaired; and then appear œdema, dyspnœa, debility, and emaciation—in short, all the familiar symptoms of cardiac failure. A speedy end may be brought about by uræmia, or cerebral embolism, or hæmorrhage; but in other cases the patient suffers for years, both from the heart disease and from fresh arthritic attacks.

The cardiac hypertrophy just mentioned is the result of the contracted kidney. Other disturbances of the circulatory system appear to be referable directly to the gout itself. Among these are chronic endocarditis or myocarditis, and perhaps certain "functional" symptoms, such as palpitation and angina. An important symptom is chronic endarteritis or arterio-sclerosis. This is often seen in gouty subjects, and in many instances seems to be immediately connected with the gout. Sometimes, also, there are gouty lesions of the veins, such as varicosities or thrombosis. Of course, these changes in the blood-vessels may, in their turn, give rise to various disorders.

In a few very rare instances gout seems to attack the brain and spinal cord. Usually, however, the nervous disturbances seen in gout are, as already stated, symptoms of uræmia, or of circulatory disturbance, and the like. The patient may also have certain functional nervous troubles, like neuralgia or migraine. The direct cause of these is seldom evident.

The joints, despite frequent attacks, may yet maintain an almost normal appearance, inasmuch as the acute inflammatory changes completely vanish after each separate attack. They may, however, undergo permanent enlargement and deformity from gouty concretions (*tophi arthritici*). In many instances similar masses can be felt here and there in the muscles and tendons, the skin (for instance, of the eyelids), and quite often in the external ear. They consist essentially of accumulations of urates (*vide infra*). Occasionally these concretions break externally, discharging a thick pus mingled with urates, and forming indolent ulcers, slow to heal. Sometimes atmospheric germs thus find ingress into the system, and give rise to phlegmon.

It should be stated, in conclusion, that gout may be complicated with other diseases. Thus, it may be associated with renal calculi, and sometimes with diabetes mellitus (*vide supra*).

Anatomical and Chemical Changes in Gout. Theory of its Nature.—The essential anatomical lesion in gout consists of an abundant deposit of crystalline urates in the tissues. This is most evident in the affected joints, the cartilaginous surfaces of which are often completely covered with white, chalk-like material. In severe cases the same appearance may also be presented by the articular ligaments, tendons, and bursæ, while there are at the same time numerous concretions of urates here and there beneath the skin. These deposits are mainly composed of acid sodic urate, with traces of calcic urate, calcic phosphate, and sodic chloride. Ebstein has lately explained how these collections are formed. He found that the deposition of uric acid is invariably preceded by a necrosis of the tissues. The uric acid while still in solution acts as a chemical irritant upon the cartilage here and there, and thus produces necrosis, whereupon the urates are crystallized out and deposited. Then a secondary inflammation develops around these foci of necrosis. By ligaturing the ureters in fowls, Ebstein succeeded in artificially producing similar changes.

The nephritis of gout corresponds in its pathological appearances to that of the true contracted kidney, with one exception; the organs usually present deposits of uric acid or urates, in stripes, both in the lumen of the urinary tubules and also in the epithelial cells and the interstitial tissue. In the connective tissue it is probable that a necrosis precedes the deposit.

The lesions presented by the heart, blood-vessels, and remaining parts of the body are not especially characteristic of the disease. The blood of gouty subjects, as Garrod demonstrated, contains an excess of uric acid.

There are many theoretical questions which remain unanswered: Whether in this disease there is an over-production, or merely a defective excretion of uric acid? What the true cause of this strange anomaly in metamorphosis may be? What conditions are essential to the crystallizing out of uric acid in the tissues? Why certain parts, particularly the joints, and still more particularly the first joint of the great toe, are especially liable to suffer? And, finally, What circumstances decide the course of the disease, and determine its separate attacks? Not one of these questions has been satisfactorily answered. The fact that gout is prone to attack individuals who have led a luxurious life has led to the belief that in every case of gout the ingesta are not completely oxidized, and an excess of uric acid is consequently accumulated in the system. We have already pointed out that this assumption is too sweeping. And, indeed, our present knowledge with regard to the manufacture of uric acid out of the albuminoids, and its further transformations, is not at all adequate for the establishment of any hypothesis of this sort. We must simply confess that the true cause of the accumulation of uric acid in the tissues is unknown. It may be conjectured, however, that the deposit of crystals of uric acid is either occasioned or promoted by an excessively acid reaction of the blood or lymph in which that acid is dissolved. It is not known what acids give rise to this reaction, nor how they are formed. It has been seen that the articular cartilages are especially apt to present a gouty deposit. Perhaps this is occasioned by the sluggishness of the circulation in these parts. It is doubtful whether the uric acid is produced in the cartilage itself. Ebstein believes that it originates mainly in the muscles and bone-marrow, and is thence conveyed to the cartilage. Others, and among them Cantani, believe that the uric acid is formed in the cartilage and the connective tissue.

Diagnosis.—It is usually easy to recognize an acute attack of gout. The sudden onset of the pain at night and its localization in one of the great toes are very characteristic symptoms, and render it easy to distinguish between it and other acute affections of the joints. The diagnosis is more difficult in the advanced stages, where symptoms are more confused. Here the history of the case will often include the description of typical attacks and also ætiological factors, such as heredity and mode of life, which may assist in diagnosis. It must be said that many gouty subjects are reticent as to their past experiences, and sometimes even deny previous attacks of gout. If there is a chronic gouty arthritis, it may be necessary to make a differential diagnosis between it and arthritis deformans. The deformities of arthritis deformans are usually seen first of all in the hands and fingers, and it has a persistent, chronic course. Furthermore, in gout we sometimes can feel the characteristic deposits in the tendons or skin (for instance, in the eyelids or external ear).

If there is chronic nephritis of gouty origin, its source can be recognized only from its association with other indubitable symptoms of gout, unless possibly the knowledge of certain ætiological factors, such as a history of gout in the family or of chronic lead-poisoning, should put us upon the right track. Ebstein has reported cases of "primary renal gout" where there was no arthritis during the whole course of the disease. Such cases can seldom be correctly diagnosticated during life.

Brief mention should here be made of an experiment performed by Garrod, which may be employed to demonstrate the existence of uric acid in the blood, for diagnostic purposes. A drachm or two of blood-serum, or of the serum obtained from a fly-blister, is put into a shallow watch-glass, and six or eight drops of a thirty-per-cent. solution of acetic acid are added to it. A linen thread is then laid in the fluid, and the whole allowed to remain at a moderate temperature for about a day. If the proportion of uric acid in the fluid is sufficiently large, crystals of that acid will now be found on the thread and may be recognized by their shape and chemical reaction. This "thread test" of Garrod's is not extensively used, because it does not succeed unless there is a comparatively large amount of uric acid in the blood, and, furthermore, uric acid may exist in the blood in health, or in other diseases than gout.

Prognosis.—However favorable the prognosis may be with regard to the single gouty attack, yet a permanent release from the disease is rarely to be hoped for. Only such patients as, from the first appearance of gout, pursue most carefully all the requisite prophylactic and dietetic measures, can expect that future attacks will be rare and comparatively mild, and that severe lesions of the internal organs will not occur. So long as the kidneys and other viscera are intact, there is no immediate danger to life, and the patient may attain advanced years despite his gout. The gradual and chronic alterations in the joints may, however, impede locomotion as well as all other movements of the body. Except for this, the general condition of the patient in the intervals between the attacks is often one of tolerable comfort. Indeed, it is frequently the case that the patient will feel his very best directly after a severe paroxysm, while rudimentary or atypical attacks are often regarded as of ill omen. Really, however, there is no serious danger until a chronic nephritis is developed. The prognosis then becomes as unfavorable as in the other forms of contracted kidney (*q. v.*), and involves the same possibilities.

Treatment.—All authorities agree that the first essential in treating gout is a proper regimen. Unless the patient has energy enough to yield the most implicit obedience to the injunctions regarding diet and mode of life in general, from the first appearance of his disease, no great benefit can be obtained from treatment.

Various authorities have, of late, prescribed special diets for the gouty. These differ considerably from one another, but the discrepancies are not actually so great as they seem to be at first glance; and, after all, more importance attaches to the quantity of the ingesta than to their quality. As most gouty patients are corpulent, the diet to be prescribed for them is mainly that indicated by their corpulence. The first point is to limit the total amount of ingesta. No more should be eaten than is absolutely required to satisfy hunger. The diet may be a mixed one—that is, may contain albuminoids, carbohydrates, and fats; but the quantities of each of these ingredients should be small (*vide* following chapter). The albuminoids should not be too abundant, in order that the formation of uric acid may be limited; the fats and carbohydrates should be cut down, in order that the albuminoids may be thoroughly oxidized and thus any further deposit of adipose tissue avoided. Acid articles of diet should be forbidden, lest they promote the deposit of uric acid in the tissues. The experience of certain physicians tends to show that a diet mainly vegetable is better borne by gouty patients than animal food. In this case also, however, it will be seen that the essential point is the quantity. With a vegetable diet, the amount of ingested food, and still more the amount of nourishment absorbed from the primæ viæ, is almost sure to be less than upon an exclusive meat diet. The patient should, therefore, be told that his diet should consist mainly of lean meat, fish, broth, green vegetables, small amounts of milk, eggs, and bread. Sweet puddings, fat meat, potatoes, or sour and acid food of any kind should be avoided. Fruit may be allowed in small quantities. Water is the best beverage; but it is not advisable, and may even be prejudicial, to drink too large an amount of any fluid (*vide infra*). Large quantities of alcohol are sure to injure the patient. If they can not be absolutely proscribed, yet at least their amount should be reduced to a minimum.

[Many gouty persons, especially those suffering from the irregular form of the disease and acid dyspepsia, are more comfortable and seem to do better on a diet which is largely nitrogenous, the starches and sugars being greatly limited in amount. The diet, in fact, should be similar to that laid down for diabetes mellitus, though not so strictly carried out. In chronic and irregular cases it is often desirable to prescribe a stimulant, for a time at least; brandy, whisky, or gin, well diluted with water, is perhaps the best form.]

By thus limiting the amount of food taken, we shall promote metamorphosis and avoid any excessive formation of uric acid. A more direct means of hastening the conversion of the albuminoids is muscular exercise. If a corpulent patient is still vigorous and is not threatened by any serious internal disease, he should be urged to take a large amount of exercise in mountain-climbing, gymnastics, gardening, or similar pursuits. The motto of such a patient should be "little sleep and great activity." This same indication of accelerating tissue-metamorphosis is fulfilled also by bathing. In early stages cool baths and sponging are useful, as are also baths containing common salt, or perhaps even sea-bathing cautiously employed. In more advanced cases, particularly if the joints present permanent lesions, the warmer baths are more desirable, such as are found in Teplitz, Wildbad, Wiesbaden, Baden-Baden, Carlsbad, Ems, and Aix.

The internal administration of alkalies is an efficient adjuvant to the dietetic and hygienic prescriptions above enumerated. For a long period the use of alkaline mineral-waters has been found to be most beneficial. Experience confirms the confidence which theoretical considerations would lead us to have in their value in checking the deposit of uric acid, so far as it is occasioned by excessive acidity of the circulatory fluids (*vide supra*). The sodic chloride contained in these waters stimulates the conversion of the albuminoids. The waters also increase the activity of the kidneys, benefit gastric catarrh, and overcome constipation, and in all these ways combine to produce a favorable effect upon the patient's health. Still another factor is the judicious diet and mode of life at such health resorts. The waters of Carlsbad and Vichy have gained the greatest repute in gout, although the waters of Ems and Neuenahr have an analogous composition, and doubtless would produce similar effects. Of the waters which contain sodic chloride, the most advisable are Wiesbaden, Baden-Baden, Kissingen, and Homburg. The salts of lithium especially promote the solution of uric acid, and of late the waters which contain lithium have been strongly recommended. The natural springs of this sort, such as the Crown Spring in Obersalzbrunn, and those of Assmannshausen and Salzschlirf, contain comparatively insignificant amounts of lithium; and it might be a better way to use the artificial lithium waters, such as are made by Struve or Ewich. Another way is to prescribe carbonate of lithium, in powders containing two or three grains each (grm. 0·10–0·20), one powder two or three times a day, in a glass of Seltzer or Biliner water.

Other remedies formerly in vogue were said to correct the "gouty diathesis," but their efficacy is extremely dubious, and they need not be especially mentioned.

As to the treatment of the acute attack, it has ceased to be customary to employ any potent remedies. The patient must, of course, keep his bed. The affected joint should be wrapped up in cotton wool, the whole leg elevated, and a proper diet strictly enjoined. Free movement of the bowels should be maintained by means of an enema. If there is considerable gastric disturbance, bicarbonate of sodium, magnesia, or some bitter may be prescribed. The most certain remedy for severe pain is a subcutaneous injection of morphine. Less efficient are narcotics locally applied, and warm compresses. Whether any internal remedies are calculated to abbreviate the attack is doubtful. Formerly colchicum (twenty or thirty drops of vinum colchici seminis three or four times a day) was the favorite medicine, but it seems to be going out of use. Salicylic acid and salicylate of sodium may be administered in the same way as in acute articular rheumatism, and sometimes, although not always, they are followed by improvement. Antipyrine, and perhaps antifebrine, sometimes succeed in allaying the pain in the affection of the joints. Some physicians recommend subcutaneous injections of antipyrine in the neighborhood of the joint, but we have no personal experience on this point.

[One reason why colchicum has fallen into relative disuse is that the relief obtained from it is often so prompt and complete that patients are tempted into continued indulgence in a faulty manner of life. It is also supposed by some that the drug interferes with the effort of nature to eliminate the poison, which becomes generally diffused, and sets up changes in the vessels and internal organs.

Precisely how colchicum acts we do not know, but that it does act, and sometimes with marvelous success, there can be no question.]

The chronic affections of the joints in gout are treated as are other varieties of chronic arthritis (*vide* page 915). The most efficient remedies are cautious massage and baths, including hot sulphur baths and mud-and-sulphur baths. The internal administration of the alkalies, lithium, and similar drugs, to combat the general gouty diathesis, should be associated with these external remedies. Some physicians report that iodide of potassium favors the absorption of the gouty deposits.

The treatment of the nephritis and other complications need not be discussed at length. The gout itself should always be the main object of our therapeutic efforts, and beyond this we may be guided by general principles.

CHAPTER XII.

OBESITY.

(*Corpulence. Polysarcia adiposa.*)

Definition and Ætiology.—The amount of adipose tissue in the body is subject to considerable variation, and it is not possible to state absolutely what should be considered as normal and what as abnormal. For practical purposes we may draw the line where the increased size grows burdensome to the individual. After a certain point, any further addition to the amount of fatty tissue is almost sure to work serious injury, and is therefore to be regarded as an actual disease, and not merely an inconvenience. It should be said, however, that in such cases the symptoms of obesity are very frequently confounded with those springing from other disorders—these latter possessing, indeed, the same ætiology as obesity, but distinct from it.

The most frequent and important cause of obesity is the habitual ingestion of too large an amount of food. By "too large" is meant an amount sufficient to occasion a continual increase of the adipose tissue of the body, when this is already fairly well developed. It is a matter of indifference whether the excess is composed mainly of albuminoids, or carbohydrates, or fats; for in either case, if the quantity be sufficient, an increase of adipose tissue may take place. We shall soon see, however, that the excess is usually in fat and carbohydrates. Inasmuch as this overfeeding is almost certain to be habitual, the excess at any one time need not be at all large. We often hear a corpulent person express his surprise that he grows heavier every day, although he "does not eat any more than others who are lean." The explanation is easy if we consider that a daily increase of five grammes of fat (one and one fourth drachms) suffices to increase the weight in ten years, or, say between the thirty-fifth and forty-fifth years, forty pounds avoirdupois. In reality the daily increase is sometimes greater than this.

The basis for a detailed consideration of the causes which lead to the deposit of fat will be gained by a consideration of the physiological laws relating to nutrition discovered by Voit, Pettenkofer, and their pupils. It has been shown that both the albuminoids and the carbohydrates of the food may be a source of fat, formed within the economy, and also that the fat contained in the food may be to a large extent directly deposited in the fat-cells of the body. One product of the decomposition of albuminoid substances is invariably fat. This usually undergoes further oxidation, but it is sometimes retained unaltered in the system. It would even seem that the albuminoids give rise to much more of the fatty tissues of the body than are produced from the carbohydrates ingested, although there is no doubt that these latter also yield fat. Carbohydrates do promote obesity, but less because they are a direct source of fat than because they are easily decomposed, and so shield both the ingested fat and that which is formed out of the albuminoids from oxidation. In this indirect way they do greatly favor a tendency to corpulence.

We thus perceive that various diets may, each one of them, permit of an increase of adipose tissue. In actual life, of course, the most frequent conditions are such as result from the customs and habits of the population in general. The diet is almost invariably a "mixed" one—that is, it contains albumen and fat and carbohydrates—and in most instances the obesity is due to an excessive amount of all three elements, or at least of the fat and carbohydrates. A person may, however, become corpulent who eats very little fat, provided he consumes a large quantity of albuminoids and carbohydrates; or if he eats very little starchy food, provided he consumes a large amount of meat and fat. Perhaps these facts may be made clearer by giving a concrete example. Voit tells us that a vigorous man who requires daily 118 grm. of albumen and 259 grm. of fat to maintain a physiological equilibrium as regards fat and albumen will, other things being equal, begin to store up fat if there is any further increase in the amount of fat in his diet. The same result will also take place if, instead of the rations previously stated, he ingests more than 118 grm. of albumen and 600 grm. of starch, or more than 664 grm. of albumen alone, or, finally, more than 118 grm. of albumen, 100 grm. of fat, and 368 grm. of starch.[*] It is obvious that this last diet, which closely resembles the average diet of an adult[†] in good circumstances, whose weight is neither increasing nor diminishing, is the one most likely to be exceeded; whereupon there must take place a deposition within the system of the superfluous fat.

Among the various kinds of food is one group which deserves mention, as being an important factor in many cases of obesity; we refer to alcoholic beverages. There can be no doubt that intemperance in this regard plays a prominent part in many instances. Sufficient illustration is furnished by brewers, hotel-keepers, and the inhabitants of countries like Bavaria, where beer drinking is prevalent. In this particular it is evident that beer works more harm than does wine or strong liquor; for beer contains, in addition to the alcohol, an appreciable amount of starchy material, making the sum total from the beer drunk during the entire day a considerable one. Many persons who would be extremely indignant if called "tipplers," habitually take five or six glasses of beer a day, equivalent to 150 grm. of starch, or, in other words, one half the total amount of starch required by the system. Even this quantity is frequently exceeded. Of course, the three or four per cent. of alcohol which the beer contains also promotes the deposition of fat. Alcohol is readily oxidizable, and it thus shields to a considerable extent the fat already present in the body; and it also, in all probability, works such an injury to the tissues as to diminish their power of effecting decomposition. We must also remember that most beer drinkers take little exercise. The long sitting at the beer table, and the mental and physical sluggishness which immoderate indulgence in beer always causes, and finally the increasing corpulence itself, make it clear why most beer drinkers dislike protracted exercise.

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[*] This statement is founded upon an important discovery of Rubner, that, as far as the storing up of fat is concerned, the measure of value for different foods is the amount of heat given off by them when they undergo oxidation into carbonic-dioxide gas and water. Measured in this way, 100 grm. fat = 211 grm. albumen = 232 grm. starch = 234 grm. cane sugar = 256 grm. grape sugar.

[†] Probably the amount of fat contained in the food is often less than the above, and the amounts of albumen and starch somewhat larger. Voit estimates the diet of a well-to-do person at 127 grm. albumen, 89 grm. fat, and 362 grm. starch; and that for a vigorous laborer at 118 grm. albumen, 56 grm. fat, and 500 grm. starch. Of course, these figures are merely approximate.

We have thus seen that in by far the larger number of cases obesity is mainly due to the ingestion of too much food. No weight need be attached to the usual statement of corpulent persons, that they eat no more than others. Few of them have any idea how much nourishment they do consume. Others, having already become corpulent, eat less, to be sure, than they used to, but nevertheless an amount sufficient to maintain the acquired weight.

Other factors may no doubt exert an influence upon the increase of adipose tissue by diminishing the consumption of fat in the system. The most important factor of this class is physical inactivity. Muscular contractions lead to the decomposition of a large amount of fat. This explains why people of sedentary habits, and those who sleep long and exercise little, are more apt to become corpulent than are manual laborers. Again, there are certain diseases which seem to promote corpulence. In anæmia there is sometimes a striking tendency to obesity. in part due to the diminished supply of oxygen and in part to diminished muscular activity. This same inactivity is probably the main cause of corpulence in paralysis (hemiplegia). It may be, however, that disturbances of the nervous system may directly affect metamorphosis. Thus, idiots and other subjects of congenital defects of the brain are liable to obesity. Disturbances of the circulatory system seem to favor the production of corpulence by impairing oxidation. This is seen in many young persons with cardiac disease, although it is not easy to exclude in this case the influence of still other factors, such as a sedentary life.

Finally, some cases of obesity seem to result from a constitutional and inborn predisposition. Young children sometimes suffer from obesity; and the condition seems in many cases to be hereditary. Many races and nations exhibit an especial tendency to corpulence—for example, the Jews. Age and sex have some importance in this regard: extreme obesity is seldom seen previously to the thirtieth year; and women appear to be somewhat more subject to the disease than men. The importance of a "tendency" to obesity should not be overrated. Upon careful investigation, we shall almost invariably find in the habits of the individual, as regards food and exercise, a satisfactory explanation of his obesity. Strictly speaking, the condition can not be regarded as a disease unless the habits as to diet and exercise fail to account for it.

Pathology.—After corpulence has passed a certain point the condition is evident at the first glance. The subcutaneous cellular tissue is one of the chief places in which the fat is deposited. Consequently, the panniculus adiposus soon attains considerable thickness. The countenance grows more round and plump; beneath the chin is formed a second prominence known as the "double chin"; the chest appears broadened; the waist enlarges; and, particularly in women, the breasts are changed to great shapeless masses, over which the skin is so tightly stretched as to present lineæ albicantes. The abdominal walls are greatly altered. The belly projects more and more until it becomes actually pendulous, and its lower surface touches the interior surface of the thighs. Intertrigo is apt to occur in the groins, below the breasts, and between the buttocks. The skin everywhere has a fatty feel, due to the increased secretion of the sebaceous glands. This hyperplasia of the fatty tissue in the panniculus adiposus is associated with a deposit of fat in many parts of the interior of the body, including the mesentery, mediastinum, pericardium, and the capsules of the kidneys. Some of these will be mentioned again further on.

Of course, the circumference and weight of the body become greatly increased. As an approximate measure, it may be stated that for men of middle height a weight exceeding 90 kilo. (200 pounds), and for women 75 kilo. (165 pounds), may be regarded as abnormal.* This increase in bulk is the first cause of subjective symptoms. An obese person has to exert a greater effort in making any motion than do other people, and as a necessary consequence he gets easily tired, and seeks as far as possible to avoid exertion. The increased demand upon the muscles explains the familiar fact that corpulent persons perspire so readily.

The graver symptoms of obesity, and properly the first pathological phenomena of the condition, relate to the respiration and the circulation. The patient begins to complain of shortness of breath, and is subject to marked dyspnœa upon running or climbing stairs. There may be, at the same time, cardiac disturbance, indicated by a rapid pulse, palpitation, intermission of the pulse, or other slight irregularities in cardiac action. All these symptoms grow gradually worse; and to them are added symptoms of cardiac failure and consequent passive congestion. There is a tendency to bronchitis and other catarrhal troubles. The appetite and digestion are affected, and œdema appears.

A careful analysis of all these symptoms shows that many causes combine to produce them, all having a common tendency to impede respiration, and, still more, circulation. One source of disturbance is the increased deposit of fat upon the framework of the body. It is probable that the excess of adipose tissue covering the thorax exerts a direct influence in obstructing the respiratory motion of the thoracic walls, and renders the respiration more superficial. In this way the return of venous blood to the heart and the pulmonary circulation are both impeded, because the negative pressure in the chest is less than normal. Likewise the diminished amount of bodily exercise affects the circulation unfavorably. Brauner has shown how numerous are the arrangements connected with the fasciæ for promoting the venous currents, by means of the negative pressure resulting from the movements of the body. Whether the fat deposited around the heart directly obstructs the cardiac movements is somewhat doubtful, though this view is held by many. The fatty infiltration of the myocardium is of more importance: the fat is deposited in the intermuscular connective tissue. This lesion, however, is not of very frequent occurrence, nor does it produce so grave results as do certain other cardiac changes, which are either secondary to the obesity or complications of it.

There is no doubt that, in almost all cases where the corpulence actually induces grave disturbance, the cardiac symptoms are of prime importance. These are due, as has just been indicated, in part to the increased amount of adipose tissue, and in still greater part to complications, most of which are excited by the same causes as is the obesity itself. The abundant adipose tissue may obstruct the circulation in the smaller blood-vessels and capillaries inclosed within it. And, furthermore, the excessive development of fatty tissue probably leads to the growth of new blood-vessels, and, as a consequence, to an increase in the total volume of the blood. This is one way in which the demands made upon the heart are rendered greater than normal, and explains why the corpulent frequently exhibit cardiac hypertrophy. Other influences are also at work to produce this same result: they are, in the first place, the same factor which occasions the obesity itself, namely, the ingestion of increased amounts of food and drink (see the chapter on cardiac hypertrophy), and, secondly, certain other lesions which are frequently associated with obesity and are referable to the same causes as it is. Chief among this latter class is general arterio-sclerosis. If this involve the coronary arteries, it may in turn occasion still further damage—for example, degenerative myocarditis (compare page 309.) Chronic interstitial nephritis is another not infrequent complication. This is in part referable to the same causes as is the obesity. Gout and diabetes are less frequent.

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* Even 150 kilo. (330 pounds) has been repeatedly observed.

We thus see that obesity is often merely one of many injurious results occasioned by an improper mode of life. It is, in a certain sense, the first danger-signal, warning the patient and his physician to avoid the graver disturbances which threaten. This is a point of great practical importance; for, when once we have a combination of obesity with cardiac hypertrophy, fatty infiltration of the heart, arterio-sclerosis, or interstitial nephritis, the various causes and effects act and react upon one another in a way most perilous to health, and even to life. There is no need of describing the grave disturbances which invariably develop at the close of the scene. They are the result of chronic cardiac insufficiency, and have been fully described under cardiac disease.

In every case of obesity the physician should examine the heart, lungs, vascular system, and kidneys, particularly if there be already subjective disturbance. The examination of the heart may present considerable difficulties, because the results of palpation and percussion are so obscured by the thick cushion of fat which covers the thorax. We can, however, have recourse to auscultation, and can feel the pulse. The pulse may be either rapid, or slow, or irregular. We need not mention any minute particulars as to the examination. It may be stated, however, that a hepatic enlargement is often found, but it is much less often the result of fatty infiltration than of simple hypertrophy or passive congestion.

We have thus seen that corpulence may sometimes be associated with grave and dangerous lesions; but, on the other hand, it should be stated that this unfortunate condition by no means invariably exists. Not infrequently the corpulence remains moderate, in which case it is not really dangerous, however inconvenient. This is true of a proportion of those cases which are due to the ingestion of a large amount of nourishment, associated with defective oxidation, and where there are no other unfavorable influences at work. The obesity of hard drinkers is almost always a more or less dangerous condition, while that seen in many elderly persons and in women is often comparatively free from peril. These latter individuals are, to be sure, discommoded by their great weight, can accomplish less than they used to, are easily put out of breath, and have a certain tendency to catarrhal inflammations and rheumatic difficulties; but they escape the severer lesions above enumerated. Even these apparently harmless conditions should not be disregarded by the physician, as he can never be absolutely certain that grave complications may not be developed eventually.

Treatment of Obesity.—To cause the disappearance of the accumulated fat, it is necessary to promote its oxidation in the system and to prevent the ingestion of new supplies of fat. To accomplish this purpose we possess only two means—first, a limitation of the ingestion of such kinds of food as may lead to the formation of fat in the system, and, secondly, stimulation of those factors which occasion the destruction of the fat already present. All the various methods of treating obesity, without exception, aim either to diminish the supply or to increase the destruction of fat.

The methods vary greatly. It must be borne in mind that the diminution of the adipose tissue must not involve injury to the body itself. The treatment should not weaken the constitution, but should invigorate the patient, or at least be innocuous.

It is of prime importance, in every method of treatment, that the total quantity of ingested food should be diminished. It is a mistake to forbid the patient some particular kind of food—for instance, the carbohydrates or the fats—with the idea that they alone do harm, or to allow him unlimited quantities of other kinds of food, in the belief that they are harmless. Any person can eat albuminoids, fat and starch at every meal, and yet not grow fat; while, on the other hand, too much of any one of these may lead to an increase of adipose tissue. The amount of food which a person can take without increasing the amount of adipose tissue varies with the individual. It depends upon the amount of material already present in the body, and upon the various demands made upon the system, as well as other factors. This renders it difficult to draw up a universal dietary for the obese. We can best judge of the value of any course of treatment by its results, and these are best measured by the weight and the subjective condition of the patient.

Of the various elements of food, the albuminoids should be diminished least of any, because it would be sure to work injury to the system if they were supplied in too small an amount. Of course, the albuminoids must not be eaten to such an extent that the fat into which they decompose remains intact in the body. An increase in the amount of nitrogenous tissue is, however, desirable, because this promotes the vigor of the muscles and the heart, and so leads to the oxidation of larger quantities of the non-nitrogenous tissues.

The amount of fat and starch must be much more limited. The fats and starches are more potent in increasing adipose tissue and in shielding from oxidation the fat already stored up in the body than is nitrogenous food. It would not be at all advisable to forbid the use of either one of these two constituents of a mixed diet, allowing the other alone to be eaten. A varied diet is extremely desirable, even for one who is corpulent; and we should exclude neither fat nor starch wholly from the dietary, but we should merely limit the amounts to be taken. As already indicated, the amount of albuminoid food remaining unchanged, a person can eat double the quantity of starchy food that he can of fat, without increasing his adipose tissue. It is therefore irrational to allow the corpulent fat in larger proportions than starchy foods. The diet which Ebstein has recently proposed for the treatment of obesity does prove successful, but the explanation of its success lies in the comparatively small amounts of meat and fat ingested. Precisely the same results would be attained if a corresponding amount of starch were substituted for all or a portion of the fat; and in practice it is desirable, at least in most cases, to allow the patient both starches and fats. Of course, the likings and experience of the individual should be considered in each separate case. The Banting treatment, introduced in 1864, enjoyed for a time a great reputation. Its inventor applied it first of all to his own case, and with success. It rests upon a rational basis, inasmuch as the albuminoids are allowed in abundance, and the ingestion of fat and starch is limited. It lays too much stress, however, upon the exclusion of fat as compared with starch.

The principles here expounded are direct corollaries to the laws which Voit has taught us in regard to the different kinds of food. The physician who bears them in mind can lay down his own rules for the diet of his patient. As already stated, it is impossible to give figures which will apply to every case. If we take as a basis the average diet for an adult—that is, about 125 grm. albumen, 80 grm. fat, and 350 grm. starch—we might say that most cases of obesity would be sure to undergo improvement upon a diet containing 125 grm. albumen (or possibly even more than this), 40 grm. fat, and 150 grm. starch. The amounts of fat and starch could be even more diminished, but it is usually best not to be too precipitate. A gradual diminution of, say, two or three pounds a week, extending over a long period without interruption, is to be preferred to the rapid treatment common at many health resorts. Of course, the loss of fat is greater at the commencement of treatment than later on, when the amount of adipose tissue has already approached more nearly to normal, and the diet must undergo a gradual and corresponding change. It is of particular importance to increase the amount of non-nitrogenous foods in the later stages of treatment, lest the albuminoid tissues of the body become wasted.

The following dietary may be taken as an illustration of what would be suitable for a patient in the beginning of treatment: For breakfast, a cup of coffee with milk, and about 75 grm. of bread. At noon, a plate of soup, 150 to 175 grm. lean meat or fish, lettuce, green vegetables, and about 25 grm. bread. For dessert, about 75 grm. boiled rice, or some simple pudding, or 100 grm. fruit. To quench the thirst, water, or half a pint of light wine. In the afternoon, a cup of coffee, and with it not more than 20 to 30 grm. bread. For supper, two eggs, or 100 to 120 grm. meat, with 30 grm. bread, a little fruit, lettuce, half a pint of wine, or one or two cups of tea, not much sweetened. Butter should be entirely proscribed at first; later on it may be used in small amounts.

Some approach to this bill of fare must be enforced not merely for a few weeks, but for months. It is absolutely necessary that the patient should be weighed every two or three weeks. If the weight diminishes slowly and gradually, without any subjective disturbance, we have the best proof that the diet is a suitable one. If the weight does not diminish, then the amount of ingesta must undergo further reduction. If more food can be taken without the weight increasing again, a larger amount may be unhesitatingly permitted, and indeed may even be advisable if the patient be languid. The increase should at first, however, be mainly in the amount of albuminous food, the amount of starches and fats not being much increased. The "cure" can not be regarded as complete until the weight has been brought down to that of the average individual of the given age and sex. This goal having been reached, greater freedom in diet is permissible.

The object of the treatment just suggested is exclusively the limitation of the production of fat. We may also promote the destruction of the fat already stored up in the system. A chief means to this end is muscular exercise, which undoubtedly increases the oxidation of the adipose tissues. Carried out in a proper manner, it is therefore a most valuable adjuvant in treatment. Oertel has recently pointed out that muscular exertion does good in still another way—namely, by promoting cardiac activity and inducing deep respiratory efforts. Thus the heart is strengthened and circulation promoted. Mountain-climbing is one of the best modes in which to take the desired exercise. We need hardly say that the increased muscular activity makes it possible for the patient to take an increased amount of food without injury.

Baths also promote oxidation, but they are far less potent than is muscular exercise. Cold baths, brine baths, or baths containing carbonic-acid gas, may be employed. One way in which they do good is by stimulating the nervous system.

Oertel regards it of great importance to diminish the amount of water in the system, a point which has until very lately received little attention. The diminution in the amount of fluids may ameliorate any circulatory disturbance (*vide supra*) and relieve venous congestion, and it undoubtedly has some value in the treatment of obesity. Oertel has shown that a simple diminution in the amount of fluids ingested, when there is no other change in the diet or mode of life, will effect a diminution of the adipose tissues. This result is probably due mainly to the diminished strain upon the heart and the consequent increase of oxidation; but the chief cause of the rapid loss of weight, which we actually see in the corpulent as a result of the deprivation of fluid, is to be found not in the loss of fat, but in the marked loss of water contained in the body. "Desiccation" may further be promoted by stimulating the perspiration by bodily exercise or by steam baths. This withdrawal of liquid from the system, however, is advisable only in cases where there is already incipient cardiac failure.

It is evident that numerous excellent methods are at our disposal for the treatment of obesity; but their application to any particular case should be the result of a careful consideration of the special circumstances presented. A very essential point is that the injunctions of the physician should not merely be made, but be carried out; and it is precisely here that the treatment of many cases suffers shipwreck. We may be baffled by the patient's lack of energy and persistency, or by the importunate demands which his profession or social position make upon him. Indeed, it is sometimes absolutely impossible to prosecute the treatment at home, in which case bathing and health resorts are to be urgently recommended. There alone can the patient muster up the resolution necessary for carrying out the desired changes in his mode of life. The incontestable success of treatment at Carlsbad, Marienbad, Kissingen, Tarasp, and similar resorts is doubtless only to a very small extent the result of their specific medicinal influence, but it is mainly due to a strict observance of the above-described diet and regimen. The internal use of mineral waters is not entirely without a beneficial effect. Their laxative qualities diminish the absorption of food from the intestinal canal. It should be said, however, that the patient is at the same time exposed to the danger of a waste of his nitrogenous tissues. This is why patients frequently complain of the debilitating effect of these mineral springs; to avoid which, it would be well to increase the amount of albuminoids in the diet. We must also consider the need of guarding against too great a supply of fluid, with regard to what was said above as to the occasional service of "desiccation" of the body.

CHAPTER XIII.

SCROFULA.

Definition and Symptoms of what is called Scrofula.—We desire to present, at the close of this section, a brief description of scrofula, but merely from a practical standpoint. From a scientific point of view scrofula is not to be regarded as any special variety of disease. The term is applied to a group of symptoms seen most frequently in childhood, the essential features of which consist in the appearance of chronic enlargements of the lymph-glands, and in certain diseases of the skin and mucous membranes. The simultaneous appearance of these various phenomena does really produce a somewhat characteristic picture, which can frequently be recognized at the first glance.

Most scrofulous children appear pale, with a flabby skin and soft muscles.

The panniculus adiposus may nevertheless be tolerably well developed. Not infrequently the face is puffy, with prominent lips. This is called the "torpid habitus." In other cases the child has small features and a remarkably delicate white skin, which but partly conceals the superficial veins, and is readily suffused with blushes. To these the name "erethitic habitus" is applied. Enlarged lymph-glands are to be felt in the throat, at the angles of the lower jaw, and in the back of the neck, and occasionally in other parts of the body. These glands may remain indolent for a long while, or they may suppurate and break externally. Chronic cutaneous eruptions are often seen in various places. The most common of these is a scaly or impetiginous eczema, affecting the face, scalp, or extremities. More severe affections are lupus ("*lupus scrophulosorum*"), prurigo, and lichen scrophulosorum.

Of the mucous membranes, the conjunctiva and the lining membrane of the nostrils are most frequently affected. Conjunctivitis in various forms is a characteristic symptom of scrofula; as are also blepharitis ciliaris, keratitis, and chronic rhinitis, which last often terminates in a pronounced ozæna (*q. v.*). Chronic diseases of the ear are also frequent, such as otitis media, with perforation of the tympanum, and occasionally caries of the mastoid cells and its unfortunate results.

Of the deeper lying tissues, mainly the bones and joints suffer. The affections located here are almost exclusively "fungous"—namely, fungous ostitis and periostitis, white swelling, and caries. Formerly there was frequent use of such terms as "scrofulous inflammation of the knee-joint," or "scrofulous caries of the ribs."

If we inquire into the nature of this strange group of symptoms thus briefly enumerated, we shall find that by far the greater number of cases of well-marked scrofula are examples of tuberculosis. Tubercular bacilli have been demonstrated in connection with most of the fungous or "scrofulous" diseases of the bones and joints. Ozæna is often a tubercular disease of the nose, lupus is a tuberculosis of the skin, and many forms of otorrhœa are really tuberculosis of the ear. The ætiology of "scrofula" is therefore in main part identical with that of tuberculosis (*q. v.*), and this explains why the old physicians habitually insisted upon the intimate relationship between the two diseases. It was formerly thought that scrofula often terminated in tuberculosis—that is, a scrofulous child is apt to suffer eventually from tuberculosis of the lungs, intestine, or brain. To-day we know that most scrofulous children do not become, but that they are already, tuberculous.

It must, however, be borne in mind that in practice many diseases are termed scrofulous which have nothing to do with tuberculosis. Many cases of perfectly innocent eczema of the face and scalp lead to swelling of the glands in the throat, and are therefore termed scrofulous eczema. These cases are probably most of them secondary, and the result of external irritation and the like. Again, many enlarged glands in the neck are the result of pharyngeal trouble, as after scarlet fever, and are equally devoid of a tubercular taint. Pseudo-leukæmic lymphomata may also occur in children; and it should be borne in mind that hereditary or acquired syphilis may produce in children lesions closely resembling those of scrofula.

It is therefore the duty of the physician in every case of "scrofula" to analyze the ætiology and symptoms carefully, in order to determine with what he has to deal. "Scrofula" should be regarded merely as a short way of naming a certain group of symptoms. It is convenient to retain the term as being less likely to startle the friends of the child than would the true name of the disease.

Treatment.—In the treatment of scrofula we have first to attack the various local diseases, and, second, to invigorate the general health. We can not here enter into all the details of local treatment, but we must refer the reader to the special descriptions already given of the various local affections. We may, however, briefly mention a few facts with regard to the treatment of scrofulous swelling of the lymph-glands. Painting the overlying skin with tincture of iodine is a very common practice, but it seldom does much good. We have obtained more satisfactory results from iodoform collodium, or iodoform salve, or from the repeated inunction of sapo viridis. For particulars as to the opening of abscesses, or the extirpation of glands, we must refer to works on surgery.

Second, in the general treatment of scrofula every possible means is to be employed to invigorate the system. Abundant nourishment and fresh air are essential. The child may be taken either to the country, or the mountains, or the seashore. Cod-liver oil is regarded by some as a specific in scrofula; but its undoubted value really lies in the fact that it is an easily digested fat. Some children can take a considerably larger dose than others without its disturbing the stomach. Usually we prescribe two or three tablespoonfuls per diem. Salt baths enjoy a great reputation as a remedy in scrofula. If circumstances permit, the best way is to visit some place where there are brine baths, such as Kösen, Sulza, Salzungen, Arnstadt, Kreuznach, Münster am Stein, Rehme, Reichenhall, Ischl, and Colberg.

Treatment at these resorts is preferable to artificial baths at home, because it is under more favorable hygienic surroundings.

The chief internal remedies are iron, iodine, and arsenic. A favorite prescription is syrup of the iodide of iron. Arsenic promotes nutrition, and perhaps exerts some specific influence upon certain scrofulous (tubercular) local diseases, particularly the "fungous" affections and lupus.

APPENDIX I.

SUMMARY OF THE SYMPTOMS AND TREATMENT IN CASES OF POISONING.

1. **Sulphuric Acid.**—Mucous membrane of mouth, throat, œsophagus, and stomach deeply corroded. In the worst cases rapid death ushered in by convulsions and asphyxia, or more rarely consequent upon perforation of the stomach. Usually the case is more protracted. The mouth and throat are whitened, or in severe cases blackened. They are soon attacked by an intense ulcerative inflammation. Deglutition is extremely painful, and there are most distressing choking and retching. The vomitus contains black lumps. Profuse salivation. Pain along the œsophagus. Abdomen usually distended and very tender on pressure. There may be intestinal discharges of a bloody character, resembling dysentery. Urine is generally scanty, and often contains albumen and blood. Collapse. Small and rapid pulse.

In mild cases, slow recovery, the necrosed tissues gradually sloughing off. Cicatricial stricture of the œsophagus frequently ensues and may prove fatal. Neuralgia, hyperæsthesia, and various other nervous disturbances may also occur as sequelæ.

In fatal cases, the autopsy discloses necrosis, ulceration, and inflammation in the upper portion of the digestive tract. The lining of the stomach is usually coal-black. Well-marked parenchymatous degeneration of the liver and kidneys. Perhaps nephritis. In later stages, extensive cicatrices.

Treatment: If used at all, the stomach-pump must be introduced very cautiously, for fear of causing perforation. The best remedy to give at once is several teaspoonfuls of magnesia in water, or a few drops of liquor sodæ in mucilage. Later, the symptoms are to be combated with bits of ice, disinfecting mouth-washes and gargles, tonics, and cautious feeding with milk, eggs, etc. If stricture of the œsophagus develops afterward, an endeavor should be made to dilate it with bougies.

2. **Hydrochloric and Nitric Acids.**—Symptoms similar to those of sulphuric acid. The most prominent symptoms are the local ones, intense stomatitis, pharyngitis, etc. There is usually albuminuria, with casts and blood in the urine. Nitric acid sometimes stains the corroded spots, especially the angles of the mouth, yellowish; and the vomitus may have the same tinge (xanthoproteic acid). In poisoning from fuming nitric acid the inhaled vapors cause pulmonary symptoms. Prognosis and treatment as in case of sulphuric acid.

3. **Nitrous and Sulphurous Acid Fumes.**—Intense local inflammation of the air-passages. Violent dyspnœa, cough, abundant bloody or yellowish expectoration. There may also be grave nervous disturbance and collapse. *Treatment:* Symptomatic: sinapisms, narcotics, expectorants, and inhalations.

4. **Oxalic Acid.**—Local corrosive action similar to that of the other acids, only

less severe. In severe cases collapse from cardiac paralysis. Apt to occasion certain nervous symptoms—formication, anæsthesia of the finger-tips, tonic and clonic convulsions, trismus, and tetanus; later paresis. There is sometimes sugar in the urine, and nephritis may develop. Anuria is frequent, and is due to plugging of the tubules by calcic oxalate crystals. The treatment should be symptomatic, and should also include the administration of preparations of calcium —liquor calcis, calcii carbonas, or even egg-shells—to form insoluble calcic oxalate. Magnesia is also useful.

5. **Ammonia.**—The fumes affect the air-passages chiefly; the solution, the upper part of the digestive tract. The specific local effect is the production of an intense croupous inflammation of the mucous membrane. Accordingly, the symptoms are salivation, dysphagia, vomiting of strongly alkaline matter, and diarrhœa, or cough, dyspnœa, etc. In severe cases there is collapse, with rapid pulse, and such nervous symptoms as pain, paræsthesia, vertigo, convulsions, etc. *Treatment:* In fresh cases the stomach-pump. The cautious use of acids—for instance, acetic or citric. Also, symptomatically, emulsions of oil, bits of ice, and narcotics.

6. **Caustic Potash or Soda.**—Symptoms and treatment as in case of ammonia. In contrast to acid poisoning, it is to be noted that alkalies do not withdraw water and precipitate albumen, but dissolve it. The corroded spots are therefore not dry and brittle, but softened ("colliquation").

7. **Potassic Nitrate.**—Vomiting and diarrhœa. Severe abdominal pain. Collapse, with cold skin and rapid, thready pulse. Occasionally the pulse is slow. Nervous disturbances, such as painful muscular contractions, and, in severe cases, convulsions and coma. *Treatment:* Symptomatic; opium and other narcotics, stimulants (camphor, ether), and bits of ice.

8. **Chlorine Gas.**—Violent convulsive cough. Bloody expectoration. Spasm of the glottis. Dyspnœa. Darting pains through the chest. Sneezing and profuse flow of tears. In severe cases pneumonia. *Treatment:* Fresh air. Inhalation of warm aqueous vapor, or of ammonia to form ammonic chloride. Chloroform may also be tried, and narcotics.

9. **Iodine.**—1. *Acute iodism,* as seen, for instance, after the injection of large amounts of tincture of iodine into ovarian cysts: collapse, with pallor and cyanosis, and small and very rapid pulse. Vomiting. Often marked dyspnœa. Suppression of urine. Sometimes hæmoglobinuria. Later, the skin becomes very red. There is albuminuria; also sore throat, coryza, conjunctivitis, severe frontal headache, and cutaneous eruptions. 2. *Chronic iodism,* caused, for example, by long-continued internal administration of potassic iodide: coryza, conjunctivitis, sore throat. Gastric symptoms. Vertigo, headache, and similar nervous phenomena of a mild character. Acne or erythema, sometimes in the form of erythema nodosum. *Treatment:* In acute cases, white of egg and stimulants. Other than this, treatment must be symptomatic. Prophylaxis demands caution in the internal administration of iodine or its compounds. According to Ehrlich, the symptoms of iodism often disappear rapidly after the exhibition of about a drachm and a half (grm. 6) of sulpho-nitro-salicylic acid (sulphanilic acid).

10. **Bromine.**—1. *Acute poisoning* from the fumes of bromine excites the same symptoms as does chlorine gas. 2. *Bromism,* resulting from the long-continued use of potassic bromide, causes languor, debility, mental apathy, and impaired intellectual vigor. The reflexes are diminished, particularly the reflex irritability of the soft palate and pharynx. Anorexia. Diarrhœa. Impotence. Almost invariably a characteristic acne, the onset of which may be delayed by giving arsenic at the same time.

11. **Lead.**—(*a*) *Acute lead poisoning* produces severe gastro-enteritis. The best antidote is sulphate of sodium or magnesium; or phosphates, white of egg,

and milk. In fresh cases the stomach-pump, or emetics and purgatives. Other than this symptomatic treatment.

(*b*) *Chronic lead poisoning:* Seen in type-setters, type-founders, painters, potters, and others. General symptoms: Lead line on the gums, lead anæmia, and lead cachexia. Important groups of symptoms are: 1. *Lead colic:* Violent colicky pains, radiating from the umbilicus. Usually constipation, exceptionally diarrhœa. Abdomen concave and hard. Vomiting. Hard, slow pulse. Temperature usually normal. Urine sometimes contains a trace of albumen. Duration, one or two weeks. *Treatment:* If severe pain, opium and hot compresses. Atropine may also be tried. For constipation, enemata and gentle laxatives. Warm baths. 2. *Lead paralysis* (*vide* page 569). Paralysis of the laryngeal muscles due to lead poisoning has been reported. 3. *Saturnine encephalopathy:* Sudden development of grave cerebral symptoms; convulsions, coma, delirium, great mental uneasiness, and excruciating headache. Saturnine amaurosis. In severe cases, death. Cerebral lesions are very rarely found post mortem. *Treatment* is symptomatic. Lukewarm baths, with douching, narcotics, and stimulants. Later, potassic iodide. 4. *Lead arthralgia:* Most frequently attacks the knee. Also seen in the joints of the upper extremities. Sometimes associated with painful muscular contractions. Objective lesions are rarely seen. The treatment consists in warm baths and the administration of potassic iodide. It should be remembered that chronic lead poisoning may occasion gout and chronic interstitial nephritis. The reader is referred to the chapters describing these diseases.

12. **Copper** (*Blue Vitriol, Verdigris*).—(*a*) *Acute copper poisoning:* Copper taste. Vomiting of greenish matter, colic, tenesmus, and bloody stools. Also nervous symptoms, headache, vertigo, anæsthesia, paralysis, delirium. Collapse. Dyspnœa. *Treatment:* White of egg, milk, wood charcoal. Magnesia is also valuable. Yellow potassic ferrocyanide, milk sugar. (*b*) *Chronic copper poisoning* is rare. It occasions gastro-intestinal disturbance, colic, and a reddish or greenish discoloration of the hair.

13. **Zinc** (*Zinc Sulphate and Chloride*).—In acute poisoning, symptoms of severe gastro-enteritis, especially vomiting. Also albuminuria. *Treatment:* Albumen, tannin, and alkaline carbonates. *Chronic poisoning* (zinc fumes!): Fever, distress, vertigo, vomiting, metallic taste. General anæmia and emaciation.

14. **Mercury.**—(*a*) *Acute poisoning from corrosive sublimate:* The mucous membrane of the mouth, throat, œsophagus, stomach, and intestines is deeply corroded. Metallic taste. Vomiting. Diarrhœa with painful tenesmus. Ischuria or complete anuria. Collapse. Generally quickly fatal. *Treatment:* Milk, white of egg, reduced iron, narcotics. (*b*) *Chronic mercurial poisoning:* Seen in the makers of thermometers, scientific instruments, and mirrors. Rarely occasioned by the prolonged use of antisyphilitic remedies. The symptoms are anæmia, emaciation, with great muscular weakness, and gastro-intestinal disturbance. In the therapeutic use of mercury, besides the rarer mercurial enteritis, mercurial stomatitis is the most important toxic symptom; onset of foul-smelling necrosis of mucous membrane, with ulceration of mucous membrane of cheeks, jaws, gums, etc. *Treatment:* Immediate discontinuance of medication; potassic chlorate as mouth-wash and gargle.

Mercurial Tremor.—Tremor is common in chronic mercurial poisoning and is of special interest. It is usually preceded by a stage of pronounced mental irritability ("mercurial erethism"), and often some mental excitement, fright, etc., is the exciting cause of the tremor. The tremor itself is a pronounced intention tremor—that is, it is usually not present when the body is kept quiet, but it comes on with all movements, like the tremor in multiple sclerosis. The finer the movements the patient tries to make the more marked is the tremor. Mental excitement usually increases the tremor considerably. In severe cases the tremor is so great that the patients can not leave their beds. In rare cases there is also paralysis.

Treatment: Dietetic and hygienic. Iodide of potassium, hyoscine, and sometimes galvanism, are also useful.

15. **Phosphorus.**—(1) *Acute phosphorus poisoning,* as from matches: Violent epigastric pain, vomiting—the vomitus smells of phosphorus, and it may be phosphorescent. After these initial symptoms usually comes a period of comparative comfort, lasting two or three days. Then appear grave symptoms: jaundice, severe pain in the hepatic region and whole abdomen, hepatic enlargement, fever, feeble and rapid pulse, sometimes gastric or intestinal hæmorrhage, cutaneous ecchymoses, hæmaturia, epistaxis, or metrorrhagia. The intellect usually remains clear, except that just before death there may be drowsiness or convulsions. The urine contains albumen, blood, casts, bile-pigment, and sometimes leucine and tyrosine. No urea is excreted. Death occurs usually at the end of one or two weeks, but it may be more speedy. In mild cases the above symptoms are not strongly marked, and recovery may ensue. A very grave prognosis should be made in every case at the start. *Post-mortem appearances in acute phosphorus poisoning:* Jaundice. Dark blood. Numerous internal ecchymoses—for example, in the serous and mucous membranes and kidneys. Fatty degeneration of most of the internal organs, including the liver, heart, muscles, and kidneys. *Treatment:* In fresh cases, washing out of the stomach. Laxatives. As an emetic, sulphate of copper. The best antidote is old oil of turpentine (30–40 drops in mucilage). Oily substances should be avoided, as phosphorus is soluble in oil. Narcotics and other symptomatic remedies may also be indicated. (2) *Chronic phosphorus poisoning:* Necrosis of the lower jaw, less often of the upper jaw, extending from caries of the teeth. Necrosis of the bone, with exuberant growth of osteophytes.

16. **Arsenic** (Arsenious Acid, Schweinfurth Green, Scheele's Green, Arsenical Wall-paper).—(1) *Acute arsenic poisoning:* Symptoms of intense gastro-enteritis, suggesting cholera. Violent vomiting. Rice-water stools. Severe abdominal pain. Nervous symptoms: Vertigo, headache, faintness, twitchings. Cardiac weakness. Cyanosis. Collapse. Not infrequently a cutaneous eruption resembling urticaria or eczema. Sometimes albumen and blood are present in the urine. Severe cases are fatal in one or two days. With regard to arsenical paralysis, *vide* page 571. *Treatment:* At first the stomach-pump or emetics—for example, sulphate of zinc. The best antidotes are freshly prepared ferric hydrate in water, two to four tablespoonfuls every fifteen to thirty minutes; ferrum oxydatum saccharatum solubile (P. G.), in teaspoonful doses; magnesia; and, best of all, the compound of magnesia and ferric hydrate known as "*antidotum arsenici*" (P. G.), of which a tablespoonful may be given every fifteen to thirty minutes. (2) *Chronic arsenic poisoning:* Acquired in arsenic works and glass factories, or from arsenical fabrics, papers, and flowers. Conjunctivitis, chronic gastro-intestinal catarrh, eczema, and cutaneous ulcerations. Anæmia and cachexia, falling out of the hair, loss of sleep. Treatment is purely symptomatic, except as regards prophylaxis. In poisoning with arseniuretted hydrogen, hæmoglobinuria and jaundice occur, with severe nervous symptoms.

17. **Alcohol.**—(1.) *Acute alcoholic poisoning:* Unconsciousness; anæsthesia; pupils either dilated or contracted, usually not reacting to light; pulse small, sometimes slow; skin cold and clammy; vomiting; stertorous respiration. There may be delirium and clonic convulsions instead of coma. Such cases last three or

four days. Death has been repeatedly observed. *Treatment:* Bathing and douching. Stimulants.

(2.) *Chronic alcoholism.* (*a*) *Physical and mental debility:* Chronic catarrh of the throat, larynx, stomach, and intestines. Alcoholic tremor. Numerous organic diseases, including cirrhosis of the liver, contracted kidney, cerebral disease, and neuritis.

(*b*) *Delirium tremens:* Usually a sudden onset, as in connection with some acute disease or after a surgical injury. Disordered intellect. Great restlessness, hallucinations (vermin, etc.), excitement, and loss of sleep. *Treatment:* Bathing and douching. Injections of strychnine. The use of chloral and other narcotics should be cautious. Physical restraint should be avoided if possible. The patient may often be allowed to go about the room as he likes, if only he be watched. Alcohol should be given if collapse be threatened.

18. **Chloroform.**—Unconsciousness. Loss of sensibility and reflexes. Slow pulse. Pupils dilated. Failure of respiration, and finally of the heart. Danger of heart failure, especially in persons with weak hearts. *Treatment:* Artificial respiration. Injections of strychnine. Stimulants. Counterirritation.

19. **Iodoform.**—(Repeatedly seen from the use of iodoform on wounds). First of all, nervous symptoms: headache, vertigo, sleeplessness. Peculiar psychoses, maniacal attacks, delusions of persecution, refusal of food. In severe cases convulsions, deep coma. Very small, rapid pulse. *Treatment:* symptomatic, by stimulants, baths, etc. Alkalies and atropine are recommended, but their action is doubtful.

20. **Carbonic-oxide Gas** (Illuminating Gas).—At first, vertigo, headache, throbbing in the temples, ringing in the ears, and spots before the eyes. The patient gradually becomes unconscious. Skin pale and cyanotic. Respiration intermittent. Subnormal temperature. The urine may contain albumen and sugar. The carbonic oxide may be demonstrated in the blood by means of the spectroscope. Its color is clear cherry-red (CO-hæmoglobine). Subsequently paralysis, disturbances of sensation and of speech. *Treatment:* Fresh air, artificial respiration, stimulants, transfusion.

21. **Sulphuretted Hydrogen.**—Headache, vomiting, diarrhœa. In severe cases, unconsciousness, dyspnœa, cyanosis, convulsions, and death. The blood is thin, fluid, and black (sulph-hæmoglobine). *Treatment:* Artificial respiration, fresh air, and the cautious inhalation of chlorine gas.

22. **Bisulphide of Carbon.**—(Workers in rubber factories). Vomiting. Severe nervous symptoms. Incontinence of urine, atrophic paralyses, anæsthesia, mental disturbances, especially loss of memory, spasmodic conditions, etc. The red blood-corpuscles are destroyed; the black blood contains many flakes of pigment. *Treatment:* symptomatic.

23. **Hydrocyanic Acid** (Potassic Cyanide; Bitter Almonds).—Characteristic odor of bitter almonds. In severe cases death may occur in a few minutes. If the course be more protracted, convulsive and extremely slow respiration, the expiratory act being much prolonged; the eyeballs protrude, and the pupils are somewhat enlarged and do not react to light. Cardiac weakness, cyanosis, unconsciousness. Twitching of the muscles. Trismus. *Treatment:* Merely symptomatic. Emetics, artificial respiration, cool douches, stimulants. Atropine may be tried; also, hydrated ferric oxide and chlorine water.

24. **Nitrobenzine** (Nitrobenzole, Oil of Mirbane).—Strong odor of bitter almonds. At first, dizziness. The skin soon assumes a bluish hue, rapidly increasing to the deepest cyanosis. Increasing anxiety, sense of suffocation, and gradual loss of consciousness. In severe cases, death, ushered in by convulsions. In milder cases, gradual recovery. *Treatment:* Stomach-pump. Artificial respira-

tion. Stimulants. In the Leipsic clinique, transfusion has worked admirably in two cases. The symptoms caused by aniline and the aniline dyes closely resemble poisoning from nitrobenzole.

25. **Carbolic Acid.**—Corrosion of mouth, throat, and stomach. In mild cases, vertigo and headache; in severe cases, coma, preceded in rare instances by symptoms of cerebral irritation. Contracted pupils. Vomiting. Pulse slow at first, then rapid. The urine has a dark, olive-green color. Sometimes hæmoglobinuria and nephritis. *Treatment:* Stomach-pump. Slaked lime and water. Large doses of sulphate of sodium are especially to be recommended.

26. **Atropine** (*Belladonna*).—Dryness of the mouth and throat. Excessive thirst. Dizziness and headache. Peculiar mental disturbances: hallucinations are particularly frequent. Pupils very widely dilated. Cutaneous erythema resembling that of scarlet fever. In severe cases, pulse enormously accelerated, with violent pulsation in the arteries. Convulsions may occur. Nervous symptoms persist for some time. *Treatment:* The following physiological antidotes should be tried: physostigmine (eserine), pilocarpine, and morphine.

27. **Digitalis.**—Vomiting. Diarrhœa. Pulse very slow (forty beats per minute, or less). Dyspnœa. Symptoms of collapse. Cold extremities, muscular tremor. Somnolence. In the worst cases, sopor and death. Even the milder cases are protracted. *Treatment:* Emetics, stomach-pump. Tannin. Camphor, strong *café noir*, ether, ammonia. Counter-irritation.

28. **Nicotine** (Smoking; Tobacco Juice; Tobacco Enemata).—1. *Acute:* Pulse small and slow, syncope, sense of oppression, salivation, vomiting. In severe cases, loss of consciousness, tetanic spasms, both pulse and respiration intermittent. 2. *Chronic* (from excessive use of tobacco): Palpitation, irregular action of the heart, paroxysms of asthma and angina pectoris. Tremor, muscular weakness. Loss of sleep. Sometimes there are symptoms of ataxia ("nicotine tabes" seen in cigar makers). Amblyopia, scintillating scotoma. Gastric disturbance, chronic catarrh of the pharynx and larynx. *Treatment:* In acute cases, stimulants. Chronic poisoning necessitates the giving up of tobacco. Further treatment is symptomatic.

29. **Strychnine.**—Violent tetanic reflex convulsions. Exaggeration of the cutaneous and tendon reflexes. Trismus. Opisthotonos. Pulse small and very rapid. The convulsions come in paroxysms, with intervals between them. The intellect is usually perfectly clear. Recovery occurs only in mild cases. *Treatment:* Emetics, stomach-pump. Tannin. Tincture of iodine. Castor-oil. The convulsions are to be combated by morphine, chloroform, chloral, or potassic bromide. Curare has also been tried.

30. **Coniine** (Hemlock).—In severe cases convulsions, then general paralysis, especially of the respiratory muscles, loss of consciousness, and death. Pupils dilated. In milder cases confusion, muscular weakness, vomiting, and diarrhœa. *Treatment:* Emetics, tannin, stimulants.

31. **Morphine** (Opium).—1. *Acute:* Begins with fatigue, headache, darkening of the visual field, nausea, vomiting. Then coma comes on with slow, stertorous, sometimes irregular respiration. Muscles completely lax. Pupils usually very small. Pulse often slow, but in other cases rapid and small. Toward the end of life Cheyne-Stokes's respiration. In milder cases only vomiting, stupor, headache, etc. *Treatment:* Sulphate of zinc, or some other emetic; stomach-pump. Tannin. *Café noir.* Atropine may be tried as a physiological antidote. Stimulants (camphor, ether) are best, also cold baths with shower-baths; artificial respiration. 2. *Chronic* (morphine habit): Emaciation, anæmia, headache, vertigo, wakefulness. Tremor. Mental disturbance. Unconquerable longing for morphine; and, if this be denied, the appearance of grave symptoms. To break up the morphine habit is almost impossible except in hospitals and special asylums. The with-

drawal of the drug is abrupt according to the practice of some, and gradual according to others. For particulars consult monographs.

32. **Ergot** (Ergotine).—1. *Acute:* At first nausea, vomiting, colic, and diarrhœa. Then vertigo, headache, and muscular weakness. Pulse slow. In severe cases, sopor, disturbance of respiration, and sometimes death. *Treatment:* Emetics and purgatives. Tannin. Ether, camphor, and *café noir* as stimulants. 2. *Chronic ergotism:* Gastric symptoms, vertigo, languor, cardiac weakness. The nervous disturbances are, however, of especial importance. Of these, paræsthesia has long been recognized. Recently attention has been attracted to the great resemblance of the nervous symptoms to those of tabes dorsalis; and there is, moreover, an anatomical change in the posterior columns of the cord. Convulsions and psychical phenomena are also observed. A second form of chronic ergotism is called gangrenous ergotism. It results in dry gangrene of the hands and feet. A line of demarcation forms and the gangrenous parts slough off. The process may be attended by fever and pyæmia. The probable explanation is that the minute blood-vessels become spasmodically contracted and thrombi form under the influence of the poison. The different symptoms are due in part to the different constituents of the ergot. The best known are sphacelinic acid, which is probably the cause of gangrenous ergotism and ergotine tabes; cornutine, which causes severe convulsive symptoms (convulsive ergotism) and uterine contractions; and finally ergotinic acid. The *treatment* of chronic ergotism is purely symptomatic.

33. **Poisonous Mushrooms.**—1. *Poisoning from morels:* Fresh morels ("morcheln" or "lorcheln") contain a poison which is readily soluble in hot water, and which evaporates completely if the morels be dried. Morels that have been dried or parboiled are therefore perfectly harmless, but the fresh ones are poisonous. The symptoms are nausea, vomiting, diarrhœa, headache, coma, and, above all, hæmoglobinæmia and hæmoglobinuria (*q. v.*), associated with which is a hæmatogenous icterus. In severe cases death occurs, ushered in by convulsions. *Treatment* is symptomatic, and includes the administration of emetics, purgatives, and stimulants. 2. *Poisoning from the red agaric* (*amanita muscaria*); This contains the poisonous alkaloid muscarine. Gastric symptoms and diarrhœa. Mental excitement, delirium, tetanic and epileptiform convulsions. A rapid pulse, small pupils, disturbed vision from spasm of accommodation, sweating, salivation, and in most of the severe cases sopor and death. *Treatment*: Emetics, etc. Atropine, which acts as a physiological antidote to muscarine. Also tannine, and stimulants. 3. *Poisoning from tubercular mushrooms (amanita phalloides*), confused with young button mushrooms (champignons). Digestive disturbances, later jaundice, somnolence, coma. The autopsy shows fatty degeneration of the liver, kidneys, and stomach, as in phosphorus poisoning.

34. **Poisoning from Sausages** (*Botulismus*).—This sometimes occurs as the result of eating partially decayed sausages. The symptoms are pain in the stomach, nausea, vomiting, colic, and diarrhœa. There are also marked feebleness, præcordial anxiety, and dyspnœa; vertigo, headache, somnolence; and very often disturbance of vision (amblyopia, spots before the eyes), and, what is surprising, ptosis. In severe cases, dysphagia, as a result of more or less complete paralysis of the tongue and the constrictors of the pharynx. The mouth is dry. The heart is feeble; this and the general prostration and malnutrition may prove fatal. The cases are usually protracted, rarely being very acute. The active principles (alkaloids of putrefaction) have lately become known in part. In sausage and also in meat poisoning the most important substance is ptomato-atropine, a substance which acts almost exactly like atropine. *Treatment:* Emetics, purgatives (calomel), stimulants, and, if indicated, artificial feeding.

35. **Poisoning from Meat.**—In repeated instances severe symptoms have been occasioned by eating tainted meat, or possibly that obtained from animals which had been diseased. These symptoms are certainly due to the products of putrefaction, substances which act partly like muscarine, partly like atropine, neurine, methylguanidine, etc. The special poison is not yet fully known. Probably there are several poisons, either chemical or organic and infectious. The usual symptoms are vomiting and diarrhœa. The case may closely simulate cholera. In most instances certain nervous phenomena are also observed—wakefulness, delirium, headache, and changes in the pupils. There may be roseola or wheals or erythema. Frequently there is a high fever, but sometimes the temperature is subnormal. The pulse is small and slightly accelerated, although it may occasionally be slower than normal. There is a sense of thoracic oppression. The cases are often protracted. Tendency to relapses. Death may occur. Post mortem, there is usually found an intense and often hæmorrhagic enteritis with secondary changes in the spleen, kidneys, lungs, and other organs. *Treatment:* Symptomatic: calomel, emulsions, stimulants, and baths. Food should be given cautiously.

36. **Poisoning from Fish.**—The eating of tainted fish has likewise caused grave disturbance. The symptoms vary. Usually there are pain in the stomach, præcordial anxiety, vertigo, dryness of the throat, aphonia, and labored respiration. There may also be disturbance of vision, amblyopia, and colored vision, or paralysis of the motores oculi and of accommodation. In severe cases there may be dysphagia and general paresis. Sometimes dyspnœa and cardiac weakness are observed. The cases are frequently very tedious. Here, too, there are many active poisonous products of putrefaction, among them one which acts like muscarine. *Treatment* similar to that recommended in the two preceding paragraphs.

37. **Poisoning from Mussels** (*Mytilus Edulis*). This is also common. A drawing feeling in the neck, a blunt feeling in the teeth, crawling and burning in the arms and legs, dullness in the head, mental excitability, feeling as if everything were light, as if the patient must fly; in the later stages dilated, immobile pupils, difficult speech, paresis and ataxia of the muscles; also nausea, skin eruptions (urticaria), fall of temperature. In severe cases death may ensue in a few hours. At the autopsy we find severe enteritis, enlargement of the spleen, and often a peculiar speckling of the liver. The poison of mussels that is known has been called mytilotoxine.

38. **Poisoning from Cheese.**—Vomiting, colicky pains, diarrhœa, vertigo, sense of thoracic oppression, headache, languor, and disturbance of vision. *Treatment* as in paragraphs 35 and 36.

APPENDIX II.

TABLE OF WEIGHTS AND MEASURES.

Table of Relation of U. S. Fluid to Metric Measure.

Minims		Cubic Centimetres.	Minims.		Cubic Centimetres.	Fluid Drachms.		Cubic Centimetres.	Fluid Ounces.		Cubic Centimetres.
1	=	·06	30	=	1·85	4	=	14·79	4	=	118·24
2	=	·12	40	=	2·46	6	=	22·18	6	=	177·39
5	=	·31							8	=	236·53
10	=	·62	Fluid Drachms.		Cubic Centimetres.	Fluid Ounces.		Cubic Centimetres.	12	=	354·82
15	=	·92	1	=	3·70	1	=	29·57	16	=	473·11
16¼	=	1·00	2	=	7·39	2	=	59·10			
20	=	1·23	3	=	11·09	3	=	88·67			

Table of Relation of Troy Weight to Grammes.

Grains.		Grammes.	Grains.		Grammes.	Drachms.		Grammes.	Ounces.		Grammes.
1/8	=	·008	8	=	·52	1	=	3·89	1	=	31·1
1/6	=	·011	10	=	·65	1½	=	5·83	1½	=	46·6
1/4	=	·016	15	=	·97	2	=	7·77	2	=	62·2
1/2	=	·032	15·43	=	1·00	3	=	11·66	3	=	93·3
1	=	·065	20	=	1·29	4	=	15·55	4	=	124·4
2	=	·13	30	=	1·94	6	=	23·3	6	=	186·6
4	=	·26	40	=	2·59				8	=	248·8
5	=	·32	61·73	=	4·00						
6	=	·39									

The Metric System in Medicine.

Old Style.		Metric.	
♏ j or gr. j	=		06 grm.
f ʒ j or ʒ j	=	4	"
f ℥ j or ℥ j	=	32	"

The decimal *line*, instead of *points*, makes errors impossible. As ·06 (drug) is less than a grain, while 4· and 32· (vehicle) are more than the drachm and ounce, there is no danger of giving too large doses of strong drugs.

C.c. (cubic centimetres), used for grms. (grammes), causes an error of five per cent (excess).

A teaspoonful is usually 5 grms.; a tablespoonful, 20 grms.

INDEX.

ABASIA, 803 (foot-note).

Abdomen in typhoid fever, 10.

Abdominal typhus, 1.

Abducens nerve, paralysis of, 556.

Abortion in acute yellow atrophy of the liver, 488; in chorea, 784; in pernicious anæmia, 944; in typhoid fever, 18.

Abscesses, embolic, in lungs, 247; in appendicitis, 420; in glanders, 116; in parotitis, 349; in perinephritis, 868; in perityphlitis, 420; in smallpox, 56; in septico-pyæmia, 110; in typhoid fever, 17; of brain, 741; of liver, 476; of mediastinum, 273; of tonsils, 353; retropharyngeal, 360; sub-diaphragmatic, in peritonitis, 456.

Absorption, impaired, in chronic gastric catarrh, 375.

Accentuation of the aortic second sound in chronic nephritis, 859; in arterio-sclerosis, 333; of the pulmonic second sound in emphysema, 178; in mitral regurgitation, 285; in mitral stenosis, 287.

Accessorius nerve, spasm of, 574; position of head in spasm of, 574; paralysis of, 562.

Acetonæmia in diabetes, 969.

Acetone, odor of, in diabetes, 971; reaction, 969.

Achilles' tendon reflex, 545.

Acholia, 489.

Achromatopsia in hysteria, 801.

Acid dyspepsia, 382.

Acid, free, in gastric juice, tests for, 378.

Aconitine in neuralgia, 520; in trigeminal neuralgia, 523.

Acoustic nerve, atrophy of, in locomotor ataxia, 643.

Acromegaly, 589.

Actinomycosis, 274.

Acupuncture in aneurism, 338; in cardiac dropsy, 306.

Acute bulbar paralysis, 691.

Acute tuberculosis, 236.

Acute yellow atrophy of the liver, 485.

Addison's disease, 878.

Adenia, 951.

Adeno-carcinoma, 426.

Adenoid growths in pharynx, 358.

Adherent pericardium, 326.

Adrenals in Addison's disease, 879.

Ægophony, 260.

After-sensations, 509; in locomotor ataxia, 640.

Agaricine in pulmonary tuberculosis, 235.

Ageusia, 533.

Agraphia, 720.

Ague, 90.

Air, inspired, as carrier of infection, 209.

Alalia, 685.

Albumen in urine, tests for, 823.

Albuminous expectoration in pleurisy, 266.

Albuminuria, accidental, 824; in acute ascending spinal paralysis, 675; in acute Bright's disease, 841; in anæmia, 934, 942; in bulbar apoplexy, 692; in chronic Bright's disease, 852, 859; in diabetes, 970; in diphtheria, 70; in epilepsy, 773; in erysipelas, 64; in fevers, 837; in gout, 987; neurotic, 825; in osteomalacia, 927; physiological, 823; in pneumonia, 197; renal, genuine, 824; in scarlet fever, 41; in scurvy, 959; in smallpox, 56; spurious, 824; transitory, 825; in typhoid fever, 17; in yellow fever, 102.

Albuminuric retinitis, 854, 861.

Alcohol, effects of, on the digestive system, 372, 374; on the kidneys, 856; on the nervous system, 584; poisonous effects of, 1006.

Alcoholic beverages in anæmia, 936; in diabetes, 979; in gout, 991; in neurasthenia, 818; in obesity, 993; in pneumonia, 206; in tuberculosis, 232.

Alcoholic neuritis, 584.

Alcoholic poisoning, acute, 1006.

Alcoholism, 1006; acute, 1006; chronic, 1007; treatment of, 1006.

Alexia, 720.

Alimentary canal, tuberculosis of, 215, 423.

Alkalies in diabetes, 981; in gout, 991.

Allantiasis (botulismus), 1009.

Alopecia, 589.

Altitude in tuberculosis, 233.

Alum in intestinal catarrh of children, 417.

Amaurosis, anæmic, 932; in gastric ulcer, 387; hysterical, 802; uræmic, 832.

Amblyopia, 751; in hysteria, 802.

Ambulatory typhoid fever, 18.

Amimia, 720.

Ammoniæmia, 883, 890, 895.

Ammonia in asthma, 172; in diabetes, 981; poisoning from, 1004.

Amnesia, 717.

Amœba coli (amœba dysenteriæ), 81; in liver abscess, 81; in lungs, 81.

Amœbic dysentery, 81.

Amphoric breathing, 222, 269.

Amyl nitrite in asthma, 172; in epilepsy, 778; in hemicrania, 598; in trigeminal neuralgia, 523; in valvular disease, 307.

Amyloid disease, chronic nephritis combined with, 865; of kidney, 864; of liver, 498; in phthisis, 228; in syphilis, 865.

Amyotrophic lateral sclerosis, 650.

Anæmia in Addison's disease, 880; in anchylostomiasis, 448; in articular rheumatism, 906; fever in, 934 ; from gastric atrophy, 939 ; in gastric cancer, 391 ; in gastric ulcer, 387 ; idiopathic, 928 ; tuberculosis as a complication of, 226, 424 ; progressive pernicious, 938 ; primary or essential, 928 ; secondary, 929 ; toxic, 931.

Anæmia, pernicious, 938 ; associated with atrophy of gastro-intestinal walls, 939 ; chemical examination of blood in, 943 ; diagnosis, 944 ; post-mortem lesions in, 940 ; symptoms of, 941 ; treatment of, 944.

Anæmia, simple constitutional, 928 ; diagnosis of, 936 ; symptoms of, 931 ; treatment of, 936.

Anæmia, spastic, 511 ; splenic, 944.

Anæmic murmurs, 933.

Anæsthesia, 510 ; dolorosa, 512 ; in hemiplegia, 733 ; in hysteria, 800 ; in locomotor ataxia, 636 ; in Morvan's disease, 679 ; in myelitis, 619 ; in neuralgia, 517 ; in neuromata, 586 ; in railway spine, 820 ; in unilateral lesions of the spinal cord, 683 ; of the skin, 510 ; of the trigeminus, 513.

Analgesia, 508 ; in hysteria, 800 ; in locomotor ataxia, 636, 640 ; in Morvan's disease, 679 ; in syringo-myelia, 678.

Anarthria, 685 ; in bulbar apoplexy, 692.

Anchylostomum duodenale, 448.

Aneurism, 334 ; cylindrical, 334; dissecting, 340 ; fusiform, 334 ; sacciform, 334 ; of the abdominal aorta, 339 ; of carotid artery, 339 ; of the cerebral arteries, 725 ; of the heart, 308, 330 ; of subclavian artery, 339 ; of pulmonary artery, 218, 339.

Aneurism of thoracic aorta, 334 ; diagnosis of, 337 ; dyspnœa in, 336 ; hæmorrhage in, 337 ; pain in, 337 ; physical signs of, 335 ; symptoms of, 335 ; treatment of, 338 ; Tufnell's treatment of, 338.

Angina pectoris, 397, 309, 310, 318 ; pseudo or hysterical, 318.

Angina (see Sore Throat).

Angio-neurotic œdema, 588.

Animal lymph, 59.

Animals, lower, diphtheria in, 67.

Ankle clonus, 545 ; in cerebral hæmorrhage, 732 ; in hysterical paraplegia, 803 ; in myelitis, 620 ; in spastic paraplegia, 664 ; in tetanus, 792.

Anosmia, 532.

Anterior crural nerve, paralysis of, 568.

Anthracosis of lungs, 245.

Anthrax, 117 ; bacillus, 117 ; in animals, 118.

Antidotes in poisoning from atropine, 1008 ; lead, 1004 ; mushrooms, 1009 ; phosphorus, 1006.

Antidotum arsenici, 1006.

Antimony, arsenite of, in cardiac valvular disease, 303.

Antipyrine in typhoid fever, 24.

Antiseptic medication in typhoid fever, 20.

Antiseptics in pyelitis, 884.

Anuria in hydronephrosis, 892; in nephritis, 841.

Aorta, aneurism of, 334 ; hypoplasia of, in chlorosis, 929 ; rupture of, 339.

Aortic incompetency, 287 ; sudden death in, 290 ; symptoms of, 287.

Aortic orifice, congenital lesions of, 294.

Aortic stenosis, 290.

Apex of lungs, catarrh affecting, 222.

Apex pneumonia, 195, 202.

Aphasia, 716 ; amnesic, 717 ; anatomical localization of, 716 ; ataxic, 717 ; diagnosis of, 719 ; hemiplegia with, 736 ; in typhoid fever, 16.

Aphemia (see Aphasia), 716.

Aphonia, hysterical, 803 ; in acute laryngitis, 130 ; in adductor paralysis, 140 ; in pericardial effusion, 325.

Aphthæ, 343.

Aplasia of the lungs, 181.

Apoplectic cyst, 727 ; habit, 726 ; scar, 727.

Apoplexy, 728 ; delayed, 728 ; from cerebral syphilis, 758 ; from hæmatoma of the dura mater, 697 ; from hæmorrhage into medulla and pons, 691 ; from tumors of the brain, 750 ; ingravescent, 728 ; in multiple sclerosis, 630 ; premonitory symptoms of, 728 ; pulmonary, 247 ; spinal, 604.

Appendicitis, 420.

Appendix vermiformis, perforation of, in typhoid fever, 11 ; situation of, 420.

Apraxia, 720.

Apyrexia in relapsing fever, 32.

Arachnitis (see Meningitis).

Aran-Duchenne type of muscular atrophy, 653.

Arbutine in cystitis, 896.

Arch of aorta, aneurism of, 334.

Argyll-Robertson pupil, 642 ; in ataxia, 642 ; in progressive general paralysis, 763.

Arm, peripheral paralysis of, 564.

Arhythmia, 297.

Arsenical neuritis, 571.

Arsenical poisoning, 931, 1006 ; paralysis in, 571.

Arsenic in anæmia and chlorosis, 938 ; in asthma, 172 ; in cerebral tumor, 756 ; in chorea, 783 ; in diabetes, 981 ; in exophthalmic goitre, 598 ; in habitual headache, 531 ; in hysteria, 813 ; in leukæmia, 950 ; in locomotor ataxia, 648 ; in malaria, 97 ; in neuralgia, 519 ; in paralysis agitans, 787 ; in pernicious anæmia, 945 ; in pseudo-leukæmia, 953 ; in scrofula, 1001 ; in spasm of the muscles of the neck, 575 ; paralysis from, 571 ; poisoning from, 931, 1006.

Arteries, diseases of, 331 ; calcification of, 331.

Arterio-sclerosis, 331 ; in diabetes, 972 ; in obesity, 996 ; symptoms of, 332 ; treatment of, 334.

Arteritis, syphilitic, 757.

Arthralgia from lead, 1005.

Arthritis, acute rheumatic, 900 ; gonorrhœal, 901 ; in cerebro-spinal meningitis, 106 ; in dengue, 99 ; in diphtheria, 70 ; in dysentery, 79 ; in hæmophilia, 962 ; in purpura, 965 ; in scarlet fever, 43, 901 ; in tabes dorsalis, 644 ; multiple secondary, 901, 907 ; rheumatoid, 911 ; septic, 111, 907.

Arthritis deformans, 911 ; monarticular form of, 914 ; of the poor, 912 ; of the rich, 985 ; polyarticular form of, 914 ; sicca, 913.

Arthrogryposis, 576.

Arthropathies in tabes, 644.

Arthrospores, 82.

Asafœtida in hysteria, 813.

Ascaris lumbricoides, 445.

Ascites, 460 ; from cancerous peritonitis, 463 ; in cancer of the liver, 493 ; in cancer of the pancreas, 504 ; in children, 459 ; in chronic endocarditis, 299 ; in cirrhosis of the liver, 480 ; in scarlet fever, 42 ; in suppurative hepatitis, 477 ; in syphilis of the liver, 491 ; in thrombosis of the portal vein, 502 ; in tuberculous peritonitis, 459 ; physical signs of, 461 ; treatment of, 462.

Ascitic fluid, 461 ; hæmorrhagic, 461.

Aspect, facial, in paralysis agitans, 785.

Asphyxia, local, 588 ; death by, in phthisis, 217 ; in diphtheria, 68.

Aspiration in empyema, 264 ; in pericardial effusion, 328 ; in pleuritic effusion, 261.

Aspiration pneumonia, 185.

Associated movements, 541 ; in cerebral hæmorrhage, 735 ; in facial paralysis, 561 ; in infantile cerebral paralysis, 745 ; of the facial muscles in progressive paralysis, 762.

Astasia-abasia, 803.

Asthenopia, neurasthenic, 816.

Asthma, bronchial, 168 ; cardiac, 296 ; crystals, 169 ; ætiology of, 170 ; hay, 124 ; humid, 152 ; Millar's, 142 ; nasal affections in, 171 ; nervous, 168 ; origin of, 170 ; renal, 860 ; sputum in, 860 ; symptoms of, 168 ; symptomatic, 170 ; thymic, 142 ; treatment of, 172 ; uræmic, 860.

Atavism, in hæmophilia, 963 ; in gout, 985, 987.

Ataxia, 542 ; after smallpox, 56 ; cerebellar, 723 ; drunkard's, 584 ; hereditary, 648 ; in diphtheria, 70 ; in locomotor ataxia, 636 ; in myelitis, 619 ; in peripheral neuritis, 583 ; in progressive paralysis, 764 ; in typhoid fever, 16 ; locomotor, 632.

Ataxic gait, 637.

Ataxic paraplegia, 649.

Atelectasis, pulmonary, 181.

Atheroma of the arteries, 331.

Athetosis, 541, 787 ; congenital, 788 ; idiopathic, 788 ; in cerebral paralysis of children, 745, 788 ; movements of, 541 ; nature of, 789 ; symptomatic, 788.

Athrepsia, 414.

Atresia ani, 432.

Atrophy, 414 ; idiopathic muscular, 658 ; juvenile muscular, 658 ; of brain, diffuse, in general paresis, 765 ; of cardiac muscle in pericarditis, 323 ; of muscles, various forms of, 658 ; progressive muscular, of spinal origin, 653 ; unilateral, of face, 594.

Atropine in asthma, 172 ; in epilepsy, 778 ; in exophthalmic goitre, 598 ; in neuralgia, 520 ; in progressive bulbar paralysis, 690 ; in pulmonary tuberculosis, 235 ; poisoning from, 1008.

Attitude in paralysis agitans, 785 ; in pseudo-hypertrophic muscular paralysis, 660.

Auditory center, affections of, 721 ; nerve in facial palsy, 559 ; vertigo, 769.

Aura, forms of, in epilepsy, 771.

Automatism in petit mal, 773.

Bacilli of anthrax, 117 ; Asiatic cholera, 82 ; cerebro-spinal meningitis, 103 ; diphtheria, 66 ; glanders, 116 ; laryngeal tuberculosis, 136 ; pneumonia, 189 ; pulmonary tuberculosis, 208 ; tetanus, 791 ; typhoid fever, 1.

Bacteria in hepatitis suppurativa, 476 ; gangrene of lungs, 241 ; pleurisy, 253, 254, 263 ; septico-pyæmia, 109.

Balanitis in diabetes, 972.

Balsams in chronic bronchitis, 154 ; in pulmonary emphysema, 180.

Bandage for compression in chronic hydrocephalus, 769 ; for extension in pressure paralysis of the spinal cord, 614 ; for the legs in locomotor ataxia, 648 ; in anæsthesia of the trigeminus, 515 ; in neuralgia of the joints, 529.

Banting's treatment of obesity, 997.

Baræsthesiometer, 507.

Barrel-shaped chest in emphysema, 177.

Basedow's disease (see EXOPHTHALMIC GOITRE), 595.

Basilar artery, embolism and thrombosis of, 693.

Basilar meningitis, 702.

Baths in acute ascending spinal paralysis, 676 ; in acute poliomyelitis, 671 ; in anæsthesia of the trigeminus, 515 ; in bronchitis, 150, 155 ; in cerebral hæmorrhage, 737 ; in cerebral hyperæmia, 710 ; in cholera morbus, 413 ; in chorea, 783 ; in chronic spinal leptomeningitis, 601 ; in cutaneous anæsthesia, 515 ; in cystitis, 896 ; in diabetes, 980 ; in diphtheria, 73 ; in erysipelas, 64 ; in gout, 992 ; in habitual headache, 531 ; in hyperpyrexia of rheumatism, 911 ; in hysteria, 812 ; in multiple sclerosis, 631 ; in myelitis, 627 ; in nephritis, 847, 864 ; in nephrolithiasis, 888 ; in neuralgia, 521 ; in neurasthenia, 818 ; in neuritis, 584, 585 ; in obesity, 998 ; in osteomalacia, 927 ; in pachymeningitis cervicalis hypertrophica, 603 ; in paralysis agitans, 787 ; in paralysis of the upper limbs, 568 ; in pneumonia, 205 ; in progressive bulbar paralysis, 690 ; in progressive paralysis of the insane, 767 ; in purulent meningitis, 701 ; in pyelitis, 884 ; in rachitis, 924 ; in railway spine, 827 ; in rheumatism (acute articular), 910, 911 ; in rheumatism (muscular), 919 ; in scarlet fever, 45 ; in smallpox, 60 ; in spasms of the respiratory muscles, 577 ; in tetanus, 794 ; in tetany, 791 ; in typhoid fever, 23.

Baths, electric, in neurasthenia, 818.

Beaded ribs in rickets, 923.

Bedsores in myelitis, 622.

Beer-drinkers, heart disease in, 313.

Belladonna in asthma, 172 ; in diabetes, 981 ; in epilepsy, 778 ; in whooping-cough, 163 ; poisoning from, 1008.

Bell's palsy, 558.

Benzine in trichinosis, 124 ; in whooping-cough, 163.

Beri-beri, 582 ; in Japan, 582 ; in United States, 582.

Biermer's change of note in pneumothorax, 269.

" Big-jaw " in cattle, 274.

Bile coloring matter, tests for, 467.

Bile-ducts, ascarides in, 445 ; cancer of, 492.

Bile in intestinal catarrh, 464 ; in typhoid fever, 11.

Biliary abscess, 474, 476 ; acids in the blood, 466 ; acids in the urine, 467.

Biliary colic, 470.

Biliary fistulæ, 473.

Bilious remittent fever, 95.

Birth palsies, 567.

Bismuth, subnitrate of, in gastric catarrh, 382 ; in intestinal catarrh of children, 417, 418.

Bitters in gastric catarrh, 381 ; in scurvy, 960 ; in valvular cardiac disease, 307.

Black expectoration, 245.

Black vomit, 102.

Bladder, cancer of, 897 ; care of, in myelitis, 627 ; catarrh of, 893 ; diphtheria of, 894 ; " hæmorrhoids " of, 429 ; paralysis of, in injuries to spinal cord, 607 ; paralysis of, in locomotor ataxia, 643 ; paralysis of, in myelitis, 621 ; paralysis of, in pressure-paralysis of the spinal cord, 613 ; paralysis of, in progressive paralysis, 764 ; paralysis of, in spinal apoplexy, 605.

" Bleeders," 963.

Bleeding. See HÆMORRHAGE.

Bleeding in cerebral hæmorrhage, 736 ; in heart disease, 306 ; in pneumonia, 205 ; in sunstroke, 748.

Bleeding (local) for cerebral abscess, 743 ; for cerebral hæmorrhage, 736 ; for cerebral hyperæmia, 710; for cystitis, 896 ; for hæmatoma of the dura mater, 698 ; for hæmorrhage into the spinal meninges, 603 ; for infantile spinal paralysis, 670 ; for meningitis, 108, 701 ; for pericarditis, 328 ; for peri-

tonitis, 456 ; for pneumonia, 205 ; for sciatica, 527 ; for typhlitis, 421.
Blepharitis ciliaris, 1000.
Blepharospasm, 572.
Blindness from anæmia (amaurosis, anæmic), 932 ; from tumors of the brain, 751.
Blisters for neuralgia, 519 ; for sciatica, 527.
Blood-casts, 826, 827.
Blood, changes in the, 924.
Blood, character of, in anæmia, 935 ; in cancer of the stomach, 393 ; in chlorosis, 935 ; in cholera, 87 ; in diabetes, 965, 975 ; in gout, 984 ; in hæmoglobinuria, 953, 955 ; in hæmophilia, 963 ; in leukæmia, 947 ; in pernicious anæmia, 943 ; in pseudo-leukæmia, 952.
Blood, poverty of the, 928.
Blood-test, Heller's, 828.
Blood, transfusion of, in leukæmia, 951 ; in pernicious anæmia, 945 ; in poisoning from carbonic oxide gas, 1007 ; in poisoning from nitrobenzine, 1008.
Blood-vessels, affections of, 331.
Blue line on gums in lead poisoning, 1005.
Boils after small-pox, 56 ; in diabetes, 973, 976.
Bones, growth of, in infantile spinal paralysis, 669 ; in rachitis, 921.
Bones, lesions of, in leukæmia, 946, 948 ; in rickets, 921; in scrofula, 1000 ; in typhoid fever, 17 ; marrow of, in leukæmia, 946 ; in osteomalacia, 926 ; in pernicious anæmia, 940, 941 ; in pseudo-leukæmia, 952; pain in, in leukæmia, 948 ; in pernicious anæmia, 942 ; softening of, in osteomalacia, 926 ; in rachitis, 921.
Borborygmi in intestinal catarrh, 405.
Bothriocephalus latus, 440, 442.
Botulismus, 1009.
Bovine tuberculosis, 208.
Brachial paralysis, 564 ; combined, 567 ; in acute ascending spinal paralysis, 675; peripheral, prognosis and treatment of, 567.
Brachial spasm, 575.
Brain, abscess of, 741 ; diagnosis, 743 ; distinguished from tumor of, encapsulated, 743 ; focal symptoms, 742 ; idiopathic, 741 ; metastatic, 741 ; traumatic, 741 ; treatment of, 743.
Brain, anæmia of, 708 ; ætiology of, 709 ; symptoms of, 709 ; treatment of, 709.
Brain and cord, multiple sclerosis of, 627, 744. See also Multiple Sclerosis.
Brain, atrophy of, in progressive general paralysis, 765.
Brain, cancer of, 749 ; congestion of, 709; cortical centers of, 711 ; cysts in, 441, 756.
Brain, diseases of, remarks in regard to topical diagnosis of, 710.
Brain, echinococcus of, 756; foci of sclerosis in syphilis, 757.
Brain, glioma of, hæmorrhages into, 749 ; situation of, 749.
Brain, hyperæmia of, 708 ; symptoms of, 710 ; treatment of, 710.
Brain in cerebro-spinal meningitis, 104 ; inflammation of, 741.
Brain, œdema of, in uræmia, 831.
Brain, porencephalus of, 745.
Brain, psammoma of, 749 ; sarcoma of, 749.
Brain, sclerosis of, 627, 744 ; diffuse, 744 ; insular, 627.
Brain, softening of, red, yellow, and white, 737, 739 ; ætiology of, 737 ; diagnosis of, 740 ; idiopathic, 744 ; in tumor of, 752.
Brain, syphilis of, 757 ; apoplectic symptoms in, 758 ; diagnosis of, 759 ; disease of arteries in, 757 ; hereditary, 757 ; new growths in, 758 ; symptoms of, 758.
Brain, syphiloma of, 749 ; situation of, 757, 758.
Brain, tubercle of, 749.
Brain, tumors of, 748 ; at base, 752 ; cerebral compression caused by, 749 ; diagnosis of, 754 ; in the cerebellum, 753 ; in the cerebral hemispheres, 752 ; in the cortex, 752, 754 ; involving nerves at base, 752 ; medical treatment of, 755 ; originating in the meninges, 749, 754 ; surgical treatment of, 756 ; symptoms, general, 749 ; symptoms, localizing, 751 ; varieties, 748.
Brand's method in typhoid fever, 22.
Breakbone fever, 99.
Breathing (see Respiration) ; mouth, 356.
Breath, odor of, in diabetic coma, 973.
Brickmaker's anæmia, 448.
Bright's disease, 822 ; acute, 836 ; diagnosis of, 845 ; ætiology of, 836 ; parenchymatous form of, 839 ; prognosis in, 845 ; symptoms of, 841 ; treatment of, 846.
Bright's disease, chronic, 849, 856 ; cardio-vascular changes in, 853, 859 ; causes of, 849, 856 ; interstitial form of, 856 ; parenchymatous form of, 849 ; symptoms of, 852, 858 ; treatment of, 855, 863.
Bromide of ammonium in epilepsy, 778.
Bromide of potash in abscess of the brain, 746 ; in athetosis, 789 ; in bronchial asthma, 172 ; in chorea, 783 ; in diabetes, 981 ; in epilepsy, 777 ; in epileptiform attacks following cerebral paralysis of children, 746 ; in habitual headache, 532 ; in hysteria, 813 ; in infantile convulsions, 780 ; in neuralgia, 520 ; in neurasthenia, 818 ; in paralysis agitans, 787 ; in spasm of the face, 573 ; in spasm of the glottis, 143 ; in spasm of the muscles of mastication, 572 ; in spasm of the muscles of the neck, 575 ; in tetanus, 794 ; in tetany, 791 ; in whooping-cough, 163.
Bromide of sodium in epilepsy, 778.
Bromine, poisoning from, 1004.
Bromism, 1004.
Bronchi, casts of the, 158 ; dilatation of, 164 ; stenosis of, 167.
Bronchial blennorrhœa, 152.
Bronchial catarrh (see Bronchitis), 146, 151.
Bronchiectasis, 164 ; cylindrical, 164 ; saccular, 164 ; sputum in, 165.
Bronchiolitis, 148; exudative, 171.
Bronchitis, acute, 146 ; ætiology of, 146 ; symptoms of, 147 ; treatment of, 150 ; capillary, 148 ; after whooping-cough, 161 ; asthmatic, 171.
Bronchitis, chronic, 151 ; ætiology of, 151 ; symptoms of, 151 ; treatment of, 153.
Bronchitis, croupous, 158 ; fibrinous, 158 ; fœtid, 155 ; hæmorrhagic, 147 ; in children, 149 ; in erysipelas, 64 ; in gout, 987 ; in malaria, 94 ; in measles, 47, 49 ; in rachitis, 923, 924 ; in small-pox, 56 ; in typhoid fever, 12 ; in valvular cardiac disease, 296 ; pseudo-membranous, 158 ; putrid, 155 ; secondary, 146 ; symptomatic croupous, 158 ; tubercular, 213.
Bronchophony in pneumonia, 195.
Broncho-pneumonia, 184.
Bronchorrhœa, serous, 152.
Bronzed skin, 878.
Brown induration of lung, 249.
Brown-Séquard's paralysis, 682.

Bruit. See MURMUR.
Bruit, d'airain, 269; de cuir neuf, in pericarditis, 258; de diable in anæmia, 933; in chlorosis, 933; in pernicious anæmia, 942; de pot fêlé, 222.
Bulbar. See also MEDULLA.
Bulbar myelitis, acute, 694.
Bulbar paralysis, acute, 694; chronic, 685, 690; inflammatory, 694; in progressive muscular atrophy, 652; progressive, 685; progressive, ætiology of, 685; progressive, complications of, 657, 689; progressive, extension of, to the nerves, 690; progressive, treatment of, 689.
Bulbar symptoms in acute ascending spinal paralysis, 675; in amyotrophic lateral sclerosis, 652; in compression of the medulla, 695; in embolism and thrombosis of the basilar artery, 694; in progressive general paralysis, 764; in tubercular meningitis, 704.
"Bulbar pulse," 292.
Butyl chloral in neuralgia of the trigeminus, 523.
Butyric acid in gastric juice, 379.

Cachexia in cancer of the stomach, 393; malarial, 95; saturnine, 1005.
Cachexie pachydermique, 589.
Cæcitis, stercoral, 418.
Cæcum, cancer of, 426; inflammation of, 418.
Caffeine in hemicrania, 593.
Caisson disease, 608.
Calabar-bean in tetanus, 794.
Calcaneus in paralysis of the tibial nerve, 569.
Calculi, biliary, 470; pancreatic, 504; renal, 884; tonsillar, 353; urinary, forms of, 885.
Calculous pyelitis, 882.
Calm stage in yellow fever, 101.
Calomel in cholera morbus, 413; in intestinal catarrh of children, 416; in purulent meningitis, 702; in relapsing fever, 36; in tubercular meningitis, 706; in typhoid fever, 21.
Camphor in articular rheumatism, 911; in erysipelas, 65; in scarlet fever, 45; in typhoid fever, 26.
Cancer, colloid, 391; fibroid, 391; medullary, 391.
Cancer, metastatic, in cancer of the œsophagus, 370; in the bones, 393; in the pleura, 272.
Cancer of the bile-ducts, 492; bowel, 426; brain, 749; intestines, 426; kidney, 872; larynx, 145; liver, 492; lungs, 250; œsophagus, 369; pancreas, 504; peritoneum, 463; pleura, 272; stomach, 391.
Cancrum oris, 347.
Cannabinum tannicum in neurasthenia, 819.
Cannabis indica in diabetes, 981; in hemicrania, 593; in neurasthenia, 819.
Cantani's dietary for diabetes, 978.
Cantharides as a cause of cystitis, 893.
Capillary pulse in aortic insufficiency, 289.
Capsule, internal, lesions of, 721; as a cause of cerebral hemianæsthesia, 721, 725; as a cause of hemiplegia, 721, 724; as a cause of post-hemiplegic chorea, 721, 725; involving facial nerve, 721.
Caput Medusæ, 481.
Caput obstipum, 574.
Caput quadratum in rickets, 923.
Carbolic acid in articular rheumatism, 910; in diabetes, 981; in erysipelas, 65; in scarlet fever, 44, 45; in whooping-cough, 163; poisoning from, 1008.
"Carbolic mask," 157.
Carboluria, 1008.
Carbuncle in diabetes, 973.
Carbunculus contagiosus, 117.
Carcinoma. See CANCER.
Carcinosis, miliary, of the lungs, 250; of the peritoneum, 463.
Cardiac. See also HEART.
Cardiac compensation, rupture of, 283.
Cardiac failure, 283.
Cardiac murmurs, anæmic, 933; in chlorosis, 935; in chorea, 782; in idiopathic anæmia, 942; in leukæmia, 949.
Cardiac murmurs, organic, in aortic insufficiency, 287, 288; in aortic stenosis, 290; in congenital heart affections, 293, 294; in mitral incompetency, 283; in mitral stenosis, 285; in tricuspid valve disease, 292.
Cardiac muscle, atrophy of, from pericarditis, 326, 327; cloudy swelling of, 16, 298; fatty degeneration of, 315; lesions of, 298.
Cardiac neuroses, 318; overstrain, 312.
Cardiac septa, anomalies of, 394.
Cardialgia in gastric ulcer, 385; in malaria, 95.
Cardio-sclerosis, 307.
Cardio-vascular changes in renal disease, 834, 858.
Carotids, aneurism of, 339; compression of, in epileptic paroxysms, 778; ligature and compression of, in cerebral hæmorrhage, 736.
Caseation, 213.
Castoreum in hysteria, 813.
Castration for hysteria, 813.
Casts of bronchial tubes in fibrinous bronchitis, 158.
Casts of urinary tubules, 826; epithelial, 827; fatty, 827; granular, 827; hyaline, 826; waxy, 827.
Casts, tube, in acute Bright's disease, 842; in chronic Bright's disease, 852, 859.
Catalepsy, 796; hypnotic, 796; hysterical, 796; in cerebral disease, 797; in connection with psychoses, 797.
Cataract in diabetes, 972.
Catarrh, acute gastric, 372; bronchial, 146; chronic bronchial, 151; chronic gastric, 374; chronic laryngeal, 132; dry bronchial, 152; gastro-duodenal, 464; nasal, 125; of the bladder, 893; simple chronic (nasal), 126.
Catarrhal inflammation, influence in tuberculosis, 210.
Catarrhe pituiteux, 152; sec, 152.
Catarrhus æstivus, 125.
Cathartics in anæmia and chlorosis, 938; in cerebral hæmorrhage, 736; in cerebral hyperæmia, 710; in cirrhosis of the liver, 483; in dysentery, 80; in hæmatoma of the dura mater, 698; in hæmophilia, 965; in infantile convulsions, 779; in intestinal catarrh, 409; in locomotor ataxia, 648; in trichinosis, 123.
Catheterization in myelitis, 627.
Cat's purr, 284, 286.
Caustic potash, poisoning from, 1004; soda, poisoning from, 1004.
Cautery, actual, in acute ascending spinal paralysis, 676; in neuralgia, 519; in pachymeningitis cervicalis hypertrophica, 603; in pressure paralysis of the spinal cord, 614; in spasm of the muscles of the face, 573; in spasm of the muscles of the neck, 575.
Cavities and fissures in the spinal cord, 677.
Cavities, pulmonary, physical signs of, 222.
Centrum ovale, lesions of, as a cause of ataxic aphasia, 721; as a cause of hemiopia, 721; as a cause of

hemiplegia, 721 ; as a cause of monoplegia, 721 ; as a cause of word deafness, 721.
Cephalæa (see HEADACHE, HABITUAL), 530.
Cephalalgia (see HEADACHE, HABITUAL), 530.
Cerebellar, ataxia, 723 ; vertigo, 723, 724.
Cerebellum, tumors of, 753.
Cerebral arteries, aneurism of, 725 ; arterio-sclerosis of, 725 ; endarteritis of, 725 ; syphilitic endarteritis of, 757 ; syphilis of, 757 ; syphilis of, as a cause of apoplectiform attacks, 758 ; syphilis of, as a cause of cerebral softening, 738, 757 ; syphilis of, histology of, 757.
Cerebral embolism, 737 ; in gout, 988 ; repeated attacks caused by, 740.
Cerebral hæmorrhage, 725 ; ætiology of, 725 ; aneurisms, miliary, in, 725 ; as a cause of cerebral hemiplegia, 731 ; convulsions in, 729 ; diagnosis of, 736 ; focal symptoms of, 731 ; hereditary tendency to, 726 ; in arterio-sclerosis, 725 ; in gout, 726 ; in tumors of the brain, 750 ; in valvular cardiac disease, 301 ; morbid anatomy of, 726 ; prognosis in, 736 ; symptoms of, 727 ; treatment of, 736 ; usual situation of, 726.
Cerebral localization, 710.
Cerebral meninges, diseases of, 696.
"Cerebral rheumatism," 905.
Cerebral sinuses, thrombosis of, 707.
Cerebral symptoms in contracted kidney, 861, 862 ; in gout, 988 ; in nephritis, 831, 832 ; in pneumonia, 198 ; in pseudo-leukæmia lymphatica, 952 ; in purpura hæmorrhagica, 962 ; in relapsing fever, 32 ; in scarlet fever, 37 ; in septico-pyæmia, 111.
Cerebral tuberculosis, 749, 755 ; symptoms of, 749.
Cerebro-spinal meningitis, epidemic (see also MENINGITIS), 103 ; anomalous forms of, 107 ; complications of, 106 ; distinguished from tubercular meningitis, 107 ; encephalitic foci in, 104 ; malignant form, 104 ; sequelæ of, 107 ; treatment of, 108 ; varieties of, 105, 107.
Cervical muscles, paralysis of, 562 ; in acute ascending spinal paralysis, 675 ; resulting from stasis, 677.
Cervical muscles, spasm of, 574, 575 ; in hysteria, 804 ; prognosis of, 574 ; treatment of, 574.
Cervical pachymeningitis, 602.
Cervico-brachial neuralgia, 524.
Cervico-occipital neuralgia, 523.
Cestodes, 440.
Chalicosis pulmonum, 245.
Champagne in yellow fever, 103.
Charbon, 117.
Charcot's crystals, 169 ; joints, 644.
Cheek, gangrene of, 347.
Cheese, poisoning from, 1010.
Cheyne-Stokes breathing in tuberculous meningitis, 704.
Chicken breast, 923.
Chicken-pox, 60.
Children, diabetes in, 967 ; pneumonia in, 200 ; rheumatism in, 900 ; broncho-pneumonia in, 185 ; tuberculosis of mesenteric glands in, 424 ; typhoid fever in, 4.
Chloral in chorea, 783; in diabetes, 981; in eclampsia, 780 ; in neuralgia, 520 ; in tetanus, 794 ; in trigeminal neuralgia, 523.
Chlorine gas, poisoning from, 1004.
Chloro-anæmia in phthisis, 936.
Chloroform in epilepsy, 778 ; in hepatic colic, 475 ; in hysteria, 804 ; in infantile convulsions, 779 ; in renal colic, 888 ; in spasms of the diaphragm, 577.
Chloroform, poisoning from, 1007.
Chloroform test for bile, 467.
Chlorosis, 928, 934 ; diagnosis of, 936 ; distinguished from gastric diseases, 930 ; distinguished from renal diseases, 931 ; distinguished from syphilis, 931 ; distinguished from tuberculosis, 941, 936 ; Egyptian, 448 ; chlorosis, ætiology of, 928, 929 ; fever in, 934 ; heart symptoms in, 933 ; menstrual disturbance in, 935 ; morbid anatomy of, 933, 935 ; relapses of, 936 ; symptoms of, 934 ; treatment of, 936 ; with regard to sexual disturbances, 929, 935.
Choked disk. See NEURITIS, OPTIC.
Cholæmia, 468, 489.
Cholelithiasis, 470.
Cholera (Asiatic), 81 ; asphyxia in, 84 ; bacillus of, 82 ; complications of, 86 ; contagiousness of, 82 ; dejections in, 84 ; diagnosis of, 84 ; distinguished from acute arsenical poisoning, 87 ; eruption in, 86 ; exciting causes of, 83 ; period of incubation in, 84 ; post-mortem appearances in, 86 ; predisposition to, 83 ; prognosis of, 88 ; specific poison of, 82 ; treatment of, 88.
Choleraic diarrhœa, simple, 84 ; premonitory, 84.
Choleraic nephritis, 86.
Choleraic typhoid, 86 ; uræmic, 86.
Cholera morbus, 410 ; diagnosis of, 412 ; mortality in, 412 ; treatment of, 412 ; cholera nostras, 410 ; cholera sicca, 85 ; cholera typhoid, 86.
Cholerine, 84.
Cholesterine, calculi formed of, 471.
Cholesterine and pigment, calculi formed of, 471.
Chorditis tuberosa, 133.
Chorditis vocalis inferior hypertrophica, 133.
Chorea, 780 ; ætiology of, 780 ; as related to chorea major, 780 ; as related to embolic processes, 783 ; complications of, 780, 782 ; diagnosis of, 783 ; gravidarum, 781 ; in articular rheumatism, 780 ; nature and situation of the disease, 782 ; predisposition to, 780 ; premonitory symptoms of, 781 ; prognosis of, and treatment, 783 ; relapses in, 782 ; unilateral, 781.
Chorea, endemic, 780 ; major, 780 ; rhythmic or hysterical, 810 ; Sydenham's, 780.
Chorea, heart symptoms of, 782 ; infectious origin of, 780 ; seasonal relations of, 780.
Chorea, Huntington's or hereditary, 780.
Chorea, spasm, 573, 576, 810.
Choroid, tubercles in, 240.
Choroid, tuberculosis of the, in meningitis, 704.
Chyluria, parasitic, 875.
Cicatrices of the œsophagus, 366 ; of the œsophagus from sulphuric acid, 1003.
Cicatricial induration caused by syphilis of the brain, 757.
Cinchona, in purpura hæmorrhagica, 962 ; in scurvy, 961.
Circulatory system, diseases of, 276.
Circumcision, dangerous in hæmophilia, 964.
Circumflex nerve, affections of, 564, 574.
Cirrhosis, hypertrophic, of the liver, 483.
Cirrhosis of kidney, 856 ; of liver, 478 ; of lung, 223 ; of pancreas, 504.
"Clap-threads" in cystitis, 895.
Clavicles, deformed, in rachitis, 923.
Claw-shaped hand, 566.
Climate, change of, in asthma, 172 ; in chronic

Bright's disease, 855 ; in pulmonary tuberculosis, 232, 233.
Clitoris, cauterization of, in hysteria, 813.
Clonus, 545.
Clownismus in hysteria, 808.
Club-foot resulting from infantile spinal paralysis, 669.
Coagulation necrosis, 213.
Coccyodynia, 528 ; in locomotor ataxia, 528, 643 ; operative removal of coccyx for, 528.
Cod-liver oil in diabetes, 979 ; in osteomalacia, 927 ; in pulmonary tuberculosis, 232 ; in rachitis, 925 ; in scrofula, 1001.
Coffee-ground vomit, 392.
Coin sound, 269.
Colchicum in articular rheumatism, 910 ; in gout, 992.
Cold pack, method of giving, 188.
Colic, biliary, 470 ; hepatic, 470 ; in angio-neurotic œdema, 588 ; lead, 1005 ; renal, 884.
Collapse stage, in cholera, 85 ; in cholera morbus, 411 ; in hepatic colic, 472 ; in peritonitis, 454 ; in pneumonia, 196 ; in pneumothorax, 268 ; in typhoid fever, 8, 26.
Colloid cancer of peritoneum, 463 ; of stomach, 391.
Colon, cancer of, 426.
Colo-typhoid, 9.
Columns, posterior, gray degeneration of, 632.
Coma, diabetic, 973 ; causes of, 974 ; treatment of, 981.
Coma, epileptic,773 ; from heat stroke, 747 ; in abscess of the brain, 742 ; in acute yellow atrophy, 487 ; in alcoholic poisoning, 1006 ; in apoplexy, 729 ; in cerebral hæmorrhage, 729 ; in cerebral syphilis, 758 ; in multiple sclerosis, 630 ; in pernicious malaria, 94 ; in thrombosis of the cerebral sinuses, 707 ; in typhus fever, 30 ; in uræmia, 832, 834.
Coma, post-epileptic, 773.
Coma-vigil, in typhoid fever, 14.
Comma bacillus, 82.
Commotio spinalis, 819.
Compensation in valve lesions, 282.
Complexion in anæmia, 931, 934, 941 ; in diabetic coma, 973 ; in leukæmia, 949 ; in pseudo-leukæmia, 952.
Compression as a cause of thrombosis, 502.
Compression of the medulla oblongata,695 ; diagnosis and prognosis of, 695.
Compression of the œsophagus, 366.
Conchinine in malaria, 97.
Concretio pericardii, 326.
Concussion of spinal cord, 819.
Congenital heart affections, 293.
Congo-red test for free acid, 378.
Conjugate deviation in hemiplegia, 729.
Conjunctivitis in difficult dentition, 351 ; in diphtheria, 69 : in gout, 987 ; in measles, 48 ; in scarlet fever, 41 ; in scrofula, 1000 ; in whooping-cough, 160.
Conscience musculaire, 509, 802.
Constipation, habitual, 430 ; in diseases of the nervous system, 430 ; treatment of, 431.
Constipation in anæmia and chlorosis, 932 ; in cases of cerebral tumor, 751 ; in cirrhosis of the liver, 480 ; in gastric catarrh, 379 ; in intestinal stenosis, 435 ; in jaundice, 466 ; in locomotor ataxia, 643 ; in myelitis, 621 ; in typhlitis, 419 ; in typhoid fever, 9.
Constitutional diseases, 928.
Consumption. See TUBERCULOSIS.
Consumption, galloping, 217.
Contracted kidney, 856 ; embolic, 872 ; genuine, 856 ; in arterio-sclerosis, 857 ; in gout, 856, 987 ; in obesity, 996 ; secondary, 849, 856.
Contractions, rhythmical, 540.
Contracture des nourrices, 789 ; hysterical, 803 ; in hemiplegia, 734.
Convulsions, cataleptic, 541 ; epileptiform, 540, 764, 770, 773.
Convulsions, epileptic, 771 ; hysterical, 806 ; in acute yellow atrophy, 487 ; in alcoholism, 1006 ; in cerebral embolism, 739 ; in cerebral hæmorrhage, 729 ; in cerebral hydatids, 756 ; in cerebral syphilis, 758 ; in cerebral tumors, 750 ; in chronic Bright's disease, 831 ; in compression of the medulla, 695 ; in diffuse sclerosis of the brain, 744.
Convulsions, infantile, 779 ; diagnosis of, 779 ; ætiology of, 779 ; relation to rickets, 779 ; symptoms of, 779 ; treatment of, 779.
Convulsions in focal lesions of the cortex, 714, 724 ; in general paralysis, 764 ; in hæmatoma of the dura mater, 697 ; in hepatic colic, 472 ; in hydrophobia, 114 ; in hysteria, 806 ; in infantile cerebral paralysis, 744 ; in infantile spinal paralysis, 668 ; in meningitis, 700, 704 ; in multiple sclerosis, 630 ; in sunstroke, 747 ; in thermic fever, 747 ; in uræmia, 831 ; Jacksonian, 714.
Convulsive tic, 573.
Co-ordination, disturbance of, in tabes, 639.
Copaiba, balsam of, in cirrhosis of the liver, 483 ; in cystitis, 896.
Copaiba, resin of, in cirrhosis of the liver, 483.
Copper, paralysis due to, 571 ; poisoning from, 1005 ; sulphate of, in chorea, 783 ; test for sugar, 968.
Coprolalia, 573.
Coprostasis, 432.
Cor adiposum, 315.
Cor villosum, 322.
Coronary arteries, in angina pectoris, 308, 318 ; obliteration, 308.
Corpora quadrigemina, lesions of, 722 ; as a cause of ocular disturbances, 722, 725 ; tumors in, 753.
Corpulence, 992.
Corrigan's pulse, 289.
Corset-liver, 499.
Coryza, 125 ; fever caused by, 126 ; fœtida, 127 ; from the iodides, 125.
Cortical epilepsy, 714.
Costiveness, 430.
Cough, during aspiration of pleural effusion, 266 ; hysterical, 577 ; in pertussis, 160 ; paroxysmal, in bronchiectasis, 164 ; spasmodic, 577.
Cow's milk as food for infants, 411.
Cow-pox, 58.
Cracked-pot sound, 222.
Cramps in cholera, 85.
Cramps in the legs, 575 ; after severe physical exercise, 575 ; in hysteria, 575 ; liability to, 575.
Craniometric anomalies in the epileptic, 775 ; in the rachitic, 923.
Craniotabes, in rachitis, 923 ; relation (alleged) to congenital syphilis, 921.
Cranium, in osteomalacia, 927 ; in rachitis, 923.
Creasote in gastric catarrh, 381 ; in intestinal catarrh of children, 417 ; in tuberculosis of the lungs, 231.
Cremaster reflex, 543 ; in cerebral hæmorrhage, 732.
Crepitant râles in croupous pneumonia, 195.
Crepitatio redux, 195.
Crescents in blood in malaria, 92.
Crises, gastro-intestinal, 643; in locomotor ataxia, 643.

Crisis, in pneumonia, 199 ; in typhus fever, 29.
Croup, 65 ; ascending, 68 ; relation to diphtheria, 68.
Croup, false, 131.
Croupous pneumonia, 189.
Crura cerebri, lesions of, 723.
Crural, anterior, paralysis of, 568.
Cry, cephalic, 705.
Crystals, Leyden's, 169.
Cundurango bark in cancer of the stomach, 395.
Cups, dry, in acute ascending spinal paralysis, 676 ; in pressure paralysis of the spinal cord, 614.
Curare in paralysis agitans, 787 ; in spasms of the facial muscles, 573 ; in tetanus, 794 ; in tetany, 791.
Curschmann's spirals, 169.
Cutaneous. See also Skin.
Cutaneous abscesses in erysipelas, 64.
Cutaneous anæsthesia, 510 ; bilateral, 511 ; cerebral, 510, 512 ; in diseases of the internal capsule, 721, 725 ; in hysteria, 512, 800 ; in laundresses, 511 ; in locomotor ataxia, 511 ; painful, 512 ; peripheral, 510, 511 ; spinal, 510, 511 ; symptoms of, 512 ; treatment of, 514.
Cutaneous ecchymoses in articular rheumatism, 904 ; in typhoid fever, 17.
Cutaneous reflexes, 543 ; diminution or absence of, 544 ; exaggeration of, 544 ; in cerebral hæmorrhage, 732 ; in epilepsy, 773 ; in infantile spinal paralysis, 669 ; in locomotor ataxia, 641 ; in myelitis, 620 ; in poliomyelitis of adults, 672 ; in pressure paralysis of the spinal cord, 612 ; in tetanus, 792 ; in the extremities, 543 ; sluggishness of, 543.
Cutaneous sensibility, electro-, 508.
Cutaneous sensibility, in locomotor ataxia, 639 ; in tubercular meningitis, 704 ; varieties, and modes of testing, 505.
Cyanosis, in acute tuberculosis, 237 ; in congenital heart disease, 293, 294 ; in emphysema, 176, 177 ; in epileptic paroxysms, 772 ; in stenosis of the pulmonary orifice, 293.
Cynanche contagiosa, 65 ; gangrænosa, 349.
Cystic disease, of kidney, 874 ; of liver, 494.
Cystic duct, obstruction of, 473.
Cysticercus cellulosæ, 441 ; in the brain, 756.
Cysticercus racemosus, 441.
Cystine, calculi composed of, 885.
Cystitis, 893 ; ætiology of, 893 ; chronic, 895 ; complications of, 896 ; in locomotor ataxia, 643 ; in myelitis, 621 ; in pressure paralysis of the spinal cord, 613 ; in spinal apoplexy, 605 ; symptoms, 894 ; treatment of, 896 ; tuberculous, 888 ; varieties, 894.
Cysts, in kidneys, 874 ; of brain, apoplectic, 727 ; of brain, thrombotic, 739 ; of larynx, 144 ; pancreatic, 504 ; porencephalic, 745.

Dancing mania, 780.
Dandy fever (dengue), 99.
Deaf-mutism after cerebro-spinal fever, 107.
Deafness, in cerebral tumor, 753, 754 ; embolism and thrombosis of the basilar artery, 694 ; in epidemic cerebro-spinal meningitis, 107 ; in epilepsy, 772 ; in hysteria, 801 ; in lesions of the centrum ovale, 721 ; in locomotor ataxia, 643 ; in Ménière's disease, 769 ; in scarlet fever, 40 ; in uræmia, 832.
Debility, nervous, 815.
Decubitus, acute malignant, in hemiplegia, 735 ; (bedsores) in transverse myelitis, 622 (treatment) 627.
Decubitus laryngis in typhoid, 13.
Degeneration, fatty, in acute phosphorus poisoning, 1006 ; in acute yellow atrophy, 486 ; in anæmia, 933, 940 ; in typhoid fever, 16 ; of the heart, 315 ; of the liver, 486, 498.
Degeneration, parenchymatous, in typhoid fever, 11, 16 ; of the kidney, 839.
Degeneration, physical signs of, in epileptics, 775.
Degeneration, reaction of, 551 ; anatomical changes in nerves and muscles in, 553 ; complete, 552 ; in amyotrophic lateral sclerosis, 652 ; in infantile spinal paralysis, 669 ; in lead paralysis, 570 ; in myelitis, 622 ; in paralysis of the deltoid, 564 ; in paralysis of the facial, 560 ; in poliomyelitis of adults, 672 ; in progressive bulbar paralysis, 687 ; in progressive muscular atrophy, 656 ; in secondary neuritis, 582 ; partial, 551.
Degeneration, sclerotic, of the heart, 307.
Deglutition, impairment of, in acute bulbar myelitis, 694 ; in acute bulbar paralysis, 692 ; in amyotrophic lateral sclerosis, 652 ; in chronic poliomyelitis, 673 ; in compression of the medulla, 695 ; in dilatation of the œsophagus, 363 ; in diphtheria, 70 ; in laryngeal catarrh, 131 ; in pericarditis, 325 ; in progressive bulbar paralysis, 686 ; in progressive muscular atrophy, 657 ; in pseudo-leukæmia lymphatica, 952 ; in thoracic aneurism, 337 ; in tonsillitis, 352 ; in trichinosis, 122.
Deglutition, paralysis of, in bulbar hæmorrhage, 692 ; in embolism and thrombosis of the basilar artery, 694 ; in hysteria, 803 ; in progressive general paralysis, 764.
Deglutition pneumonia, 185.
Delayed resolution in pneumonia, 202.
Delayed sensation in tabes, 640.
Delirium, acute, in lead poisoning, 1005 ; expansive, 763 ; in acute rheumatism, 905 ; in acute yellow atrophy, 487 ; in cerebral hæmorrhage, 728 ; in dengue, 100 ; in diabetes, 973 ; in hysteria, 807, 808 ; in meningitis, 105, 700, 705 ; in pneumonia, 198 ; in pylephlebitis suppurativa, 501 ; in typhoid fever, 14 ; in typhus fever, 30.
Delirium tremens, 1007 ; in croupous pneumonia, 198.
Delivery paralysis, 567.
Deltoid paralysis, 564.
Delusions of grandeur, 763.
Dementia from epilepsy, 775.
Dementia paralytica, 760.
Dengue, 99.
Dentitio difficilis, 350.
"Desiccation" in small-pox, 54 ; in the treatment of obesity, 999.
Desquamation, in measles, 47 ; in scarlet fever, 41 ; in small-pox, 54 ; in typhoid fever, 17.
Diabetes inositus, 983.
Diabetes insipidus, 982 ; heredity in, 982, 983 ; related to diabetes mellitus, 976, 982 ; related to polyuria, 982
Diabetes mellitus, 965 ; "accidental," 967 ; coma in, 973 ; complications of, 967, 971, 972 ; decipiens, 968 ; diagnosis of, 977 ; gangrene in, 973 ; hereditary influences in, 967 ; intermittent, 976 ; in children, 967 ; in obesity, 976 ; pancreas in, 966, 967 ; related to glycosuria, 976 ; theories of, 976 ; treatment of, 978 ; urine in, 967.
Diagnosis, topical, in brain diseases, 710.
Diaphoresis for bronchitis, 150 ; for nephritis, 847.
Diaphragm, paralysis of the, 568 ; in acute inflammatory bulbar paralysis, 694 ; modifying the movements of respiration, 568 ; treatment of 568.

Diaphragm, spasm of, clonic, 577 ; in hysteria, 810 ; tonic, 576.
Diarrhœa, in cholera Asiatica, 84 ; in cholera morbus, 411 ; in cirrhosis of the liver, 480 ; in dysentery, 78 ; in exophthalmic goitre, 597 ; in gastric catarrh, 379 ; in hysteria, 805 ; in intestinal catarrh, 404 ; in locomotor ataxia, 643 ; in noma, 347 ; in pneumonia, 197 ; in pulmonary gangrene, 243 ; in pulmonary tuberculosis, 227 ; in pylephlebitis, 501 ; in rachitis, 924 ; in septico-pyæmia, 112 ; in typhoid fever, 9 ; in uræmia, 832 ; in whooping-cough, 162.
Diathesis, gouty, 991.
Diathesis, hæmorrhagic, in acute articular rheumatism, 904 ; in anæmia, 934 ; in jaundice, 466 ; in scurvy, 958.
Diazo-reaction in typhoid fever, 18.
Dicrotism of the pulse in typhoid fever, 16.
Diet, in chronic gastric catarrh, 380 ; in constipation, 431 ; in diabetes, 978 ; in gout, 990 ; in infantile diarrhœa, 415 ; in obesity, 997 ; in scurvy, 960 ; in tuberculosis, 232 ; in typhoid fever, 22.
Digestive system, diseases of, 341.
"Digitalis eaters," 305.
Digitalis in nephritis, 848 ; in pericarditis, 328 ; in pleurisy, 264 ; in pulmonary emphysema, 180 ; in typhoid fever, 26 ; in valvular cardiac disease, 304 ; poisoning from, 1008.
Dilatation, bronchial, 164 ; stomachic, 396.
Diphtheria, 65 ; and croup, 65, 68 ; bacillus of, 66 ; contagiousness of, 67 ; diagnosis of, 70 ; gangrenous, 69 ; in animals, 67 ; in measles, 49 ; laryngeal, 68 ; nephritis in, 70 ; neuritis in, 70 ; of nares, 68 ; prognosis of, 71 ; septic, 66 ; symptoms of, 67 ; systemic infection, 69 ; treatment of, 71.
Diphtheritic colitis, 77, 78.
Diplegia, facial, 690.
Diplococcus pneumoniæ, 189.
Diplopia, 555 ; in multiple sclerosis, 630.
Disease, trichinatous, 121.
Disinfection in infectious diseases, 26 ; in typhoid fever, 26.
Dislocation, paralysis of the upper arm following, 564.
Displacement, symptoms resulting from, of other organs, in pleurisy, 260 ; in pneumonia complicated by pleurisy, 196 ; in pneumothorax, 269.
Dissecting aneurism, 340.
Distoma hæmatobium, 874.
Disuse, atrophy from, 622.
Dittrich's plugs, 156.
Diuretics in cirrhosis of the liver, 483 ; in nephritis, 848 ; in pleurisy, 264 ; in pulmonary emphysema, 180 ; in valvular cardiac disease, 306.
Diverticula of the œsophagus, 363.
Dochmius duodenalis, 448.
Dorso-intercostal neuralgia, 524.
Double vision, 555.
Douche, cold, in cutaneous anæsthesia, 515 ; in diabetes, 980 ; in hysteria, 812 ; in neuralgia of the joints, 529 ; in neurasthenia, 818 ; in scarlet fever, 45 ; in spasm of the diaphragm, 577.
Drainage and diphtheria, 67 ; and tonsillitis, 351 ; and typhoid fever, 3.
Drinker's, gin, liver, 478.
Drinking water as a cause of cholera, 83 ; of typhoid fever, 3.
Dropsy. See also Œdema.
Dropsy, cardiac, treatment of, 306 ; in amyloid disease of the kidney, 867 ; in aortic insufficiency, 290 ; in cancer of stomach, 393 ; in mitral insufficiency, 284 ; in mitral stenosis, 287 ; in phthisis, 229 ; in renal diseases, 828, 843, 853, 860 ; in scarlet fever, 41, 42 ; in typhoid fever, 16 ; in valvular cardiac disease, 299.
Dropsy, intermittent, of the joints, 590.
Dropsy of the gall-bladder, 473.
Duchenne's paralysis, 685.
Dullness, movable, in pleural effusion, 260 ; in pneumothorax, 269.
Dumb ague, 94, 95.
Duodenal ulcer, diagnosis of, from gastric, 423.
Duodenum, cancer of the, 426 ; catarrh of the, 406, 464 ; fistula of the, 473, 474 ; ulcer, perforating, of the, 423.
Dura mater, diseases of, 696 ; hæmatoma of, 696.
Durande's remedy for biliary calculi, 475.
Duroziez's double murmur, 290.
Dust, diseases due to the inhalation of, 245 ; prophylaxis of, 246 ; tubercle bacilli in, 246.
Dysarthria, 685, 716.
Dysentery, 77 ; abscess of liver in, 79 ; acute, 78 ; amœba coli in, 81 ; catarrhal, 78 ; chronic, 79 ; diagnosis of, 79 ; diphtheritic, 78 ; gangrenous, 78 ; prognosis of, 79 ; secondary, 78 ; treatment of, 80 ; tropical or amœbic, 81.
Dyspepsia, acute, 372 ; chronic, 374 ; nervous, 400 ; of children, 413.
Dysphagia, convulsive, in tetanus, 792 ; hysterical, 810 ; in cancer of the œsophagus, 366, 369 ; in hydrophobia, 114 ; in œsophagismus, 371 ; in œsophagitis, 362 ; in pericardial effusion, 325 ; in thoracic aneurism, 337 ; in tuberculous laryngitis, 137 ; lusoria, 366.
Dyspnœa, cardiac, treatment of, 306 ; from aneurism, 336 ; hysterical, 807 ; in acute miliary tuberculosis, 237 ; in anæmia, 932, 942 ; in anthrax, 120 ; in bilateral paralysis of abductors, 140 ; in bronchitis, 147, 152 ; in contracted kidney, 860 ; in croup, 68 ; in diabetic coma, 973 ; in new growths of the lungs, 251 ; in new growths of the mediastinum, 273 ; in œdema of the glottis, 136 ; in paralysis of the muscles of the larynx, 140 ; in pleurisy, 257 ; in pneumonia, 186, 193 ; in pneumothorax, 268 ; in pulmonary emphysema, 176 ; in pulmonary tuberculosis, 221 ; in septico-pyæmia, 112 ; in spasmodic laryngitis, 131 ; in stenosis of the bronchi, 167 ; in stenosis of the trachea, 166 ; in trichinosis, 122 ; in valvular cardiac disease, 296.
Dyspnœa, uræmic (uræmic asthma), 833.
Dystrophy, primary muscular, 658.

Ear, care of, in scarlet fever, 45 ; complications of, in scarlet fever, 40.
Ear, symptoms in the, in anæmia, 932, 942 ; in caisson disease, 608 ; in cerebral anæmia, 709 ; in compression of the medulla oblongata, 695 ; in facial paralysis, 559, 560 ; in hæmorrhage into the medulla oblongata, 691 ; in hysteria, 801 ; in leukæmia, 949 ; in Ménière's disease, 769 ; in meningitis, cerebrospinal, 105 ; in scarlet fever, 40 ; in scrofula, 1000.
Ebstein's method in obesity, 997.
Eburnation of cartilages, 913.
Echinococcus, 494 ; diagnosis, 495 ; endogenous, 494, 495 ; exogenous, 495 ; fluid, 496 ; granulosus, 495 ; hydatidosus, 495 ; multilocularis, 495 ; of the kidney, 874.

Echolalia, 573.
Eclampsia. See also CONVULSIONS.
Eclampsia gravidarum in acute nephritis, 845.
Eclampsia infantum, 779 ; ætiology of, 779 ; associated with spasms of the glottis, 142 ; liability of rachitic children to, 779.
Eclampsia, uræmic, 831, 844.
Ectasis, alveolar, 173.
Ehrlich's reaction in typhoid fever, 18.
Elastic tissue in sputum, 219.
Electrical reactions in facial palsy, 560, 561 ; in idiopathic muscular atrophy, 660, 662 ; in Landry's paralysis, 674 ; in multiple neuritis, 583 ; in poliomyelitis anterior, 669 ; in Thomsen's disease, 795.
Electricity in acute ascending spinal paralysis, 676 ; in acute bulbar paralysis, 695 ; in amyotrophic lateral sclerosis, 653 ; in anæsthesia of the trigeminus, 515 ; in anomalies of the sense of smell, 533 ; in anomalies of the sense of taste, 534 ; in articular neuroses, 529 ; in articular rheumatism, 911 ; in asthma, 172 ; in athetosis, 789 ; in cerebral abscess, 743 ; in cerebral hæmorrhage, 737 ; in cerebral syphilis, 760 ; in cervico-brachial neuralgia, 524 ; in chorea, 783 ; in chronic poliomyelitis, 674 ; in cutaneous anæsthesia, 515 ; in diabetes, 981 ; in dilatation of the stomach, 400 ; in epilepsy, 778 ; in exophthalmic goitre, 598 ; in facial paralysis, 562 ; in facial spasm, 573 ; in habitual constipation, 431 ; in habitual headache, 532 ; in hemicrania, 593 ; in hysteria, 812 ; in incontinence of urine, 898 ; in infantile cerebral paralysis, 745 ; in intercostal neuralgia, 525 ; in lead paralysis, 570 ; in locomotor ataxia, 647 ; in mastodynia, 525 ; in multiple sclerosis, 631 ; in muscular rheumatism, 919 ; in myelitis, 626 ; in nervous dyspepsia, 403 ; in neuralgia, 519 ; in neurasthenia, 818 ; in neuritis, 584 ; in occipital neuralgia, 523 ; in pachymeningitis cervicalis hypertrophica, 603 ; in paralysis of the diaphragm, 568 ; in paralysis of the motor branch of the trigeminus, 558 ; in paralysis of the ocular muscles, 557 ; in peripheral paralysis of the arm, 567 ; in pressure paralysis of the spinal cord, 614 ; in progressive muscular atrophy, 658 ; in progressive bulbar paralysis, 689 ; in progressive general paralysis, 767 ; in pseudo-muscular hypertrophy, 663 ; in railway spine, 821 ; in sciatica, 527 ; in spasm of the diaphragm, 577 ; in spasm of the muscles of the neck, 574 ; in spasm of the muscles of the shoulder, 575 ; in spasm of the trigeminus, 571 ; in spastic spinal paralysis, 666 ; in splenic leukæmia, 951 : in tetany. 791 ; in unilateral progressive facial atrophy, 594 ; in writer's cramp, 579.
Electricity used to test sensibility, 508.
Electro-cutaneous sensibility, 508.
Electrolysis in aneurism, 338.
Embolic processes in the lungs, 247.
Embolism and thrombosis of the basilar artery, 693 ; a cause of softening of the medulla and pons, 693 ; ætiology of, 693 ; symptoms of, 693 ; treatment of, 694.
Embolism in chorea, 783 ; in typhoid fever, 16 ; of cerebral arteries, 737 ; of cerebral arteries, diagnosis of, 740.
Emetics in bronchitis, 151 ; in dysentery, 80 ; in infantile convulsions, 779 ; in pulmonary œdema, 184.
Emotion, convulsive expressions of, in hysteria, 807.
Emphysema, 173 ; acute, 162 ; atrophic, 173 ; compensatory, 175 ; complementary, 175 ; connected with whooping-cough, 162, 175 ; diagnosis of, 179 ; essential, 173 ; followed by pulmonary tuberculosis, 179 ; hypertrophic, hereditary character of, 174 ; interlobular, 175 ; interstitial, 175 ; prognosis of, 179 ; treatment of, 180 ; vesicular, 175 ; vicarious, 175.
Empyema, 262 ; necessitatis, 256 ; perforation of lung in, 262 ; terminations of, 263 ; treatment of, 264.
Encephalitis, curable form of. 744 ; in children, 744 ; non-purulent, 743 ; purulent, 741.
Encephalomalacia, 737.
Encephalopathy, lead, or saturnine, 1005 ; syphilitic, 758.
Enchondroma of the lungs, 250.
Endarteritis chronica deformans, 331.
Endocarditis, acute, 276 ; acute recurrent, 279 ; chronic, 280 ; diagnosis of, 279 ; diphtheritic, 277 ; ætiology of, 276 ; fœtal, 277 ; in articular rheumatism, 276, 903 ; in chorea, 782 ; infectious, 276 ; in gonorrhœa, 276 ; in pneumonia, 197 ; in septicæmia, 111, 276 ; in tuberculosis, 228 ; malignant, 278 ; prognosis of, 280 ; recurring, 277, 279 ; rheumatoid, 278 ; ulcerative, 277 ; verrucosa, 277.
Endothelial carcinoma of the pleura, 272.
Enemata of cold water in jaundice, 469.
"English disease," 920.
Engouement, 190.
Enteric fever (see TYPHOID FEVER), 1.
Enteritis catarrhalis, 403 ; in children, 413 ; membranous or tubular, 408,
Enteroliths as a cause of appendicitis, 418, 419.
Enuresis nocturna, 898.
Eosinophilous blood-globules, 948.
Epidemic of typhoid fever in Plymouth, Pa., 4.
Epidermis, desquamation of, in scarlet fever, 41 ; in measles, 47 ; in smallpox, 54 ; in typhoid fever, 17.
Epilepsy, 770 ; ætiology of, 770 ; cortical, 714 ; diagnosis of, 776 : diurnal, 774 ; heredity in, 770 ; in general paresis, 764 ; in lead poisoning, 1005 ; in regard to cerebral anæmia, 776 ; in regard to sudden somnolence, 773 ; in regard to teething convulsions, 774 ; Jacksonian, 714 ; masked, 773 ; nocturnal, 774 : occurrence of various forms of, 771 ; procursive, 773, 774 ; reflex, 771 ; seat of the disease, 775 ; surgical treatment of, 777 ; traumatic, 771 ; treatment, 777.
Epileptic paroxysm, 771 ; aura preceding the, 771 ; convulsive stage of, 772 ; frequency of, 774 ; in progressive general paralysis, 764 ; rudimentary forms of, 773.
Epileptoid confusion of intellect, 773 ; sweating, 774.
Epistaxis, 129 ; in hæmophilia, 964 ; in scurvy, 129 ; in typhoid fever, 129 ; "renal," 862 ; vicarious, 129.
Erethitic habitus, 1000.
Ergot (ergotine) in acute ascending spinal paralysis, 676 ; in aneurism of the thoracic aorta, 338 ; in diabetes insipidus, 984 ; in exophthalmic goitre, 598 ; in habitual headache, 532 ; in hæmophilia, 965 ; in hemicrania, 593 ; in locomotor ataxia, 648 ; in myelitis, 627 ; in neuralgia, 520 ; in paralysis agitans, 787 ; in poliomyelitis of adults, 672 ; in progressive general paralysis, 767 ; in pulmonary tuberculosis, 235 ; in purpura hæmorrhagica, 962 ; in spastic spinal paralysis, 667 ; in spinal apoplexy, 605 ; poisoning from, 1009 ; psychoses due to, 1009.
Ergotism, 1009 ; convulsive, 1009 ; gangrenous, 1009.
Erosion, ulcers due to, in laryngeal catarrh, 130, 133.

Eructations in anæmia, 932, 942 ; in diabetes, 967 ; in intestinal obstruction, 435 ; in peritonitis, 454.

Eruption, acute, in scarlet fever, 37 ; hæmorrhagic, in small-pox, 57.

Erysipelas, 61 ; abscess in, 64 ; after vaccination, 59 ; bullous, 63 ; complications of, 64 ; contagiousness of, 62 ; diagnosis of, 64 ; facial, 61 ; Fehleisen's streptococcus of, 62 ; gangrenous, 63 ; idiopathic, 61 ; inoculation of, 62 ; in small-pox, 56 ; migratory, 63 ; of the new-born, 61 ; prognosis of, 64 ; puerperal, 61 ; pustulous, 63 ; traumatic, 61 ; treatment of, 64 ; vesicular, 63.

Erythema exsudativum, 962.

Erythromelalgia, 588.

État de mal, 774.

Eucalyptus, oil of, in splenic leukæmia, 950.

Eustrongylus gigas, 875.

Exaltation of ideas in general paresis, 763.

Exophthalmic goitre, 595 ; acute form, 597 ; ætiology of, 595 ; diagnosis of, 597 ; diminution of electrical resistance in, 597 ; examination of heart in, 595 ; "Graefe symptom" in, 596 ; nervous symptoms in, 596, 597 ; pigmentation in, 597 ; treatment of, 598 ; tremor in, 596 ; urticaria in, 597.

Exophthalmus in exophthalmic goitre, 596 ; in paralysis of the motores oculi, 556.

Expectorants in bronchitis, 151, 154 ; in pneumonia, 188, 206 ; in pulmonary emphysema, 180 ; in pulmonary tuberculosis, 234.

"Expectoration albumineuse" in pleurisy, 266.

Expectoration in asthma, 168, 169 ; in bronchiectasis, 165 ; in bronchitis, 147, 151, 156 ; in laryngeal catarrh, 131, 133 ; in pleurisy, 257 ; in pneumonia, 186, 193 ; in pulmonary cancer, 251 ; in pulmonary emphysema, 176 ; in pulmonary gangrene, 242 ; in pulmonary tuberculosis, 218.

Eye, motor nerves of, paralysis of, 555.

Eye-strain in migraine, 591.

Eyes. See also OCULAR and OPHTHALMIA.

Eyes, conjugate deviation of, in cerebral hæmorrhage, 729.

Eyes, disorders of, in acute bulbar paralysis, 694 ; in acute hydrocephalus, 703 ; in anæmia, 932, 942 ; in cerebral hæmorrhage, 729 ; in cerebral tumors, 751 ; in chorea, 781 ; in chronic hydrocephalus, 768 ; in diabetes, 972 ; in hæmatoma of the dura mater, 697 ; in hemicrania, 592 ; in hysteria, 801 ; in lesions of the central ganglia, 722 ; in lesions of the corpora quadrigemina and crura cerebri, 722 ; in lesions of the occipital cortex, 715 ; in leukæmia, 949 ; in locomotor ataxia, 635, 642 ; in measles, 48 ; in meningitis, 105, 700, 704 ; in miliary tuberculosis, 239 ; in multiple sclerosis, 630 ; in progressive bulbar paralysis, 687 ; in relapsing fever, 35 ; in septicopyæmia, 111 ; in thrombosis of the cerebral sinuses, 707 ; in trichinosis, 122.

Face-ache, Fothergill's, 521.

Facial atrophy, unilateral progressive, 594 ; paralysis, 558 ; ætiology of, 558 ; diagnosis of, 561 ; in acute bulbar paralysis, 694 ; in cerebral hæmorrhage, 731 ; in cerebral tumor, 752 ; in compression of the medulla oblongata, 695 ; in hæmorrhage into the medulla oblongata, 692 ; in progressive bulbar paralysis, 686 ; mimetic, 558 ; symptoms of, 558 ; through cold, 558 ; treatment of, 561 ; varieties, 559.

Facial spasm, clonic, 572 ; in epilepsy, 772 ; in meningitis, 105 ; treatment of, 573 ; spasm, mimetic, 572 ; spasm, tonic, in tetanus, 792.

Fæcal, concretions, 418, 419 ; vomiting, 435.

Fæces, in disease of pancreas, 504 ; in jaundice, 466.

Fainting, 709 ; ætiology of, 709 ; in anæmia, 931 ; in cerebral hæmorrhage, 728 ; in cerebral tumor, 750 ; in epilepsy, 773 ; in insolation, 746 ; in leukæmia, 949 ; in pernicious anæmia, 941 ; liability to, 709 ; symptoms of, 709 ; treatment of, 709.

Falling sickness, 770.

Famine fever, 29.

Farcy, 116 ; acute, 116 ; chronic, 117.

Farcy-buds, 116.

Fascia reflex, 545 ; in cerebral hæmorrhage, 732.

Fat, ingestion of, in diabetes, 979.

Fat-drops in urinary casts, 827.

Fatty acids, crystals of, in the sputum of fœtid bronchitis, 156.

Fatty degeneration, in anæmia, 933 ; of heart, 315 ; of kidneys, 839, 851 ; of liver, 485.

Fatty granular globules in urinary casts, 827 ; heart, 315 ; kidney, 839, 851 ; inflammatory, 839, 851 ; liver, 485, 489 ; liver in pulmonary tuberculosis, 228 ; liver in trichinosis, 123.

Fatty stools, 504.

Febricula, 31.

Febris continua, 7 ; erratica, 94 ; gastrica, 18 ; intermittens, 90, 92 ; nervosa stupida, 14 ; nervosa versatilis, 14 ; quotidiana, 93 ; recurrens, 31.

Feeding, artificial, in progressive bulbar paralysis, 690 ; in stenosis of the œsophagus, 368 ; in trismus, 572.

Fehling's test for sugar, 968.

Fermentation test for sugar, 969.

Fever, anæmic, 387, 934 ; cerebro-spinal, 103 ; "cold," 85 ; enteric, 1 ; gastric, 18 ; hectic, 225 ; hysterical, 804 ; in acute ascending spinal paralysis, 675 ; in acute miliary tuberculosis, 238 ; in angina, 351 ; in appendicitis, 420 ; in arthrogryposis, 576 ; in articular rheumatism, 901 ; in bronchitis, 148 ; in cardiac valvular disease, 301 ; in cerebral abscess, 742 ; in cholera, 85 ; in cirrhosis of the liver, 481 ; in cystitis, 895 ; in dengue, 99 ; in diabetes, 971 ; in diphtheria, 68 ; in dysentery, 79 ; in endocarditis, 278 ; in erysipelas, 63 ; in gastric catarrh, 373 ; in glanders, 117 ; in gout, 986 ; in hæmoglobinæmia, 954 ; in hepatitis, suppurative, 477 ; in infantile cerebral paralysis, 744 ; in infantile spinal paralysis, 668 ; in influenza, 76 ; in intermittent fever, 93 ; in intestinal catarrh, 406 ; in measles, 48 ; in meningitis, 103, 700, 704 ; in noma, 347 ; in osteomalacia, 927 ; in parotitis, 348 ; in pericarditis, 323 ; in perinephritis, 870 ; in pleurisy, 257 ; in pneumonia, 186, 192 ; in poliomyelitis of adults, 671 ; in primary multiple neuritis, 582 ; in pulmonary emphysema, 179 ; in pulmonary gangrene, 243 ; in pulmonary tuberculosis, 225 ; in purpura hæmorrhagica, 962 ; in purpura rheumatica, 962 ; in pyelitis, 883 ; in pylephlebitis, suppurative, 501 ; in rachitis, 922 ; in relapsing fever, 33 ; in remittent fever, 33 ; in scarlet fever, 37 ; in septico-pyæmia, 111 ; in small-pox, 55 ; in sunstroke, 747 ; in thermic fever, 747 ; in trichinosis, 123 ; in tuberculosis of the genito-urinary organs, 889 ; in typhlitis, 419, 420 ; in typhoid fever, 6 ; in typhus fever, 30 ; in yellow fever, 101.

Fever, intermittent, 90 ; lung, 189 ; ship, 28 ; splenic,

117; spotted, 28; swamp, 90; typhoid, 1; typho-malarial, 98; yellow, 100.
Fibrinous, bronchitis, 158; pneumonia, 189.
Fibroid disease of heart, 307.
Fibroma of the larynx, 144.
Fifth nerve, paralysis of, 557.
Filaria Bancrofti, 875; sanguinis, 875.
Fish, poisoning from the ingestion of, 1010.
Flapping joints in infantile spinal paralysis, 669.
Flatulence in hysteria, 805; in jaundice, 466.
Flexibility, waxy, 796.
Floating kidney, 876.
Fœtus, endocarditis in, 293.
Follicular catarrh of the intestines, 404.
Foot-baths in asthma, 172.
Forced attitudes, 541.
Forced movements, 541; in disease of the crura cerebelli ad pontem, 725; in paralysis agitans, 785.
Forearm, paralysis of, 564; paralysis of, in amyotrophic lateral sclerosis, 651.
Foreign bodies in the intestinal canal, 432.
Fothergill's face-ache, 521.
Fourth nerve, paralysis of, 556.
Fowler's solution. See ARSENIC.
Fowler's solution in chorea, 783; in endocarditis, 280; in valvular cardiac disease, 303.
Fractures in rachitis, 923; in osteomalacia, 927.
Fremitus, hydatid, 495.
Fresh-air treatment in tuberculosis, 233.
Friction in pericarditis, 324; in peritonitis, 453; in pleurisy, 258.
Friedreich's ataxia, 648.
Friedreich's sign in adherent pericardium, 326.
Frontal convolutions, lesions of the, 715; connected with the centers of speech, 715, 719; connected with the cortical motor centers, 715.
Frontal sinuses, catarrh of the, 126.
Fuchsine in nephritis, 846.
Furunculosis in diabetes, 972, 973, 977; in scarlet fever, 41; in typhoid fever, 17; in typhus fever, 30.

Gait in amyotrophic lateral sclerosis, 651; in locomotor ataxia, 637; in multiple sclerosis, 629, 630; in osteomalacia, 927; in pseudo-hypertrophic muscular paralysis, 659; in rachitis, 923; in spastic spinal paralysis, 665; pseudo-tabetic, 585.
Gall-bladder, dilatation of, 473; forming abdominal tumor, 473.
Gall-stones, 470; diagnosis of, 474; prognosis of, 474; treatment of, 474.
Galloping consumption, 217.
Galvano-cautery in hypertrophic pharyngitis, 360.
Galvano-puncture in aneurism of thoracic aorta, 338.
Ganglia, basal, tumors of, 752.
Ganglia, central, of the brain, lesions of, 721; lesions of, causing hemiplegia, 721, 722; lesions of, causing hemiopia, 722; lesions of, causing post-hemiplegic chorea, 722.
Gangrene, embolic, 242, 248, 300.
Gangrene in diabetes, 971, 973; in ergotism, 1009; in exophthalmic goitre, 597; in pneumonia, 203; in small-pox, 56; in typhoid fever, 17; in typhus fever, 30; local or symmetrical, 588; of the lungs, 241; of the mouth, 347; senile, 333.
Garrod's thread-test for uric acid, 990.
Gastrectasis, 396.
Gastric. See also STOMACH.
Gastric abscess, 383; cancer, 391.
Gastric catarrh, acute, 372; absorption impeded by, 375; chronic, 375; diagnosis of, 379; exciting causes of, 372, 374; gastric juice in, 374; liability to, 374; mucus secreted in, 375; peristalsis in, 375; treatment of, 373, 380.
Gastric crises in locomotor ataxia, 643; fever, 373; hæmorrhage, 388; hæmorrhage in cancer, 392; hæmorrhage in ulcer, 385, 386, 388.
Gastric juice, chemical examination of, 377; hyperacidity of, 382; subacidity of, 378.
Gastric pain in case of ulcer, 385; ulcer, 384; ulcer, clinical forms of, 385; ulcer, peptic, 384; ulcer, round, 384; vertigo, 401.
Gastritis, acute, 372; acute suppurative, 383; chronic, 375; phlegmonous, 383.
Gastro-intestinal symptoms in emphysema, 179.
Gastromalacia, 384 (foot-note).
Gastrorrhagia, 386, 388.
Gastrotomy, 395.
Gastroxynsis, nervous, 401.
Gelsemium, tincture of, in neuralgia, 520; in trigeminal neuralgia, 523.
General paralysis, progressive, 760; ætiology of, 761; depressive form, 764; development of, 762; diagnosis of, 766; hereditary predisposition to, 761; influence of syphilis in, 761; maniacal exaltation in, 763; symptoms of, 761; treatment of, 766.
Genito-urinary organs, tuberculosis of, 888; diagnosis of, 890; treatment of, 890.
German measles, 51.
Giant cells, 212.
Gin-drinker's liver, 478.
Girdle-sensation in locomotor ataxia, 640.
Glanders, 116; acute, 116; chronic, 117; diagnosis of, 117; diagnosis from small-pox, 117; period of incubation in, 116; treatment of, 117.
Glioma of brain, 748, 755.
Globus hystericus, 810.
Glomerulo-nephritis, 841; in scarlet fever, 43.
Glossitis, acute parenchymatous, 345; treatment of, 346.
Glosso-labio-laryngeal paralysis, 685.
Glossy fingers in cervico-brachial neuralgia, 524; in trophic disorders of the nerves, 589.
Glottis, œdema of, 135; in Bright's disease, 136; in nephritis, 854; in small-pox, 56; in scarlet fever, 40; in typhoid fever, 14.
Glottis, openers of, paralysis of, 140.
Glottis, spasm of, 142; in hysteria, 810; in rachitis, 142, 924; in tetanus, 793; treatment of, 794.
Glotzaugenkrankheit, 595.
Gluteal paralysis, 569; reflex, 543.
Glycerine in diabetes, 979; in trichinosis, 123.
Glycogenic function of liver, 974.
Glycosuria, 966; ætiology of, 966; gouty, 988; in bulbar hæmorrhage, 692; in cerebral hæmorrhage; 729; in tetanus, 793.
Gmelin's test, 467.
Goitre, exophthalmic, 595; symptoms of, 595.
Goll, columns of, 510.
Gonorrhœal rheumatism, endocarditis in, 276.
Gout, 984; acute, 989; atypical, 987; chronic, 987; complications of, 988; diagnosis of, 989; ætiology of, 984; Ebstein's theory of, 989; geographical distribution, 985; hereditary influence in, 985; influence of alcohol in, 985; influence of food in, 985; influence of lead in, 985; irregular, 987; morbid

anatomy of, 988 ; nature of, 932 ; symptoms of, 986 ; treatment of, 990.
Graefe's sign, 596.
Grande hystérie, 808 ; contortions in, 808 ; epileptiform paroxysms in, 808 ; statuesque postures and "attitudes of passion" in, 808.
Grandeur, delusions of, in progressive general paralysis, 763.
Grand mal, 771.
Granular kidney, 856.
Graphospasm, 577.
Gravel, renal, 884.
Graves's disease, 595.
Green-sickness, 934. See also Chlorosis.
Green-stick fracture in rickets, 923.
Grinder's rot (pneumonoconiosis), 245.
Grippe, la, 74.
Guarana in habitual headache, 532 ; in hemicrania, 593 ; in intestinal catarrh of children, 417.
Gummata in cerebral syphilis, 749, 757 ; in hepatic syphilis, 490 ; of spinal cord, 609 ; of liver, 490 ; of lungs, 252.
Gums, affections of, in diabetes, 971 ; (blue line on), in lead poisoning, 1005 ; in scurvy, 960 ; in stomatitis, 341 ; in typhoid fever, 12.
Gustatory paralysis, 533.
Gymnastics in articular rheumatism (chronic), 916 ; in diabetes, 980 ; in gout, 990 ; in infantile spinal paralysis, 671 ; in neurasthenia, 817 ; in progressive muscular atrophy, 658 ; in writer's cramp, 579.

Habitus, apoplectic, 726; emphysematous, 177 ; erethitic, 1000 ; phthisical, 221.
Hæmatemesis, diagnosis from hæmoptysis, 388.
Hæmatemesis, hysterical, 804 ; in acute yellow atrophy, 487 ; in gastric ulcer, 386.
Hæmatemesis in leukæmia, 949 ; in scurvy, 959.
Hæmatidrosis, 590.
Hæmato-chyluria, parasitic, 875.
Hæmatogenous jaundice, 490, 956.
Hæmatoidine, granules of, in urinary casts, 827.
Hæmatoma of the dura mater, 696 ; apoplectic symptoms in, 697 ; diagnosis of, 698 ; in chronic alcoholism, 697 ; in connection with the hæmorrhagic diathesis, 697 ; in progressive general paralysis, 697 ; origin of, 696 ; seat of, 696 ; treatment of, 698.
Hæmatomyelia, 604.
Hæmatorrhachis, 603.
Hæmatothorax, 271.
Hæmatozoa of malaria, 91.
Hæmaturia, 828 ; as a sign of scurvy, 959 ; endemic, of Egypt, 874, 875 ; in acute nephritis, 842 ; in chyluria, 875 ; in renal calculus, 886 ; in renal cancer, 873 ; in tuberculosis of kidney, 889 ; in tumor of the kidney, 873 ; tropical, 875.
Hæmine test, 392.
Hæmoglobin, diminution of, in chlorosis, 935.
Hæmoglobinæmia, 953 ; ætiology of, 953 ; in connection with malaria, 953 ; in connection with syphilis, 956 ; in poisoning from mushrooms, 953 ; paroxysmal, 954, 955 ; symptoms of, 954.
Hæmoglobin, granules of, in the urine, 954.
Hæmoglobinuria, 953 ; in poisoning from mushrooms, 953 ; in Raynaud's disease, 954 ; paroxysmal, 954 ; toxic, 953.
Hæmolysis, in pernicious anæmia, 944 ; in toxic hæmoglobinuria, 954.
Hæmopericardium, 330.
Hæmophilia, 963 ; ætiology of, 963 ; complications of, 965 ; connected with anæmia, 965 ; prognosis of, 965 ; rudimentary forms of, 964 ; treatment of, (*a*) prophylactic, 965 ; (*b*) surgical, 965.
Hæmoptysis, at onset of phthisis, 210 ; causes of, 218 ; hysterical, 804 ; in aneurism, 337 ; in bronchial catarrh, 147, 152 ; in pneumonia, 193 ; in pulmonary gangrene, 243 ; in pulmonary tuberculosis, 218 ; in scurvy, 959 ; periodic, 96 ; treatment of, 235.
Hæmorrhage, cerebral, 725 ; in acute yellow atrophy, 487 ; in anæmia, 934, 939, 942 ; in cirrhosis of the liver, 480 ; in contracted kidney, 861 ; in endocarditis, 279 ; in epileptic paroxysms, 773 ; in hæmophilia, 963 ; in hysteria, 804 ; in leukæmia, 950 ; in malaria, 96 ; in malignant pustule, 119 ; in meningitis, epidemic cerebro-spinal, 104 ; in nephrolithiasis, 888 ; in pseudo-leukæmia, 952 ; in pyelitis, 833 ; in purpura hæmorrhagica, 963 ; in scarlet fever, 41 ; in scurvy, 958 ; in small-pox, 57 ; into spinal cord, 604 ; into the spinal meninges, 603 ; in syphilis of the rectum, 425 ; into ventricles of brain, 727 ; in typhoid fever, 10 ; in yellow fever, 102 ; pulmonary, 235.
Hæmorrhagic diathesis, 963.
Hæmorrhoids, 428 ; "attacks of," 428 ; bleeding from, 428 ; treatment of, 429.
Hair, loss of, in typhoid fever, 17 ; in unilateral facial atrophy, 594.
Hallucinations in hysteria, 807.
Halo surrounding the pocks of small-pox, 53.
Handwriting in general paresis, 763.
Hay fever, 125.
Headache, habitual, 530 ; in acute ascending spinal paralysis, 674 ; in anæmia, 932 ; in bronchitis, 148 ; in bulbar paralysis, acute, 694 ; in cerebral abscess, 742 ; in cerebral anæmia, 709 ; in cerebral hæmorrhage, 728 ; in cerebral syphilis, 759 ; in cerebral tumors, 750 ; in chlorosis, 935 ; in compression of the medulla oblongata, 695 ; in diabetes, 967, 973 ; in diseases of the cerebellum, 724 ; in epilepsy, 773 ; in exophthalmic goitre, 596 ; in hæmatoma of the dura mater, 697 ; in infantile spinal paralysis, 668 ; in leukæmia, 949 ; in meningitis, epidemic, 104 ; in meningitis, purulent, 700 ; in meningitis, tubercular, 703, 705 ; in nervous dyspepsia, 401 ; in neurasthenia, 815 ; in paroxysmal hæmoglobinuria, 954 ; in pernicious anæmia, 941 ; in pleurisy, 257 ; in pneumonia, 192 ; in poliomyelitis of adults, 671 ; in primary multiple neuritis, 583 ; in railway spine, 820 ; in small-pox, 53 ; in tetanus, 792 ; in thrombosis of the cerebral sinuses, 707 ; in typhoid fever, 5 ; in uræmia, 831 ; in writer's cramp, 578.
Headache, nervous, 530 ; sick, 591.
Head, deviation of, in cerebral hæmorrhage, 729 ; in progressive general paralysis, 761 ; rheumatism in, 918 ; sense of pressure in, in neurasthenia, 815.
Head, tetanus of the, 792.
Heart, aneurism of, 308 ; arrhythmia of, in valvular disease, 297 ; congenital affections of, 293.
Heart, dilatation of, 312 ; in chlorosis, 935 ; in malaria, 94.
Heart, disorders of, in diabetes, 972 ; in obesity, 995 ; in pneumonia, 197 ; in scurvy, 959.
Heart, displacement in pleuritic effusion, 259 ; displacement in pneumothorax, 269 ; fatty degeneration of, 315.
Heart, hypertrophy of, 312 ; diagnosis of, 314 ; idio-

pathic, 312; in arterio-sclerosis, 332; in obesity, 996; in renal disease, 834, 843, 853, 859; in scarlet fever, 43; treatment of, 316.
Heart, indurated degeneration of, 308; in exophthalmic goitre, 595; infarctions in, 308.
Heart, lesions of, compensated, 282; congenital, 293; lungs altered by disease of, 249.
Heart, overexertion of, 312.
Heart, palpitation of, diagnosis of the nervous form of, 319; in cardiac valvular disease, 296; in case of tapeworm, 443; in hysteria, 810; in neurasthenia, 816; in obesity, 995; in pernicious anæmia, 941; in scurvy, 958; nervous, 319; treatment of the nervous form of, 320.
Heart, parenchymatous degeneration of, 307, 315; rupture of, in cardiac aneurism, 308; sclerosis of, 307.
Heart, valvular disease of, 281; complications of, 295; multiple, 294; prognosis of, 301; treatment of, 303.
Heart, weakened, 312.
Heartburn in chronic gastric catarrh, 376.
Heart failure, in diphtheria, 69; treatment of, in typhoid fever, 26.
Heat, exhaustion, 746; stroke, 746.
Hebrews, prevalence of diabetes among, 967.
Hectic fever, 225.
Hegar's funnel for rectal irrigation, 410.
Heller's test for blood in the urine, 828.
Helminthiasis, 440.
Hemianæsthesia, in cerebral hæmorrhage, 773; in hysteria, 801, 813; in lesions of the internal capsule, 722, 725; in tumors of the cerebral hemispheres, 752; in unilateral cord lesions, 683.
Hemianopia, in cerebral hæmorrhage, 733; in hemicrania, 592; in lesions of the central ganglia, 722, 725; in lesions of the corpora quadrigemina, 722, 725; in lesions of the occipito-cortical region, 716, 725; in migraine, 592; in tumors of the base of the brain, 752; in tumors of the cerebral hemispheres, 752.
Hemiathetosis, post-hemiplegic, 788.
Hemiatrophy, progressive facial, 594; loss of hair in, 594; seat of, 594; treatment, 594.
Hemichorea, post-hemiplegic, 734; in infantile cerebral paralysis, 745; in lesions of the internal capsule, 721, 725; in lesions of the optic thalamus, 722, 725.
Hemicrania, 591 (see also MIGRAINE); course of, 592; in diabetes, 973; ophthalmic, 592; paralytic, 592; spastic, 592; symptoms of, 592; treatment of, 593.
Hemiplegia, 536.
Hemiplegia, crossed, 692; in cerebral hæmorrhage, 731, 732, 735; in cerebral syphilis, 758; in diffuse cerebral sclerosis, 744; in hæmatoma of the dura mater, 697; in hysteria, 803; in infantile cerebral paralysis, 744; in lesions of the central ganglia, 721; in lesions of the crura cerebri, 723, 725; in lesions of the internal capsule and centrum ovale, 721; in lesions of the motor cortical region, 712; in locomotor ataxia, 645; in multiple sclerosis, 631; in progressive general paralysis, 764; in purulent meningitis, 700; in tumors of the cerebral hemispheres, 752; mental defects in, 735.
Hepatic. See also LIVER.
Hepatic abscess, 476; in dysentery, 79; in pylephlebitis, suppurative, 501.
Hepatic colic, 470; pulse, 289; vein, affections of, 500, 502.
Hepatitis, chronic diffuse interstitial, 478; diffuse syphilitic, 491; suppurative, 476.
Hepatization of the lungs, 190.
Hepatogenous jaundice, 464.
Heredity in diabetes insipidus, 982, 983; in hæmophilia, 963; in idiopathic muscular atrophy, 659, 661; in tuberculosis, 210.
Herpes in acute gastric catarrh, 373; in cerebro-spinal meningitis, 106; in facial paralysis, 560; in intercostal neuralgia, 525; in locomotor ataxia, 644; in malaria, 94; in neuralgia, 517; in neuritis, 582; in pressure paralysis of the spinal cord, 612; in sciatica, 526; in trigeminal neuralgia, 522.
Herpes labialis in angina, 352; in intestinal catarrh, 406; in malaria, 94; in meningitis, epidemic cerebro-spinal, 106; in pneumonia, 192; in relapsing fever, 32; in scarlet fever, 41; in typhoid fever, 17; in typhus fever, 30.
Herpes zoster, 525.
Hiccough, 577; hysterical, 577; in anæmia, 932; in cholera, 85; in dysentery, 79; in hepatitis, suppurative, 477; in uræmia, 832; reflex, 577, treatment of, 577.
Hoarseness in diphtheria, 68; in laryngeal catarrh, 132; in trichinosis, 122.
Hodgkin's disease, 951; morbid anatomy of, 952; symptoms of, 952.
Hot Springs of Arkansas, 916.
Hyaline casts in urine, 826.
Hydatid disease, 494.
Hydatid thrill, 495.
Hydræmia, 825.
Hydrocephaloid symptoms, 411.
Hydrocephalus, acute, 702, 703; ætiology of, 702; chronic, 767; chronic, after cerebro-spinal meningitis, 107; congenital, 767; congenital, diagnosis of, 768; course of, 767; effect of, upon shape of brain, 767; enlargement of head caused by, 768; following epidemic cerebro-spinal meningitis, 107; of adults, 768; skull in, different from the rachitic, 769; spurious, 411; symptoms of, 767, 768; the fluid of, 767; treatment, 769.
Hydrochloric acid in anæmia and chlorosis, 938; in gastric catarrh, 373, 381; poisoning from, 1003; tests for, in gastric juice, 378.
Hydrocyanic acid, poisoning from, 1007.
Hydromyelus, 677, 678.
Hydronephrosis, 890; congenital, 891; intermittent, 891.
Hydropericardium, 329.
Hydroperitoneum, 460.
Hydrophobia, 113; diagnosis of, 114; hydrophobic stage, 114; maniacal stage of, 113; paralytic stage of, 113, 114; period of incubation in, 114; quiet form of, 113; raving form of, 113; treatment of, 114, 115.
Hydrops vesicæ felleæ, 473.
Hydrorrhachis, 679.
Hydrothorax, 271; in scarlet fever, 42; in valvular cardiac disease, 299; treatment of, 271.
Hyoscyamine in paralysis agitans, 787.
Hyperacusis in facial paralysis, 559.
Hyperæmia, 708.
Hyperæsthesia, 505; in ataxia, 640; in hysteria, 802; in myelitis, 619; in neuralgia, 517; in neurasthenia, 815; in pulmonary tuberculosis, 226; in relapsing fever, 32; in spinal meningitis, 600; in typhoid

fever, 16 ; in unilateral lesions of the spinal cord, 683 ; of smell, 532 ; of taste, 533.
Hyperidrosis, unilateral, 590.
Hyperosmia, 532.
Hyperpyrexia, hysterical, 804, 805 ; in rheumatic fever, 905 ; in scarlet fever, 38 ; in sunstroke, 747 ; in tetanus, 793.
Hypnotism in hysteria, 809.
Hypochondriasis and neurasthenia, 815 ; in gastric catarrh, 379 ; in habitual constipation, 430; in nervous dyspepsia, 401.
Hypodermic syringe in diagnosis of pleural effusion, 263, 265.
Hypoglossal nerve, paralysis of, 752 ; spasm of, 573.
Hypoglossus, paralysis of, in cerebral tumor, 752.
Hypophysis, tumor of, 752, 754.
Hypoplasia of aorta, 929.
Hypostatic congestion in typhoid fever, 12.
Hysteria, 755 ; ætiology of, 755 ; artificial production of spasms in, 802, 810 ; contractures and spasms in, 803 ; convulsive forms of, 806 ; course of, 810 ; diagnosis of, 811 ; disorders of sensation in, 800 ; fever in, 804 ; grand paroxysms of, 808 ; hæmoptysis in, 804 ; in connection with floating kidney, 877 ; joint affections in, 529, 804 ; liability to, 799 ; mental symptoms of, 805 ; metallotherapeutics in, 813 ; paralysis in, 803 ; produced by sexual influences, 800 ; special senses in, 800 ; stigmata in, 800 ; symptoms of, 800 ; traumatic, 798 ; treatment of, 811 ; visceral manifestations of, 805 ; with regard to exophthalmic goitre, 596.
Hysterical insanity in typhoid fever, 15.
Hystero-epilepsy, 106, 107.
Hysterogenic points, 802, 810.

Ice in cerebral hæmorrhage, 736 ; in infantile spinal paralysis, 670 ; in injuries of the spinal cord, 607 ; in meningitis, 97, 701, 706 ; in spinal apoplexy, 605 ; in spinal meningeal hæmorrhage, 603.
Ichthyosis linguæ et oris, 346.
Icteric casts in the urine, 467.
Icterus, acute febrile, 469 ; catarrhal, 464 ; gravis, 489 ; neonatorum, 490.
Idiocy in infantile hemiplegia, 745.
Idiopathic anæmia of Addison, 878.
Ileo-cæcal region, gurgling in, in typhoid fever, 10.
Ileo-cæcal tumor, 419.
Ileotyphus, 1.
Ileus from intestinal obstruction, 435 ; in typhoid fever, 11 ; paralytic, 432.
Illuminating gas, poisoning from, 1007.
Imbecility in infantile hemiplegia, 745 ; in multiple sclerosis, 630.
Imitation in chorea, 780.
Impotence in diabetes, 972 ; in locomotor ataxia, 643.
Inanition in nervous dyspepsia, 401, 402.
Incarceration, internal, of the intestines, 433 ; symptoms of, in case of floating kidney, 877.
Incontinence, nocturnal, of urine, 898.
Incoördination of arms, 542 ; of legs, 542.
Indican, test for, 436.
Induction of premature delivery, in chorea gravidarum, 784.
Induration, brown, of the lungs, 249 ; rheumatic, of the muscles, 917.
Infantile convulsions, 779.
Infantile paralysis, cerebral, 744 ; acute, 744 ; course of, 745 ; pathology of, 745 ; treatment of, 745.
Infantile paralysis, spinal, 667. See SPINAL PARALYSIS OF CHILDREN.
Infarction, embolic, of the kidneys, 871 ; hæmorrhagic, of the lungs, 247.
Influenza, 74 ; diagnosis of, 77 ; ætiology of, 74 ; symptoms of, 75 ; treatment of, 77.
Inhalation-pneumonia, 185, 434.
Inhalations in bronchitis, 154 ; in diphtheria, 71 ; in laryngitis, 132 ; in pharyngitis, 359 ; in pulmonary gangrene, 244 ; in pulmonary tuberculosis, 231 ; in whooping-cough, 163.
Injection, intravenous, of salines in diabetes, 981 ; subcutaneous, of salines in cholera, 89.
Injury, mechanical, due to chorea, 783 ; due to epilepsy, 773.
Inoculation, against small-pox, 52, 58 ; protective, in hydrophobia, 115.
Insanity, post-febrile, 15 ; relation of heart disease to, 301.
Insects, the sting of, a cause of malignant pustule, 119.
Insolation, 746.
Inspection of meat, governmental, to prevent trichinosis, 123.
Insufficiency, valvular, 281.
Insufflation of powders in chronic pharyngitis, 359.
Insular sclerosis, 627.
Intention tremor in multiple sclerosis, 629 ; in myelitis, 619.
Intercostal neuralgia, 524.
Intermeningeal apoplexy, 696.
Intermittent fever, 92 ; hepatic, 94 ; masked, 95 ; pernicious, 94 ; tertian, 93.
Internal capsule, lesions of, 721.
Intestinal calculi, 432 ; casts, 408.
Intestinal catarrh, 403 ; acute, 408 ; chronic, 408 ; follicular, 404 ; from the ingestion of poisons, 403 ; in rachitis, 924 ; of children, 413 ; treatment of, 409, 415.
Intestinal glands enlarged in leukæmia, 948 ; in pseudo-leukæmia, 952.
Intestinal hæmorrhage in dysentery, 79 ; in intussusception, 437 ; in pylethrombosis, 502 ; in typhoid fever, 10 ; in ulcer of duodenum, 423 ; in ulcer of the stomach, 386.
Intestinal parasites, 440 ; polypi, 434.
Intestinal symptoms in cholera, 84 ; in dysentery, 78 ; in erysipelas, 63, 64 ; in gastric catarrh, 373, 379 ; in glanders, 116 ; in malignant pustule, 119 ; in measles, 49 ; in pulmonary gangrene, 243 ; in pulmonary tuberculosis, 227 ; in purpura hæmorrhagica, 962 ; in typhoid fever, 9, 10.
Intestine, cancer of, 426 ; diagnosis of, 426 ; treatment of, 427.
Intestine, compression of, from without, 434 ; constriction of, 433 ; dilatation of, 435 ; incarceration of, 433 ; intussusception of, 433 ; invagination of, 433 ; new growths in, 426, 432.
Intestine, obstruction of, 437 ; by enteroliths, 432 ; by foreign bodies, 433 ; by gall-stones, 432.
Intestine, perforation of, in typhoid fever, 11 ; strangulation of, 433 ; strictures and tumors of, 432 ; twists and knots of, 433 ; ulcers of, 432.
Intestine, large, catarrh of, 407 ; desquamative, 408.
Intussusception, 434.
Inunction, mercurial, in acute ascending spinal paralysis, 676 ; in acute bulbar paralysis, 695 ; in cerebral syphilis, 759 ; in hæmoglobinuria, 956 ; in loco-

motor ataxia, 647 ; in myelitis, 626 ; in progressive general paralysis, 767 ; in spastic spinal paralysis, 667 ; in tumors of the brain, 775 ; in tumors of the spinal cord, 677.
Invagination, 434.
Iodide of iron in peritonitis, chronic, 460 ; in pressure paralysis of the spinal cord, 614 ; in scrofula, 1001.
Iodide of potassium in acute ascending spinal paralysis, 676 ; in aneurism of the thoracic aorta, 338 ; in asthma, 172 ; in bronchitis, 155, 159 ; in cerebral syphilis, 759 ; in cerebral tumors, 755, 756 ; in chronic hydrocephalus, 769 ; in gout, 992 ; in habitual headache, 531 ; in lead paralysis, 570 ; in leptomeningitis, spinal, 601 ; in locomotor ataxia, 647, 648 ; in meningitis, cerebro-spinal, 108 ; in multiple sclerosis, 631 ; in myelitis, 626, 627 ; in neuralgia, 520 ; in pachymeningitis cervicalis hypertrophica, 603 ; in peritonitis, chronic, 460 ; in sciatica, 527 ; in spasm of the trigeminus, 572 ; in spastic spinal paralysis, 667 ; in syphilitic paralysis of the motores oculi, 557 ; in tumor of the spinal cord, 677.
Iodine coryza, 125.
Iodine, poisoning from, 1004.
Iodine, tincture of, externally, in articular rheumatism, chronic, 915 ; in chronic nasal catarrh, 128 ; in pharyngitis, chronic, 359 ; in pleurisy, 264 ; in pressure paralysis of the spinal cord, 614 ; in spinal leptomeningitis, 601.
Iodine, tincture of, internally, in diabetes, 981 ; in scrofula, 1001.
Iodoform in diabetes, 981 ; in pleurisy, 264 ; in pseudo-leukæmia lymphatica, 953.
Ipecacuanha in dysentery, 80.
Iritis in gout, 987.
Iron baths in anæmia and chlorosis, 938 ; in infantile spinal paralysis, 671 ; in locomotor ataxia, 647 ; in railway spine, 821.
Iron, chloride of, in purpura hæmorrhagica, 962 ; reaction caused by, in diabetic urine, 970.
Iron in anæmia and chlorosis, 937 ; in cardiac valvular disease, 303 ; in exophthalmic goitre, 598 ; in habitual headache, 531 ; in hemicrania, 593 ; in nephritis, 855 ; in neurasthenia, 818 ; in osteomalacia, 927 ; in paralysis of the muscles of the larynx, 143 ; in pernicious anæmia, 950 ; in pulmonary tuberculosis, 235 ; in rachitis, 924 ; in scurvy, 961 ; in scrofula, 1001.
Irradiation, 517.
Irrigation in intestinal catarrh, 410 ; in the intestinal catarrh of children, 417.
Ischuria in hysteria, 805.
Itching in jaundice, 465 ; in uræmia, 833.

Jacksonian epilepsy, 714.
Japan, beri-beri in, 582.
Jaundice, catarrhal, 464 ; choluria in, 467 ; diagnosis of, 468 ; epidemic form of, 464 ; febrile, 469 ; from gall-stones, 472 ; hæmatogenous, 490 ; hepatogenous, 464 ; in acute phosphorus poisoning, 1006 ; in acute yellow atrophy, 486, 487 ; in cancer of the liver, 493 ; in cancer of the pancreas, 504 ; in cirrhosis of the liver, 480, 484 ; in diabetes, 971 ; in gastric catarrh, 373 ; in gastro-duodenitis, 464 ; in hæmoglobinuria, 954 ; in hepatic colic, 472 ; in hepatitis, suppurative, 477 ; in pneumonia, 202 ; in pylephlebitis, suppurative, 501 ; in syphilis of the liver, 491 ; in Weil's disease, 469 ; in yellow fever, 101 ; malignant, 489 ; of the new-born, 490 ; pernicious, 489 ; treatment of, 468 ; xanthelasma in, 465.
Jaw. See MAXILLA.
Joints, disorders of, in acute neuritis, 583 ; in cerebral hæmorrhage, 735 ; in dengue, 99 ; in endocarditis, 279 ; in erysipelas, 64 ; in gout, 986, 992 ; in hæmophilia, 965 ; in locomotor ataxia, 644 ; in meningitis, cerebro-spinal, 106 ; in pernicious anæmia, 943 ; in purpura hæmorrhagica, 962 ; in scrofula, 1000 ; in scurvy, 959 ; in septico-pyæmia, 111 ; in small-pox, 56 ; in typhoid fever, 17 ; in valvular cardiac disease, 301.
Joints, neuroses of, 528.
Joints, sensibility of, 509.
Jumpers, 576.
"June cold," 125 (foot-note).

Keratitis, in small-pox, 56.
Kidney, abscess of, 868 ; amyloid or lardaceous disease of, 864 ; anomalies in position of, 876 ; calculus of, 884 ; cancer of, 872 ; circulatory disturbances in, 871 ; cirrhosis of, 856 ; congestion of, 871 ; cystic disease of, 874, 890 ; echinococcus of, 874 ; genuine contracted, 856 ; gouty, 857, 987 ; granular, 856 ; hydatids of, 874 ; in diabetes, 972 ; in diphtheria, 70 ; in hæmoglobinuria, 955 ; in leukæmia, 947 ; in obesity, 996 ; in pneumonia, 197 ; in pseudo-leukæmia, 952 ; in pulmonary emphysema, 179 ; in pulmonary tuberculosis, 228 ; in scarlet fever, 41 ; in septico-pyæmia, 112 ; large red, 850 ; large white, 851 ; movable, 876 ; new growths of, 872 ; parasites of, 874 ; pelvis of, dilatation of, 890 ; pelvis of, inflammation of, 881 ; sclerosis of, 856 ; secondary contracted, 849.
Kidney, removal of, for cancer, 873.
Kidney, rhabdo-myoma of, 872 ; sarcoma of, 872 ; surgical kidney, 868 ; tuberculosis of, 888 ; tumors of, 872.
Knee-jerk, loss of, in ataxia, 641 ; in diphtheria, 70.
Knee phenomenon, 544. See also PATELLAR REFLEX.
Koumyss as a food in pulmonary tuberculosis, 232.
Kousso for tape-worm, 444.
Kyphosis in osteomalacia, 926 ; in rachitis, 923.

Lactic acid in diabetes, 981 ; in nephrolithiasis, 888 ; in the bones in osteomalacia, 926 ; in the urine in osteomalacia, 927.
Laennec's cirrhosis, 478.
Lagophthalmus in facial paralysis, 559.
Lancinating pains in locomotor ataxia, 640.
Landry's paralysis, 674.
Lardaceous liver, 498 ; in pulmonary tuberculosis, 228.
Lard, inunction of, in scarlet fever, 45.
Laryngeal crises in locomotor ataxia, 643.
Laryngitis, acute, 130 ; chronic, 132 ; hypoglottica acuta gravis, 131 ; hypoglottica chronica hypertrophica, 133 ; in measles, 48.
Laryngitis, phlegmonous, 134 ; treatment of, 135.
Larynx, abscess of, 134 ; affected by typhoid fever, 13 ; cancer of, 145 ; disturbances of sensibility in, 143 ; examination of, 130 ; muscles of, paralysis of, 139 ; in acute bulbar paralysis, 694 ; in progressive bulbar paralysis, 68[illegible] ; treatment of, 142 ; muscles of, spasm of, in hysteria, 807 ; new growths in, 144 ; treatment of, 145 ; polypi of, 144 ; stenosis of,

chronic, 133; in diphtheria, 68; in laryngitis, acute, 131; tuberculosis of, 136; diagnosis of, 137; treatment of, 138.
Lateral sclerosis, amyotrophic, 650; diagnosis of, 653; involving the medulla oblongata, 652; symptoms and course of, 651; treatment of, 653.
Laughter, spasmodic, 577.
Lead colic, 1005.
Lead paralysis, 569; bilateral, 570; localization of, 570; treatment of, 570.
Lead poisoning, chronic, 1005; chronic, related to contracted kidney, 856, 1005; chronic, related to gout, 985, 1005.
Leptomeningitis, cerebral purulent, 698; tubercular, 702.
Leptomeningitis, chronic spinal, 601; primary, 601; secondary, 601; symptoms of, 601; treatment of, 601.
Lesions, cerebral, topical diagnosis of, 710.
Lethargy in hysteria, 809.
Leukæmia, 945; ætiology of, 945; complications, 949; diagnosis of, 950; lymphatic, 947, 950; myelogenous, 946; splenic, 946, 950; symptoms of, 947; treatment, 950; with regard to anæmia, 949.
Leukocythæmia, 945.
Leukocytosis, 945.
Lids, spasm of, 572.
Lime in osteomalacia, 927; in poisoning from oxalic acid, 1004; in rachitis, 925.
Lingua geographica in glossitis, 346.
Lips, atrophy of, in amyotrophic lateral sclerosis, 652; in progressive bulbar paralysis, 686.
Lithium, carbonate of, in nephrolithiasis, 887.
Lithium water in gout, 991.
Liver. See also HEPATIC.
Liver, anomalies in the shape and position of, 499; atrophy of, 498; atrophy of, acute yellow, 485; atrophy of, acute yellow, diagnosis of, 488; atrophy of, acute yellow, treatment of, 489.
Liver, cancer of, 492; diagnosis of, 492; prognosis of, 494; secondary to cancer of stomach, 493; treatment of, 494.
Liver, cirrhosis of, 478; biliary, 483; diagnosis of, 482, 485; hypertrophic, 483; prognosis of, 482; treatment of, 482, 485.
Liver, disturbances of circulation in, 496; gin-drinker's, 478; granulated, 479; hydatids of, 494; hyperæmia of, 496, 497; hypertrophy of, 498; in gout, 987; in hæmoglobinuria, 954, 956; in jaundice, 468; in leukæmia, 947; in pseudo-leukæmia, 952; in pulmonary emphysema, 179; in pulmonary tuberculosis, 228; in rachitis, 924; in yellow fever, 101; lobulated 479; syphilis of, 490.
Lobelia, tincture of, in asthma, 173.
Locality, testing the sense of, 506.
Lock-jaw, 791.
Locomotor ataxia, 632; ataxic stage of, 636; development of, 632; diagnosis and prognosis of, 645; Friedreich's form of, 648; hereditary predisposition to, 632; histology of, 632; in chronic ergotism, 633; initial stage of, 635; involving cranial nerves, 642; symptoms of, 635; terminal stage [illegible], 636; treatment of, 646; with regard to progressive general paralysis, 645.
Loins, pains in, in relapsing fever, 32; in small-pox, 53; in typhus fever, 30.
Lordosis in pseudo-muscular hypertrophy, 660.
Lower extremities, in rachitis, 923; spasm of, 575; spasm of, in amyotrophic lateral sclerosis, 652; spasm of, in tetanus, 792.
Lower jaw. See MAXILLA.
Lumbago, 918.
Lungs, abscesses of, in septico-pyæmia, 112; aplasia of, 181; aplasia of, in kyphoscoliosis, 182; atelectasis of, 181; capillaries of, atrophy of, in emphysema, 175; cancer of, 250; compression of, 181, 259; consumption of, 207; contraction of, 164, 214, 217, 223; disorders of, in diabetes, 976; disorders of, in leukæmia, 947; disorders of, in progressive bulbar paralysis, 687; disorders of, in typhoid fever, 13; echinococcus of, 252; embolic changes in, 247.
Lungs, emphysema of, 173; caused by pertussis, 175; diagnosis of, 179; followed by pulmonary tuberculosis, 179; prognosis of, 179; treatment of, 180.
Lungs, gangrene of, 241; circumscribed, 242; development of, 241; diagnosis of, 244; diffuse, 242; in cancer of œsophagus, 370; liability to, in diabetes, 241; prognosis of, 244; treatment of, 244.
Lungs, hæmorrhage from, 218; increased volume of, 173; induration of, brown, 249; infarctions in, hæmorrhagic, 247; infarctions in, hæmorrhagic, diagnosis and prognosis of, 249; infarctions in, hæmorrhagic, symptoms of, 248; infarctions in, hæmorrhagic, treatment of, 249; inflammation of, 189 (see also PNEUMONIA); œdema of, inflammatory, 183; œdema of, influencing respiration, 183; œdema of, treatment of, 184; pigmentation of, 245; syphilis of, 252; tuberculosis of, 207; tumors of, 250; tumors of, causing symptoms of compression, 251; tumors of, prognosis of, 252; tumors of, treatment of, 252.
Lymph, animal, for vaccination, 59.
Lymph-glands, extirpation of, in pseudo-leukæmia, 953; progressive multiple hypertrophy of, 951.
Lymph-glands, swollen, in dengue, 100; in diphtheria, 67; in erysipelas, 64; in leukæmia, 947, 948, 949, 950; in malignant pustule, 119; in pseudo-leukæmia, 952; in pulmonary cancer, 251; in pulmonary tuberculosis, 228; in scarlet fever, 40; in scrofula, 999, 1001; in typhoid fever, 11.
Lympho-sarcoma, malignant, 951.

Magnesia in gastric catarrh, 374; in sulphuric-acid poisoning, 1003.
Magnet, application of, in hysteria, 814.
Main en griffe, 566.
Maladie des tics convulsifs, 573.
Malaria, 90; diagnosis of, 96; distribution of, 90; germs of, 91; liability to, 92; period of incubation of, 92; treatment of, 96.
Malarial cachexia, 95.
Malarial fever, remittent and continuous, 95.
Malarial neuralgia, 516, 518.
Malarial poison, 90.
Male fern, ethereal extract of, for tape-worm, 444.
Malignant pustule, 117, 119; in animals, 117; bacilli of, 117; diagnosis of, 120; prophylactic inoculation of, 120; spores of, 118; transmission to man, 118; treatment of, 121.
Mal perforant du pied in diabetes, 973; in tabes, 645.
Malum Cotunnii, 526. See SCIATICA.
Mammillary reflex, 544.
Marasmus in diabetes, 971.
Massage in articular neuralgia, 529; in articular rheumatism, 910, 916; in brachial paralysis, 568; after cerebral hæmorrhage, 737; in facial paraly-

sis, 562; in gout, 992; in hysteria, 812; in infantile paralysis (cerebral), 745; in infantile paralysis (spinal), 671; in migraine, 593; in muscular rheumatism, 919; in neuralgia, 521; in neurasthenia, 817, 818; in paralysis agitans, 787; in progressive muscular atrophy, 658; in pseudo-hypertrophic muscular paralysis, 663; in sciatica, 527; in scorbutic ecchymoses, 961; in writer's cramp, 579.

Mastication, disturbances of, in progressive bulbar paralysis, 686, 687.

Mastication, muscles of, paralysis of, 557; in general paralysis of the insane, 764; in tumors at the base of the brain, 752; spasm of, clonic, 571; tonic, 571.

Mastodynia, 525; treatment of, 525.

Measles, 46; black, 48; catarrh in 48; complications of, 48; contagiousness of, 46; diagnosis of, 50; diphtheria in, 49; inoculation of, 47; period of incubation in, 47; prognosis of, 50; prophylaxis of, 50; relation of, to pulmonary tuberculosis, 49; relation of, to whooping-cough, 49; treatment of, 50; typhoid, 49.

Measles, German, 51. See RÖTHELN.

Measles in pork, 441. See TÆNIA.

Meat, compulsory inspection of, in trichinosis, 123; poisoning from, 1010.

Meckel's diverticulum in obstruction of the intestine, 433.

Median paralysis, 566; disturbances of the functions of the forearm and hand in, 566; traumatic, 566.

Mediastinal tumors, 273; diagnosis of, 273; prognosis of, 274; treatment of, 274.

Mediastino-pericarditis, 325.

Medulla oblongata, acute apoplectiform paralysis of, 691; compression of, 695; diseases of, 685; progressive paralysis of, 685.

Medulla oblongata and pons, hæmorrhages into, 691; apoplexy from, 691; cysts and scars from, 691; seat and extent of, 691; treatment of, 693.

Melæna neonatorum, 390.

Mellituria, 966. See GLYCOSURIA.

Memory, weakness of, in exophthalmic goître, 596.

Ménière's disease, 769; implication of the semicircular canals in, 769; in tabes, 643; treatment of, 769.

Meningeal apoplexy, 603.

Meningeal hæmorrhage, 603.

Meningeal tumors, 676; different forms of, 676; prognosis and treatment of, 677; symptoms of, 677.

Meningitis, basilar, 702.

Meningitis of the convexity, 698.

Meningitis, epidemic cerebro-spinal, 103, 698, 701 (see also CEREBRO-SPINAL MENINGITIS); diagnosis of, 107; in pneumonia, 107; prognosis of, 107; secondary, 107, 698; sequelæ of, 107; siderans, 104; treatment of, 108.

Meningitis, purulent, 698; ætiological factors in, 698; complications of, 699; diagnosis of, 701; localization of the morbid process in, 699; metastatic, 699; primary, 698; symptoms of, 700; treatment of, 701.

Meningitis, tubercular, 702; causes of, 702; duration of, 705; hæmorrhages into the pia mater in, 703; hydrocephalic effusion into the ventricles in, 703; implication of the spinal cord in, 703; inflammatory changes in, 703; seat of miliary tubercles in, 703; symptoms of, 703.

Meningitis, tubercular, in children, 705; diagnosis of, 706; loud outcry of child in, 705; treatment of, 706.

Meningocele, 679.

Menstruation in chlorosis, 935; influence of, on epilepsy, 774; vicarious, 129.

Mental disturbances in acute general miliary tuberculosis, 239; in anæmia, 931; in articular rheumatism, 906; in athetosis, 789; in bulbar hæmorrhage, 691; in cerebral abscess, 742; in cerebral anæmia, 709; in cerebral embolism, 740; in cerebral hæmorrhage, 728, 730, 735; in cerebral hyperæmia, 710; in cerebral syphilis, 759; in cerebral tumor, 750; in cerebro-spinal meningitis, 105; in cholera, Asiatic, 85; in cholera morbus, 411; in chorea, 780, 782; in convulsions, 541; in cutaneous anæsthesia, 513.

Mental disturbances in diabetic coma, 973; in epilepsy, 770, 772, 773, 775, 777; in fainting attacks, 709; in general paralysis of the insane, 761; in habitual constipation, 430; in hæmatoma of the dura mater, 697; in hydrocephalus, 768; in hysteria, 805, 808; in infantile paralysis (cerebral), 745; (spinal), 668; in jaundice, 466; in meningitis, 700, 703; in multiple sclerosis, 630, 631; in neuralgia, 518; in neurasthenia, 815; in peritonitis, 454; in poliomyelitis of adults, 671; in pseudo-hypertrophic muscular paralysis, 660; in pulmonary tuberculosis, 226; in pylephlebitis, 501; in sinus thrombosis, 707; in typhoid fever, 14, 15; in typhus fever, 30; in uræmia, 831; in valvular heart disease, 301; in writer's cramp, 578.

Mercurialism, chronic, 1005.

Mercurial poisoning, 1005.

Metabolism, defective, in neurasthenia, 816.

Metallic tinkling, 269, 270.

Metalloscopy in hysteria, 813.

Meteorism in hysteria, 805; in intestinal obstruction, 437; in intestinal tuberculosis, 424; in peritonitis, 453; in pulmonary tuberculosis, 227; in typhlitis, 419; in typhoid fever, 9.

Micrococci in cystitis, 894; in endocarditis, 277; in erysipelas, 61, 62; in pneumonia, 189; in septicopyæmia, 109, 110.

Migraine, 591 (see HEMICRANIA); duration of the attacks of, 592; in general paralysis of the insane, 763; treatment of, 593.

Miliary tuberculosis, acute general, 236; cerebral symptoms in, 238; causes of, 236; diagnosis of, 240; intermittent form of, 238; prognosis of, 240; relation of, to tubercular meningitis, 238; symptoms of, 237; treatment of, 240; typhoid form of, 237.

Milk-cure in anæmia, 936; in nephritis, 846; in pulmonary tuberculosis, 232; in pyelitis, 884.

Milker's cramp, 579.

Millar's asthma, 142. See GLOTTIS, SPASM OF.

Mimetic expression in lesions of optic thalamus, 722.

Mineral acids in scurvy, 961.

Mineral springs in anæmia and chlorosis, 938; in bronchitis, 154; in cholelithiasis, 475; in diabetes, 981; in exophthalmic goître, 598; in gastric catarrh, 381; in gout, 991; in habitual headache, 531; in intestinal catarrh, 410; in laryngeal catarrh, 134; in nephrolithiasis, 888; in neuritis, 584; in obesity, 999; in pulmonary emphysema, 180; in pyelitis, 884.

Miserere in intestinal obstruction, 435.

Mitral insufficiency, 283.

Mitral stenosis, 285.

Mogigraphia, 577. See WRITER'S CRAMP.

Monophasia, 718.

Monoplegia, 536; in focal diseases of the centrum ovale, 721; in focal diseases of the motor cortex, 712, 724; in general paralysis of the insane, 764; in meningitis, 700, 704.
Morbilli, 46. See MEASLES.
Morbus Addisonii, 878. See ADDISON'S DISEASE.
Morbus Basedowii, 595. See EXOPHTHALMIC GOÎTRE.
Morbus Brightii, 836. See NEPHRITIS.
Morbus Gravesii, 595. See EXOPHTHALMIC GOÎTRE.
Morbus maculosus Werlhofii, 961. See PURPURA.
Morbus sacer, 770. See EPILEPSY.
Morphine in angina pectoris, 319; in cholelithiasis, 474; in endocarditis, 280; in gastric ulcer, 390; in intercostal neuralgia, 525; in intestinal obstruction, 439; in tabes, 648; in myelitis, 627; in myocarditis, 312; in nephritis, 849; in neuralgia, 520; in occipital neuralgia, 523; in peritonitis, 457; in pleurisy, 264; in pneumothorax, 270; in sciatica, 527; in spasm of the diaphragm, 577. See also OPIUM.
Morphine, poisoning from, 1008.
Morphinism, chronic, 1008.
Morvan's disease, 679.
Mosquitoes, relation of, to chyluria, 876.
Mothers' milk, substitutes for, in feeding children, 416.
Motility, disturbances of, 534; in caisson disease, 608; in chronic hydrocephalus, 768; in tabes, 636; in pressure paralysis of the spinal cord, 611.
Motion, sensations of, 507.
Motor nerves, diseases of, 534; changes of electrical excitability in, 546.
Motor region of the cortex and its focal diseases, 711, 742; centers of different muscular territories in, 711; diagnosis of focal lesions of, 712; relation of hemiplegia to, 712, 725; relation of monoplegia to, 712, 724; relation of symptoms of irritation in different muscular territories to, 713, 714, 724; relation of tonic-clonic spasms to, 713, 714.
Mouth, cavity of, inflammation of, 341; in pulmonary tuberculosis, 227; in typhoid fever, 12.
Mucous hæmorrhoids, 428.
Mucous polypi in the larynx, 144.
Mud-baths in muscular rheumatism, 919; in myelitis, 627; in tabes, 647.
Muguet, 344. See THRUSH.
Mulberry calculi in nephrolithiasis, 885.
Mumps, 348. See PAROTITIS.
Muscles, abscess of, in septico-pyæmia, 108: regeneration of, 554; sensory nerves to, 511; stretching of, in facial paralysis, 562; trichinæ in, 121.
Muscular atrophy in amyotrophic lateral sclerosis, 650, 652, 653; in articular rheumatism, 905; in cerebral hæmorrhage, 735; degenerative, 553, 655; in deltoid paralysis, 564; in infantile paralysis (cerebral), 745; (spinal), 668, 669; in lead paralysis, 570; in myelitis, 622; in neuritis, 583, 584; in pachymeningitis cervicalis hypertrophica, 602; in paralysis, 538; in poliomyelitis in adults, 672, 673; in pressure paralysis of the spinal cord, 612; in progressive bulbar paralysis, 685, 687; in radial paralysis, 565; in syringomyelia, 678; in tabes, 644, 649; in ulnar paralysis, 566; in unilateral lesion of the spinal cord, 684.
Muscular atrophy, progressive, 655; beginning of, 655; causes of, 655; complication of, with progressive bulbar paralysis, 657: diagnosis of, 657; hereditary, 655, 658, 661; juvenile form of, 661; pathological lesion in, 655; symptoms of, 655; treatment of, 658.
Muscular contractures in amyotrophic lateral sclerosis, 652; in cerebral hæmorrhage, 734; in cholera, 85; in Friedreich's ataxia, 649; in hysteria, 803, 809; in infantile paralysis (cerebral), 745; (spinal), 669; in paralysis agitans, 784; in secondary degeneration of the spinal cord, 682.
Muscular degeneration, 553.
Muscular excitability in acute ascending spinal paralysis, 674; in amyotrophic lateral sclerosis, 652; in cerebral hæmorrhage, 732, 735; electrical, 546; in facial paralysis, 560; in hysteria, 809; mechanical, 546; in myelitis, 622; in neuritis, 585; in progressive muscular atrophy, 656; in pseudo-hypertrophic muscular paralysis, 660; in radial paralysis, 565; in spinal infantile paralysis, 669; in tabes, 642; in unilateral lesion of the spinal cord, 684.
Muscular hypertrophy in congenital myotonia, 795.
Muscular pains in cholera morbus, 411; in intestinal catarrh, 406; in muscular rheumatism, 917; in scarlet fever, 43; in typhoid fever, 17.
Muscular rheumatism, 916; acute, 916; chronic, 916; diagnosis of, 918; in hæmophilia, 965; treatment of, 918.
Muscular rigidity in congenital myotonia, 795; in paralysis agitans, 785, 786; in tetanus, 792.
Muscular sense, 509.
Muscular sensibility, abnormal, 510; electro-muscular, 510; in tabes, 636, 639, 641; test of, 509.
Muscular stiffness after cerebral hæmorrhage, 729; in cholera, 87.
Muscular tonus, in spastic spinal paralysis, 664; in tabes, 639.
Mushroom poisoning, 1009.
Musk in spasm of the glottis, 143.
Mussels, poisoning from, 1010.
Mustard in asthma, 172; in pleurisy, 264.
Mutism, hysterical, 803.
Myalgia, cervical, 918; lumbar, 918; rheumatic, 916.
Mycoderma vini in relation to thrush-formation, 344.
Mycosis, intestinal, 119. See MALIGNANT PUSTULE.
Mydriasis in oculo-motor paralysis, 556.
Myelitis, 615; acute bulbar, 694; cervical, 623; diagnosis of diffuse transverse, 625; diffuse, 615, 616; dorsal, 623; lumbar, 623; pathological changes in the spinal cord in, 616; symptoms of, 618; transverse, 615; treatment of, antisyphilitic, 626; by baths, 626; electrical, 626; hygienic and symptomatic, 627.
Myelocele, 679.
Myocarditis, 307; diagnosis of, 311; prognosis of, 311; treatment of, 311.
Myoclonia, 810. See PARAMYOCLONUS MULTIPLEX.
Myodegeneration of the heart, 307.
Myopathy, 663.
Myositis, rheumatic, 916.
Myotonia, congenital, 795; congenital muscular anomalies in, 795.
Myriachit, 576.
Myxœdema, 589; atrophy of thyroid in, 589.

Nails in typhoid fever, 17.
Narcotics in acute bulbar paralysis, 695; in asthma, 172; in bronchitis, 155; in cerebral abscess, 743; in cerebral syphilis, 760; in cystitis, 896; in diabetes, 980; in epilepsy, 778; in habitual headache, 532; in hemicrania, 593; in hiccough, 577; in hysteria, 813; in mastodynia, 525; in meningitis, 108, 702, 706; in nephrolithiasis, 888; in neuralgia, 520; in neu-

rasthenia, 818 ; in neuritis, 584 ; in osteomalacia, 927 ; in palpitation of the heart, 320 ; in pneumonia, 205, 207 ; in progressive bulbar paralysis, 690 ; in pulmonary emphysema, 180 ; in pulmonary tuberculosis, 231 ; in pyelitis, 884 ; in spasm of the cervical muscles, 575 ; in spasm of the glottis, 143 ; in spasm of the trigeminus, 572 ; in spermatic neuralgia, 528 ; in tetanus, 794 ; in trigeminal neuralgia, 523 ; in valvular disease of the heart, 307 ; in whooping-cough, 163.

Nasal catarrh, chronic 126 ; diagnosis of, 127 ; treatment of, 127.

Nasal catarrh in typhus fever, 30.

Nasal douche in diphtheria, 72 ; in nasal catarrh, 128 ; in pharyngeal catarrh, 359.

Naso-pharyngeal catarrh, 357.

Nationality in relation to hysteria, 799 ; to neurasthenia, 815.

Nephritis, acute, 836, 839 ; acute hæmorrhagic, 840 ; acute infectious, 836 ; in articular rheumatism, 837, 906 ; in cholera, 86, 837 ; chronic, 849 ; chronic hæmorrhagic, 849; chronic interstitial, 856 ; chronic parenchymatous, 849 ; in diabetes, 972 ; diagnosis of, 845, 855, 870 ; in diphtheria, 70, 836 ; in endocarditis, 279, 837 ; in epidemic meningitis, 106, 837 ; gravidarum, 838 ; in intestinal affections, 837 ; in measles, 49, 837 ; in pneumonia, 197, 837 ; primary idiopathic acute, 838, 844 ; prognosis of, 845, 855 ; in pulmonary tuberculosis, 228, 837 ; in purpura hæmorrhagica, 962 ; purulent, 868 ; in relapsing fever, 35, 837 ; in rötheln, 837 ; in scarlet fever, 41, 837 ; in scurvy, 959 ; in septico-pyæmia, 112 ; septic, 837 ; in small-pox, 837 ; in sore throat, 837 ; in syphilis, 837 ; in tetanus, 792, 837 ; toxic, 837 ; treatment of, 846, 855, 871 ; in typhoid fever, 17, 837.

Nephritis, chronic and subchronic, 849 ; diagnosis of, 855 ; prognosis of, 855 ; treatment of, 855.

Nephrolithiasis, 884 ; diagnosis of, 887 ; heredity of, 885 ; origin of, 885 ; treatment of, 887.

Nephrophthisis, 888. See TUBERCULOSIS, GENITO-URINARY.

Nerves, atrophy of, in amyotrophic lateral sclerosis, 651 ; degenerative, 553, 589 ; in lead paralysis, 570 ; in locomotor ataxia, 633, 634 ; in paralysis, 539.

Nerves, degeneration of, 538 ; in amyotrophic lateral sclerosis, 651 ; in the lumbar cord in tabes, 639 ; in spinal paralysis of children, 668.

Nerves, excitability of, changes of electrical, 546 ; in facial paralysis, 560 ; in hysteria, 809 ; in neuritis, 583 ; in spinal paralysis of children, 669 ; in tabes, 639 ; in tetany, 790.

Nerves, regeneration of, 533.

Nerve-stretching in cervico-brachial neuralgia, 524 ; in facial spasm, 573 ; ataxia, 648 ; in neuralgia, 521 ; in paralysis agitans, 787 ; in sciatica, 527 ; in spasm of the cervical muscles, 575 ; in tabes, 648 ; in trigeminal neuralgia, 523.

Nervines in palpitation of the heart, 320.

Nervous fever, 14.

Neuralgia, 515 ; in the anæmic, 516 ; in aneurism of the thoracic aorta, 337 ; brachial, 524 ; causes of, 516 ; cervico-brachial, 524 ; in diabetes mellitus, 517, 973 ; dorso-intercostal, 524 ; epileptiform, 522 ; in general paralysis of the insane, 763 ; of the genitals and rectal region, 528 ; in gout, 517 ; in hæmophilia, 965 ; hereditary predisposition to, 516 ; idiopathic, 517 ; intercostal, 524 ; ischiatic, 526 ; lumbar, 526 ; in malaria, 95, 516 ; in neuromata, 586 ; occipital, 523 ; phrenic, 524 ; reflex, 517 ; rheumatic, 516; sciatic, 526 ; spermatic, 528 ; symptomatic, 517 ; syphilitic, 516, 523 ; treatment of, 518 ; trigeminal, 521 ; in typhoid fever, 16.

Neurasthenia, 815 ; causes of, 815 ; course of, 816 ; and diabetes, 976 ; diagnosis and prognosis of, 816 ; disposition to, 817 ; symptoms of, 815 ; traumatic, 821 ; treatment of, 817.

Neurectomy in neuralgia, 520.

Neuritis, 579 ; of alcoholic subjects, 584 ; ascending, 581, 616 ; causes of, 580 ; chronic, 579, 584 ; diagnosis and prognosis of multiple, 584 ; epidemic, 582. hypertrophic, after cerebral hæmorrhage, 735 ; mental symptoms in, 585 ; multiple, 582 ; new formation of connective tissue in, 580 ; nodosa, 580 ; primary simple, 582 ; purulent, 580 ; in relation to primary degenerative atrophy of the nerves, 580 ; secondary, 581 ; spontaneous, 581 ; symptoms and cause of, 581 ; traumatic, 580 ; treatment of, 584, 585.

Neuritis, optic, in acute general miliary tuberculosis, 240 ; in cerebral tumor, 751 ; in chronic hydrocephalus, 768 ; in hæmatoma of the dura mater, 697 ; in meningitis, 700, 704 ; in myelitis, 623.

Neuroma, 585 ; amputation, 586 ; diagnosis of, 586 ; extirpation of, 586 ; false, 585 ; hereditary predisposition to, 586 ; after injuries of the nerves, 586 ; multiple occurrence of, 586 ; symptoms of, 586 ; treatment of, 586 ; true, 585.

Neuroses, articular, 528 ; of the heart, 318 ; of the vagus, 319.

Neurotomy in neuralgia, 520.

Nicotine poisoning, 1008.

Night-sweats in phthisis, 216.

Nitric acid in diabetes insipidus, 984 ; in nephritis, 846 ; poisoning from, 1003.

Nitrites in contracted kidney, 864.

Nitro-benzene poisoning, 1007.

Nitro-glycerine, in contracted kidney, 864 ; in hemicrania, 593.

Nitrous-acid fumes, poisoning from, 1003.

Nocturnal incontinence of urine, 898. See ENURESIS.

Nodding spasm, 574.

Nodules, myocarditic, in valvular disease of the heart, 298.

Noma, 347 ; in measles, 49 ; treatment of, 347.

Nose, affections of, in diphtheria, 68 ; in glanders, 116 ; in measles, 48 ; in scarlet fever, 39, 40 ; in typhoid fever, 14.

Nose-bleed, 129 ; in contracted kidney, 860, 862 ; habitual, 129 ; in nephritis, 843 ; in relapsing fever, 35 ; in typhoid fever, 14 ; as vicarious menstruation, 129.

Nutmeg liver, 497.

Nux vomica in dilatation of the stomach, 400.

Nystagmus in hereditary ataxia, 649 ; in lesions of the corpora quadrigemina, 722 ; in multiple sclerosis, 629 ; in purulent meningitis, 700 ; in sinus thrombosis, 707.

Obesity, 992 ; causes of, 992 ; complications of, 995 ; treatment of, 996.

Obstruction, intestinal, 432, 474 ; tables for diagnosis of, 437.

Obturator paralysis, 569 ; symptoms of, 569.

Occipital lobes, focal diseases of their cortex, 715, 743 ; relation of, to hemiopia, 716, 725 ; relation of,

to soul-blindness, 716 ; seat of the visual sense in the cortex of, 715.
Occipital neuralgia, 523 ; bilateral, 523 ; painful points in, 523.
Ocular nerves in meningitis, 700.
Oculo-motor paralysis, 555 ; in diphtheria, 70 ; diplopia in, 555 ; in lesions of the corpora quadrigemina, 722 ; in lesions of the crura cerebri, 723, 725 ; partial, 556 ; periodical, 557 ; in purulent meningitis, 700 ; in tabes, 642 ; in tumor of the brain, 752.
Œdema, 828.
Œdema, acute angioneurotic, 588 ; in acute ascending spinal paralysis, 674 ; in anæmia, 934 ; in cancer of the stomach, 393 ; in contracted kidney, 860 ; in diabetes, 973.
Œdema in leukæmia, 949 ; in myelitis, 622 ; in nephritis, 842, 852 ; in neuritis, 583 ; in scarlet fever, 42 ; in tetany, 790 ; in trichinosis, 123 ; in typhoid fever, 16 ; in valvular disease of the heart, 299.
Œsophagitis, 361 ; catarrhal, 361 ; corrosive, 362 ; croupous-diphtheritic, 362 ; purulent, 362 ; treatment of, 362.
Œsophagomalacia, 371.
Œsophagus, cancer of, 369 ; complications of, 369 ; metastases of, 370 ; symptoms of, 369 ; treatment of, 370.
Œsophagus, diffuse dilatation of, 363 ; after stenosis of the cardiac orifice, 363 ; symptoms of, 363 ; treatment of, 363.
Œsophagus, dilatation of, 363 ; diseases of, 361.
Œsophagus, diverticula of, 363 ; causes of, 364 ; complications of, 365 ; pressure, 363 ; symptoms of, 364 ; symptoms of compression in, 364 ; traction, 365 ; treatment of, 365.
Œsophagus, paralysis of, 371 ; rupture of, 371 ; spasm of, 371 ; stenosis of, 366 ; auscultation of, 367 ; causes of, 366 ; examination of, by the sound, 367 ; prognosis and treatment of, 368 ; symptoms of, 366.
Oidium albicans, 344.
Oligocythæmia, 928.
Omalgia, 917, 918.
Ophthalmia in diphtheria, 69 ; in exophthalmic goître, 596 ; neuroparalytic, in anæsthesia of the trigeminus, 513 ; in scrofula, 1000.
Ophthalmoplegia, progressive, 690.
Opisthotonos, 541 ; in epilepsy, 772 ; in tetanus, 792.
Opium in Asiatic cholera, 89 ; in cholera morbus, 412 ; in diabetes, 980 ; in gastric ulcer, 396 ; in intestinal catarrh, 409 ; in intestinal catarrh of children, 416 ; in intestinal obstruction, 439 ; in nephritis, 849 ; in neuralgia, 520 ; in peritonitis, 456, 457 ; in tetanus, 794 ; in trigeminal neuralgia, 523 ; in typhlitis, 421 ; in typhoid fever, 25.
Opium habit, 1008 ; poisoning, 1008.
Optic atrophy in chronic hydrocephalus, 768 ; in diabetes, 972 ; in general paralysis of the insane, 763 ; in multiple sclerosis, 630 ; in tabes, 642.
Optic neuritis. See Neuritis.
Orchitis in typhoid fever, 18.
Orthopædics in pressure paralysis of the spinal cord, 614 ; in rachitis, 925 ; in spasm of the cervical muscles, 575 ; in spinal paralysis of children, 671.
Osmic acid in neuralgia, 520.
Osteomalacia, 925 ; diagnosis of, 927 ; examination of the bones in, 926 ; symptoms of, 926 ; treatment of, 927.
Osteomyelitis in septico-pyæmia, 112.
Ovarian neuralgia, 528.
Ovarie in hysteria, 802, 810.
Oxalic-acid poisoning, 1003.
Oxygen inhalations in anæmia, 945 ; in diabetes, 980 ; in leukæmia, 951.
Oxyuris vermicularis, 446 ; diagnosis of, 447 ; treatment of, 447.
Ozæna, 126 ; in scrofula, 1000.

Pachymeningitis cervicalis hypertrophica, 602 ; compression of the spinal cord in, 602 ; development of, 602 ; diagnosis of, 602 ; treatment of, 603.
Pachymeningitis hæmorrhagica, 603, 696 ; development of, 603 ; interna, 603, 696 ; spinalis, 603 ; symptoms of, 603 ; treatment of, 603.
Pain, sensations of, in chorea, 781 ; in chronic hydrocephalus, 768 ; conduction of, 619 ; in cramps, 542 ; in hysteria, 802, 810 ; in paralysis agitans, 786 ; prolonged, 509 ; rheumatoid, in general paralysis, 762 ; in tabes, 659 ; test of, 508.
Palate, inflammation of, 351, 357.
Palate, paralysis of, in compression of the medulla, 695 ; after diphtheria, 70 ; (unilateral) in cerebral hæmorrhage, 732.
Palisade-worm, 875.
Palpitation of the heart, diagnosis of nervous, 319 ; in hysteria, 806 ; nervous, 319 ; in neurasthenia, 816 ; in obesity, 995 ; in pernicious anæmia, 941 ; in scurvy, 958 ; in tape-worm, 443 ; treatment of nervous, 320 ; in valvular disease of the heart, 296.
Palsy, shaking, 784. See Paralysis Agitans.
Pancreas, atrophy of, 503 ; in diabetes, 966, 975.
Pancreas, cancer of, 504 ; diagnosis of, 504 ; symptoms of, 504 ; treatment of, 504.
Pancreas, hæmorrhage of, 503.
Pancreatitis, acute, 503 ; chronic indurated, 504.
Pancreatized meat, enemata of, in stenosis of the œsophagus, 369.
Papilloma of the larynx, 144.
Paquelin's thermo-cautery in noma, 347.
Para-anæsthesia, 512.
Paradoxical contraction, 546.
Paræsthesia, 505 ; in cerebral hæmorrhage, 733 ; in epilepsy, 772 ; in injuries of the spinal cord, 607 ; in neurasthenia, 816 ; in pressure paralysis of the spinal cord, 611 ; in spinal neurasthenia, 606 ; in subacute poliomyelitis, 673 ; in tabes, 639 ; of taste, 533 ; in tumors of the spinal cord, 677.
Paragraphia, 720.
Paraldehyde in neuralgia, 520 ; in neurasthenia, 819.
Paralysie ascendante aigue, 674. See Spinal Paralysis.
Paralysie générale spinale antérieure subaigue, 673. See Spinal Paralysis.
Paralysis, 534 ; acute ascending spinal, 674, 675 ; arsenical, 571 ; in articular rheumatism, 905 ; atrophic, 538, 669 ; bilateral, 536 ; bulbar, 685 ; in bulbar hæmorrhage, 691 ; central, 534 ; cerebral, 536 ; in cerebral hæmorrhage, 729, 731, 732, 733, 734 ; in cerebral syphilis, 758 ; in cerebral tumor, 752, 753 ; in chronic hydrocephalus, 768 ; combined, of the upper extremity, 567 ; condition of the paralyzed muscles in, 539 ; in compression of the medulla, 695 ; cortical, 536 ; of the diaphragm, 568 ; diphtheritic, 70, 537 ; in embolism and thrombosis of the basilar artery, 693 ; of the facial muscles in facial paralysis, 558 ; flaccid, 539 ; forms of peripheral, 555 ; general, 760 ; in general paralysis of the insane, 764 ; glosso-labio-laryngeal, 685 ; in hydrophobia,

114 ; hysterical, 537, 803, 810, 811, 812 ; after infectious diseases, 537 ; after injury to the spinal cord, 607 ; of the laryngeal muscles, 139 ; lead, 569 ; in tabes, 636, 639, 641, 643, 649 ; of the lower extremity, 568 ; median, 566 ; in meningeal hæmorrhage, 603 ; in multiple sclerosis, 629 ; of the muscles of the back, 564 ; in myelitis, 618 ; myopathic, 534 ; in neuralgia, 517 ; in neuritis, 582, 583, 585 ; oculomotor, 555, 556 ; of the œsophagus, 371 ; in pachymeningitis cervicalis hypertrophica, 602 ; peripheral, 536 ; in poliomyelitis of adults, 671, 673 ; in pressure paralysis of the spinal cord, 612, 613 ; in progressive bulbar paralysis, 685, 686 ; pseudo-hypertrophic muscular, 658; from psychical causes, 537 ; from ptomaïnes, 537 ; in purulent meningitis, 700 ; radial, 564 ; reflex, 537 ; refrigeratory, 537 ; rheumatic, 537 ; of the shoulder-muscles, 562 ; spastic, 539 ; in spina bifida, 680 ; spinal, 536, 600, 667, 671 ; in spinal apoplexy, 604 ; in spinal meningitis, 600 ; spinal atrophic of adults, 671 ; spinal, of children, 667 ; in spinal paralysis of children, 668 ; of the stapedius in facial paralysis, 559 ; symptomatology of, 538 ; after syphilis, 537 ; in syringomyelia, 678 ; after tuberculosis, 537 ; toxic, 537, 569 ; traumatic, 537 ; in tumor of the spinal cord, 677; in typhoid fever, 16 ; ulnar, 566 ; unilateral, 536 ; in unilateral lesion of the spinal cord, 683 ; of the upper extremity, 564 ; vaso-motor, 587.

Paralysis agitans, 784 ; development of, 784 ; diagnosis of, 786 ; displacement of the center of gravity in, 785 ; distinction of, from multiple sclerosis, 787 ; excretion in, 786 ; hereditary predisposition to, 784 ; pathology of, 786 ; symptoms of, 784 ; treatment of, 787 ; voice in, 786.

Paramyoclonus multiplex, 576, 810.

Paramyotone, ataxic, 796 ; congenital, 796.

Paraphasia, 718 ; association in, 718.

Paraplegia, 536 ; ataxic, 649 ; in acute ascending spinal paralysis, 674 ; in chronic hydrocephalus, 768 ; dolorosa, 614 ; in myelitis, 619 ; in tabes, 639.

Parietal convolutions, focal diseases of, 715 ; relation of, to cutaneous and muscular sensibility, 715.

Parkinson's disease, 784. See PARALYSIS AGITANS.

Parotitis, 348 ; contagiousness of, 348 ; diagnosis of, 348 ; duration of stage of incubation of, 348 ; metastatic, 349 ; primary, 348 ; in scarlet fever, 40 ; secondary, 349 ; in small-pox, 56 ; treatment of, 348, 349 ; in typhoid fever, 12.

Partial paralysis of sensation, 508 ; in tabes, 641 ; in syringomyelia, 678.

Passive congestion of kidney, 871 ; in pulmonary emphysema, 179 ; in valvular disease of the heart, 300.

Passive congestion of liver, 496 ; in pneumonia, 197 ; in pulmonary emphysema, 179 ; in valvular disease of the heart, 299.

Passive congestion of spleen, in cirrhosis of the liver, 480 ; in pulmonary emphysema, 179 ; in valvular disease of the heart, 300.

Passive motion, sense of, 510 ; of the extremities in hemiplegia, 737 ; in hysteria, 812 ; in spinal paralysis of children, 671.

Patellar reflex, 514 ; absence of, 641 ; in general paralysis of the insane, 763 ; in myelitis, 621 ; in neuritis, 585 ; in pressure paralysis of the spinal cord, 612 ; in pseudo-hypertrophic muscular paralysis, 660 ; in tabes, 644 ; in tetanus, 792.

Pavement epithelium in cancer of the œsophagus, 369.

Pearly distemper in cattle in relation to tuberculosis in man, 208, 209.

Pelioma typhosum, 17.

Peliosis, 961 ; rheumatica, 962 ; senile, 961.

Pelvis in osteomalacia, 926, 927 ; rachitic, 924.

Pelvis of the kidney, dilatation of. See HYDRONEPHROSIS.

Pelvis of the kidney, inflammation of. See PYELITIS.

Pepsine in gastric catarrh, 381.

Percussion, change of pitch on, over cavities, 223.

Perforation, peritonitis from, 449 ; in purpura hæmorrhagica, 962.

Peribronchitis, tubercular, 213.

Pericardial surfaces, adhesion of, 326.

Pericarditis, 321 ; adhesive, 326 ; in articular rheumatism, 904 ; chronic, 323 ; diagnosis of, 327 ; externa, 322, 325 ; fibrinous, 322 ; hæmorrhagic, 322 ; in pneumonia, 197 ; prognosis of, 328 ; in pulmonary tuberculosis, 228; purulent, 322; sero-fibrinous, 322; treatment of, 328; tubercular, 322, 327 ; in valvular disease of the heart, 298.

Pericardium, air in the, 330; blood in the, 330; dropsy of the, 329 ; inflammation of the, 321 ; obliteration of the, 326.

Perichondritis, laryngeal, 134 ; diagnosis of, 135 ; external, 134 ; internal, 134 ; secondary, 135 ; treatment of, 135.

Perinephritic abscess, 870 ; causes of, 870 ; symptoms of, 870 ; treatment of, 871.

Perinephritis, purulent, 870.

Periosteal reflex, 545 ; in cerebral hæmorrhage, 732.

Periostitis ossificans in articular rheumatism, 913.

Peripheral nerves, diseases of, 505 ; forms of paralysis of, 555 ; inflammation of, 579 ; new growths in, 585.

Peripleuritis, 267 ; diagnosis of, 267 ; prognosis of, 267.

Periproctitis, 408.

Peritoneal cancer, 463 ; diagnosis of, 463 ; treatment of, 463.

Peritoneal dropsy, 460.

Peritonitis, acute, 449 ; acute circumscribed, 451, 455 ; adhesive, 451 ; in articular rheumatism, 450, 464 ; cancerous, 463 ; in children, 459 ; in cholelithiasis, 473 ; chronic, 457 ; circumscribed, 451 ; deformans, 458 ; diagnosis of, 456, 458 ; diffuse general, 450 ; fibrino-purulent, 450 ; hæmorrhagic, with formation of hæmatoma, 458 ; in nephritis, 450 ; in pleurisy, 450 ; prognosis of, 456 ; in pulmonary tuberculosis, 227 ; sacculated, 451 ; septic, 451 ; treatment of, 456, 460 ; tubercular, 457 ; in typhoid fever, 10, 11.

Peritonsillar abscess, 354.

Perityphlitis, 418. See also TYPHLITIS.

Peroneal paralysis, 569.

Pertussis, 160. See WHOOPING-COUGH.

Petechial typhus, 28. See TYPHUS.

Peyer's patches in typhoid fever, 9.

Pharyngeal catarrh, chronic, 357 ; hypertrophic, 358 ; prognosis of, 359 ; treatment of, 359.

Pharyngitis, chronic, 357 ; granular, 357 ; sicca, 358.

Pharynx in measles, 47 ; in scarlet fever, 38 ; tuberculosis of, 226 ; in typhoid fever, 12.

Phenacetine, in chorea, 783 ; in headache, 532 ; in migraine, 593 ; in neuralgia, 520 ; in neurasthenia, 818.

Phlebitis, purulent, in septico-pyæmia, 110.

Phloridzine as cause of glycosuria, 966.

Phosphate calculi in nephrolithiasis, 885.

Phosphorus, 648 ; in pernicious anæmia, 945 ; in neuralgia, 520 ; in osteomalacia, 927 ; in rachitis, 925 ; in tabes, 648.
Phosphorus necrosis, 1006.
Phosphorus poisoning, 1006 ; acute, 1006 ; chronic, 1006.
Phrenic nerve, paralysis of, 568.
Phthisis, fibroid, 223.
Phthisis, laryngeal, 136. See TUBERCULOSIS.
Phthisis pulmonalis, 207. See TUBERCULOSIS.
Physostigmine, in chorea, 783.
Piano-player's cramp, 579.
Picric acid in trichinosis, 124.
Pigeon-breast, rachitic, 923.
Pigment calculi in cholelithiasis, 471.
Pigment induration in pulmonary tuberculosis, 214.
Pilocarpine in diphtheria, 72 ; in nephritis, 847.
Pine-needle baths in muscular rheumatism, 919.
Pin-worms, 446.
Piperin in leukæmia, 950.
Pitch, change of, on percussion over cavities, 222.
Pityriasis versicolor in pulmonary tuberculosis, 229.
Plethora, 709.
Pleura, cicatricial contraction of, in pleurisy, 258, 260 ; fistulæ of, after empyema, 264 ; new growths of, 272.
Pleura in leukæmia, 947 ; in pneumothorax, 268 ; in pulmonary tuberculosis, 226.
Pleurisy, 253 ; adhesive, 255 ; in articular rheumatism, 904 ; complications of, 261 ; diagnosis of, 262 ; with effusion, 258 ; fibrinous, 254, 257, 261 ; in pneumonia, 193 ; primary, 253 ; prognosis of, 253 ; purulent, 262 ; secondary, 253 ; in septico-pyæmia, 112 ; in small-pox, 56 ; tapping in, 264 ; treatment of, 264 ; tubercular, 254, 261 ; in typhoid fever, 13.
Pleuritic effusions, 258 ; ossification, 255 ; thickening, 255, 260.
Pleuritis, 253 ; sicca, 254, 257.
Pleuro-pericarditis, 325.
Pleuro-pneumonia, 189.
Plexus paralysis of the brachial plexus, 567.
Pneumatic treatment in bronchitis, 154 ; in emphysema, 181.
Pneumatometer, 178.
Pneumonia alba in pulmonary syphilis of the newborn, 255 ; anomalies in course of, 200 ; asthenic, 201 ; in articular rheumatism, 906 ; bilious, 197 ; catarrhal, 184 ; central, 201 ; cheesy, 187, 213 ; in children, 200 ; chronic interstitial, 165 ; complications of croupous, 193 ; crossed, 191 ; croupous, 189 ; delayed resolution of croupous, 202 ; diagnosis of croupous, 203 ; in diphtheria, 69 ; disposition to croupous, 189 ; endemic occurrence of croupous, 190 ; erysipelatous, 196 ; fibrinous, 190 ; genuine, 189 ; infectious nature of croupous, 189 ; intermitting, 199 ; lobar, 189 ; lobular, 184, 213 ; in measles, 49 ; migrans, 196 ; in nephritis, 843 ; in old people, 200 ; pathological lesion of catarrhal, 185 ; of croupous, 190 ; primary, 189 ; prognosis of croupous, 204 ; in rachitis, 923 ; in scurvy, 959 ; in small-pox, 56 ; symptoms of catarrhal, 186 ; of croupous, 193 ; in tetanus, 792 ; traumatic, 189 ; treatment of catarrhal, 188 ; of croupous, 204 ; typhoid, 201 ; wandering, 196.
Pneumonoconiosis, 245.
Pneumopericardium, 330.
Pneumothorax, 267 ; circumscribed, 268 ; closed, 269 ; diagnosis of, 270 ; open, 269 ; in pulmonary tuberculosis, 226 ; sacculated, 268 ; treatment of, 270 ; in typhoid fever, 13 ; valvular, 270.
Pneumo-typhoid, 13, 202.
Podagra, 984. See GOUT.
Points douloureux in neuralgia, 517.
Poisoning, 1003.
Polioencephalitis, 745.
Poliomyelitis, acute, of adults, 671 ; diagnosis of, 672 ; relation of, to neuritis, 672 ; symptoms of, 671 ; treatment of, 672.
Poliomyelitis, acute, in children 667. See SPINAL PARALYSIS OF CHILDREN.
Poliomyelitis, subacute and chronic, 673 ; treatment of, 674.
Polyæsthesia, 507 ; in tabes, 640.
Polyarthritis, chronic, 911.
Polydipsia in diabetes, 967, 971, 983 ; in hysteria, 805.
Polymyositis, acute, 919.
Polypi in the œsophagus, 366.
Polysarcia adiposa, 992.
Polyuria in anæmia, 934 ; in cerebro-spinal meningitis, 106 ; in contracted kidney, 859 ; in diabetes insipidus, 982–984 ; in diabetes mellitus, 967, 971, 976 ; in epilepsy, 773 ; in hysteria, 805.
Pomegranate in tape-worm, 444.
Pons, hæmorrhages into, 691. See also MEDULLA.
Pork in relation to trichinosis, 122.
Portal vein, purulent inflammation of, 500 (see PYLEPHLEBITIS) ; thrombosis of, 502. See PYLETHROMBOSIS.
Post-epileptic insanity, 774.
Posterior nasal catarrh, chronic, 126.
Potassium, acetate of, in nephritis, 848 ; chlorate of, in cystitis, 896 ; nitrate of, poisoning from, 1004 ; picro-nitrate of, in chyluria, 876 ; salts, in scurvy, 960.
Pott's boss on the vertebral column, 609.
Power, sense of, 510.
Pregnancy in epilepsy, 774 ; nephritis of, 845.
Premature delivery in chorea gravidarum, 784.
Pressure diverticula, 363.
Pressure paralysis of the spinal cord, 608 ; bending of the cord in, 610 ; causes of, 608 ; complications of, 613 ; diagnosis of, 613 ; pathological lesion of the vertebræ and of the cord in, 609 ; place of compression in, 609 ; treatment of, 614 ; trephining for, 614.
Pressure points in facial spasm, 572.
Pressure, sense of, disturbances of, in tabes, 641 ; partial paralysis of, 507 ; test of, 507.
Proctitis, 407.
Professional diseases, 245.
Professional neuroses of co-ordination, 577.
Proglottides of tape-worm, 440.
Propulsion in paralysis agitans, 785.
Prosopalgia, 521. See TRIGEMINUS, NEURALGIA OF.
Prostate in genito-urinary tuberculosis, 888.
Pseudo-crises in croupous pneumonia, 199 ; in relapsing fever, 33.
Pseudo-croup, 131.
Pseudo-hypertrophy of the muscles, 659 ; beginning of, 659 ; increase of volume of different muscles in, 660 ; symptoms of, 659.
Pseudo-leucocythæmia, 951.
Pseudo-leukæmia, 951 ; diagnosis of, 953 ; examination of the blood in, 952 ; lymphatic, 952 ; relation of, to anæmia, 951 ; to infectious tumors, 951 ; symptoms of, 952 ; treatment of, 953.
Pseudo-paralysis, spastic, 664.

Pseudo-relapse in scarlet fever, 44.
Pseudo-sclerosis, 631.
Pseudo-tabes of alcoholic subjects, 584.
Psoriasis of the tongue, 346.
Psychical equivalent of epilepsy, 774.
Ptosis in oculo-motor paralysis, 556.
Pulmonary valve, insufficiency of, 293; stenosis of, 293.
Pulsation, epigastric, in valvular disease of the heart, 284.
Pulsus bigeminus, 298; celer, 289; inequalis, 297; irregularis, 297; paradoxus, 167, 324, 326; tardus, 333.
Puncture in ascites, 462; in chronic hydrocephalus, 769; in cirrhosis of the liver, 483; in hydronephrosis, 892; in nephritis, 849; in pericarditis, 329; in pleurisy, 264; in pneumonia, 196; in pneumothorax, 271; in valvular disease of the heart, 306.
Pupils in epilepsy, 773, 777; in general paralysis of the insane, 763; in hæmatoma of the dura, 697, 698; in meningitis, 700, 704; in pernicious anæmia, 942; in tetanus, 792; in thermic fever, 747.
Pupils, immobility of, in eclampsia of children, 779; in epilepsy, 773, 777; in general paralysis of the insane, 763; in lesions of the corpora quadrigemina, 722; in tabes dorsalis, 636.
Purpura, 961; hæmorrhagica, 962; hæmorrhagica, prognosis and treatment of, 962; rheumatica, 962; simplex, 962; urticans, 962; variolosa, 57.
Pus, collections of, as a cause of septico-pyæmia, 109.
Pustule, malignant, 117. See MALIGNANT PUSTULE.
Pustules in glanders, 116; in small-pox, 53.
Putrefaction, abnormal intestinal, as cause of convulsions, 771.
Pyæmic symptoms in sinus thrombosis, 708; in suppurative pylephlebitis, 501.
Pyelitis, 881; calculosa, 881, 884; in tabes, 643; in myelitis, 621; origin of, 881; symptoms of, 882; treatment of, 884.
Pyelocystitis, 881.
Pyelonephritis, 869, 882.
Pylephlebitis, chronic adhesive, 502.
Pylephlebitis, suppurative, 500; diagnosis of, 500; of the newborn, 500; symptoms of, 500.
Pylethrombosis, 502; symptoms of, 502; treatment of, 503.
Pyonephrosis, 882.
Pyopneumothorax, 268.

Quicksilver in intestinal obstruction, 439.
Quincke's capillary pulse, 289.
Quinine in asthma, 172; in diabetes, 981; in habitual headache, 532; in hæmoglobinuria, 956; in leukæmia, 950; in malaria, 96; in Ménière's disease, 769; in neuralgia, 519; in neurasthenia, 818; in neuroses of the heart, 319; in pulmonary tuberculosis, 235; in sciatica, 520, 527; after scurvy, 961; in trigeminal neuralgia, 520, 523; in trophic disturbances, 590; in typhoid fever, 20, 24, 25; in whooping-cough, 163.

Rabies, 113. See HYDROPHOBIA.
Race, influence of, on diabetes mellitus, 967; on hæmophilia, 963; on hysteria, 799; on yellow fever, 101.
Rachitis, 920; acute, 924; chemical examination of the bones in, 921; chronic, 924; diagnosis and prognosis of, 924; fœtal, 921; origin of, 920; relation of, to spasm of the glottis, 142; symptoms of, 922; tarda, 921; treatment of, 924.
Radial paralysis, 564; chronic thickening of the extensor tendons in, 565; disturbances of function in, 565; in lead paralysis, 570; rheumatic, 565; traumatic, 564.
Radiating fungus, 274.
Rag-picker's disease, 119. See MALIGNANT PUSTULE.
Railway-spine, 572. See TRAUMATIC NEUROSES.
Raynaud's disease, 588; with hæmoglobinuria, 954.
Rectal speculum in cancer of the rectum, 426.
Rectum, cancer of, 426; inflammation of, 407; neuralgia of, 528; paralysis of, in injury of the spinal cord, 607; paralysis of, in myelitis, 621; syphilis of, 425; symptoms of, 425; treatment of, 425.
Recurrent fever. See RELAPSING FEVER.
Recurrent nerve, paralysis of, 139.
Re-enforcement of patellar reflex, 544.
Reflex centers, vaso-motor, 587.
Reflexes, 543; in acute ascending spinal paralysis, 674; after cerebral hæmorrhage, 729, 732; in chorea, 782; in facial paralysis, 561; in injury of the spinal cord, 607; in tabes, 641; in myelitis, 620; in neuralgia, 570; in neuritis, 583, 584; in paralysis, 540; in pressure paralysis of the spinal cord, 612; in progressive bulbar paralysis, 687; in progressive muscular atrophy, 657; in sciatica, 526; in spinal apoplexy, 605; in spinal meningitis, 600; tests and condition of, 543; in tetanus, 792; in trigeminal neuralgia, 531; in tubercular meningitis, 704; in unilateral lesion of the spinal cord, 684.
Reflex epilepsy, 771; neuralgia, 517; paralyses, 537; spasm, saltatory, 575.
Refraction, errors of, as a cause of headache, 530; migraine, 591.
Rehme bath, artificial, in myelitis, 627.
Relapses of cholera, 86; of erysipelas, 64; of lead paralysis, 570; of scarlet fever, 44; of sciatica, 527; of typhlitis, 492; of typhoid fever, 19; of typhus fever, 30.
Relapsing fever, 31; complications of, 35; contagiousness of, 32; epidemic occurrence of, in Germany, 31; inoculation of, 32; period of incubation of, 32; prognosis of, 35; spirilli in, 34; treatment of, 35.
Renal crises in tabes, 643.
Ren mobilis, 876.
Resonance, thoracic, in pleurisy, 259.
Respiration in acute ascending spinal paralysis, 675; in acute bulbar paralysis, 694; amphoric, 269; in amyotrophic lateral sclerosis, 652; in anæmia, 932, 942; in asthma, 168; bronchial, 223; in bronchitis, 148, 152; in bulbar hæmorrhage, 692; in cancer of the lungs, 251; in cerebral hæmorrhage, 729, 732; in chronic poliomyelitis, 673; in cirrhosis of the liver, 481; in diabetic coma, 973; in embolism and thrombosis of the basilar artery, 694; in epilepsy, 773; in exophthalmic goitre, 597; in hepatitis, 477; in hysteria, 803; interrupted, 222; in tabes, 643; metallic, 269; metamorphosing, 222; in miliary tuberculosis, 237, 239; in obesity, 995; in osteomalacia, 927; in phthisis, 222; in pneumonia, 186, 194; in progressive bulbar paralysis, 686; in progressive muscular atrophy, 656, 657, 658; in pseudo-leukæmia lymphatica, 952; in scurvy, 958; in tetanus, 793; in thermic fever, 747; in trichinosis, 122; in tubercular meningitis, 704, 705; in uræmia, 832.
Respiratory spasms, 576; complicated, 577.

Retina in chronic nephritis, 854 ; in contracted kidney, 861 ; in diabetes, 972 ; in leukæmia, 947.
Retinal hæmorrhages in septico-pyæmia, 111.
Retropharyngeal abscess, 360.
Retropulsion in paralysis agitans, 785.
Retro-tonsillar abscess, 354.
Revaccination, 59.
Rhabdomyoma, 872.
Rheumatism, acute articular, 900 ; alkaline treatment of, 910 ; cerebral, 905 ; in chorea, 780 ; chronic, 911 ; diagnosis of, 907 ; endocarditis in, 903 ; hyperpyretic, 905 ; prognosis of, 906 ; prophylaxis of, 911 ; scarlatinal, 43 ; symptoms, 901 ; treatment of, 907, 910.
Rhinitis, 125 ; chronic, 126 ; in scrofula, 1000.
Rickets, 920. See RACHITIS.
Rötheln, 51 ; period of incubation of, 51 ; prognosis of, 51 ; relation of, to measles, 51.
Romberg's symptom in locomotor ataxia, 636.
Root-zones, 510.
Rosary, rachitic, 923.
Rosenthal-Leube meat solution, 389.
Roseola in typhoid fever, 16 ; in typhus fever, 30.
Round-worms, 445.

Sac, pericardial, air in, 330 ; blood in, 330.
Saccharine in diabetes, 980.
Sage-tea in phthisis, 235.
St. Anthony's fire, 61.
St. Vitus's dance, 780.
Salaam convulsions, 574.
Salicylate of sodium in articular rheumatism, 908 ; in diabetes, 981 ; in gout, 992 ; in habitual headache, 532 ; in hemicrania, 593 ; in neuralgia, 520 ; in typhoid fever, 24.
Salicylic acid in articular rheumatism, 907 ; in diseases of the trophic nerves, 590 ; in gastric catarrh, 381 ; in gout, 992 ; in muscular rheumatism, 919 ; in neuritis, 584 ; in purpura hæmorrhagica, 963 ; in tabes, 648 ; in tetanus, 784.
Salicylic delirium, 908 ; dyspnœa, 908 ; powder in phthisis, 235.
Salivation in diabetes insipidus, 983 ; in hydrophobia, 114 ; in stomatitis, 341 ; in ulcerative stomatitis, 342.
Salol in rheumatism, 910.
Salt in epilepsy, 778 ; in hæmoptysis, 235.
Salt-baths in gout, 991 ; in scrofula, 1001.
Saltpeter-paper in asthma, 172.
Sand-baths in articular rheumatism, 916.
Santonica in ascarides, 446.
Santonin in ascarides, 446.
Sarcina ventriculi, 376.
Sarcoma, alveolar, of the lungs, 250 ; of the kidneys, 872.
Scarification of the skin in dropsy, 306.
Scarlatina, 36.
Scarlatinal diphtheria, 40 ; eruption, 41 ; nephritis, 42, 43 ; poison, 36.
Scarlet fever, 36 ; contagiousness of, 36 ; diagnosis of, 44 ; disposition to, 36 ; epidemic occurrence of, 37 ; hæmorrhagic, 41 ; inoculation of, 36 ; miliary, 41 ; papular, 41 ; period of incubation of, 37 ; prognosis of, 44 ; rudimentary forms of, 43 ; tenacity of the contagium of, 36 ; treatment of, 44 ; typhoid form of, 44 ; variegated, 41.
Sciatica in diabetes, 973 ; diagnosis of, 527 ; relapses of, 527 ; symptoms and cause of, 526 ; treatment of, 527.
Sciatic paralysis, 569 ; treatment of, 569.
Scirrhus cancer of the œsophagus, 369 ; of the stomach, 391.
Sclérose en plaques, 627. See SCLEROSIS, MULTIPLE.
Sclerosis, amyotrophic lateral, 650 ; diagnosis of, 653 ; implication of the medulla in, 651 ; symptoms and course of, 651 ; treatment of, 653.
Sclerosis, disseminated, 627. See SCLEROSIS, MULTIPLE.
Sclerosis, multiple, of the brain and cord, 627 ; distinction of, from paralysis agitans, 631 ; hereditary predisposition to, 628 ; relation of, to chronic bulbar paralysis, 631 ; to chronic myelitis, 630 ; to general paralysis of the insane, 631 ; to spastic spinal paralysis, 631 ; seat of the sclerotic nodules in, 627 ; symptoms of, 629 ; treatment of, 631.
Sclerosis, postero-lateral, 649.
Sclerosis, primary lateral, 663.
Sclerosis, renal, 856.
Sclerotinic acid in phthisis, 235.
Scolex, 441.
Scoliosis, rachitic, 923.
Scorbutic anæmia, 959 ; ulcers, 959.
Scorbutus, 956. See SCURVY.
Scrofula, 999 ; relation of, to tuberculosis, 216, 1000 ; treatment of, 1000.
Scurvy, 956 ; causes of, 957 ; contagiousness of, 957 ; distinction of, from peliosis and stomatitis, 960 ; epidemic occurrence of, 957 ; forms of, 960 ; symptoms of, 958 ; treatment of, 960.
Season and chorea, 780.
Seat-worms, 446.
Secondary degeneration. See SPINAL CORD (secondary degeneration).
Secretion, disturbances of, 590 ; in acute ascending spinal paralysis, 674 ; in anæmia, 932 ; in cerebral hæmorrhage, 723 ; in diabetes insipidus, 982 ; in exophthalmic goître, 597 ; in hæmoglobinuria, 954 ; in hysteria, 805; in myelitis, 622; in neurasthenia, 816 ; in progressive bulbar paralysis, 687 ; in subacute poliomyelitis, 673 ; in tetanus, 793 ; in tetany, 790 ;
Secretions in diabetes, 975, 982, 983.
Semilunar valves, insufficiency of, 287.
Senile emphysema, 173.
Senile kidney, 333, 857.
Sensation, conduction of, in acute ascending spinal paralysis, 674 ; delayed, 509.
Sensation, paralysis of, in tabes, 640 ; partial, 505.
Sensibility, disturbances of, in acute ascending spinal paralysis, 675 ; in arsenical paralysis, 571 ; in bulbar hæmorrhage, 692 ; in caisson disease, 608 ; in cerebral hæmorrhage, 733 ; in compression of the medulla, 695 ; in crural paralysis, 568 ; in epilepsy, 772 ; in focal diseases of the crus, 723 ; of the internal capsule, 721 ; general consideration of, 505 ; in general paralysis of the insane, 763 ; in injury of the spinal cord, 607 ; in the larynx, 143 ; in median paralysis, 566 ; in multiple sclerosis, 630 ; in myelitis, 619 ; in neuralgia, 511 ; in neuritis, 584, 585 ; in obturator paralysis, 569 ; in paralysis, 539 ; in pressure paralysis of the spinal cord, 612 ; in radial paralysis, 565 ; in sciatica, 526 ; in sciatic paralysis, 569 ; in syringomyelia, 678 ; in tabes, 639 ; in tetanus, 792 ; in tumors of the base of the brain, 752 ; in ulnar paralysis, 566 ; in unilateral lesion of the spinal cord, 688 ; in writer's cramp, 578.
Sensory centers, 715.
Sensory nerves, diseases of, 505.

Septico-pyæmia, 108; circulatory apparatus in, 111; cryptogenetic or spontaneous, 109; diagnosis of, 112; jaundice of the skin in, 112; prognosis of, 112; treatment of, 113.

Sero-pneumothorax, 267.

Serous membranes in articular rheumatism, 904.

Serratus paralysis, 563; course of, 564; rheumatic, 563; traumatic, 563; treatment of, 564; wing-like position of the scapula in, 564.

Serum albumen in albuminuria, 823.

Sewing-machine girls, affection of, 579.

Sex, influence of, in acute ascending spinal paralysis, 674; in acute yellow atrophy of the liver, 485; in amyotrophic lateral sclerosis, 650; in anæmia, 929, 934, 940; in cerebral hæmorrhage, 726; in cerebral syphilis, 757; in cerebral tumor, 748; in cholera, 83; in chorea, 781; in congenital myotonia, 795; in diabetes, 967; in exophthalmic goitre, 595; in Friedreich's ataxia, 648; in general paralysis of the insane, 761; in gout, 985; in hæmatoma of the dura mater, 697; in hæmophilia, 963; in hepatic colic, 470; in hemicrania, 591; in hysteria, 799; in leukæmia, 946; in multiple sclerosis, 628; in neuralgia, 516; in obesity, 994; in osteomalacia, 925; in paralysis agitans, 784; in phthisis, 211; in poliomyelitis, acute, of adults, 671; in pseudo-hypertrophic muscular paralysis, 659; in pseudo-leukæmia, 952; in rachitis, 921; in tabes, 633; in tetany, 789; in unilateral facial atrophy, 594.

Sexual excess, as cause of myelitis, 616.

Sexual functions in diabetes, 972; in functional disturbances of the spinal cord, 606; in injuries of the spinal cord, 607; in myelitis, 621; in tabes, 643.

Sexual organs in chlorosis, 929; in diabetes, 972; diphtheria of, 69; in exophthalmic goitre, 595; in gout, 987; in hysteria, 800; neuralgia of, 528; in neurasthenia, 816; (female) in peritontis, 450.

Shadows of red blood-corpuscles in hæmoglobinuria, 955.

Shaking, 540.

Shaking palsy, 784.

Ship fever, 28. See TYPHUS FEVER.

Shoulder, muscles of, paralysis of, 562; paralysis of, unilateral, in cerebral hæmorrhage, 732; spasm of, 575.

Siderosis pulmonum, 245.

Silver, nitrate of, in chorea, 783; in dysentery, 80; in intestinal catarrh of children, 417; in tabes, 648; in myelitis, 627; in spastic spinal paralysis, 667; in valvular disease of the heart, 303.

Singultus, 577. See HICCOUGH.

Siphon, in washing out the stomach, 399.

Skin, care of, in diabetes, 980; character of, in arthritis deformans, 915; itching of, in uræmia, 833.

Skin, diseases of, in articular rheumatism, 904; in dengue, 99; in diabetes, 972; in gout, 987; in pneumonia, 198; in scarlet fever, 41; in scrofula, 999; in scurvy, 958; in small-pox, 55; in trichinosis, 123; in typhoid fever, 16.

Skoda's resonance in pleuritic effusion, 259.

Sleeplessness in neurasthenia, 816.

Small-pox, 52; confluent, 53; contagiousness of, 52; diagnosis of, 57; hæmorrhagic, 57; mortality in, 57; period of incubation in, 53; prognosis of, 57; treatment of, 58.

Smell, sense of, anæsthesia of, 532; anomalies of, 532; in epilepsy, 772; hyperæsthesia of, 532; in hysteria, 801; relation of, to anomalies of taste, 533; subjective sensations of, 532; test of, 532; treatment of anomalies of, 533.

Sneezing spasm, 577.

Sodic bicarbonate in diabetic coma, 981; bicarbonate in gastric catarrh, 382; carbonate in nephrolithiasis, 887; hydrate in sulphuric-acid poisoning, 1003; phosphate in nephrolithiasis, 887; sulphate in carbolic-acid poisoning, 1008.

Soil theory in relation to typhoid fever, 3.

Soil water in relation to cholera, 83; in relation to typhoid fever, 2, 3.

Somnambulism in hysteria, 809.

Soor, 344.

Sore throat, 351 (see TONSILLITIS): catarrhal, 352; follicular, 353; hæmorrhagic, 354; necrotic, 354; parenchymatous, 353; phlegmonous, 354; in scarlet fever, 38; in small-pox, 56; in typhoid fever, 12.

Soul-blindness, 716.

Soul-deafness, 716.

Southey's trocar in dropsy, 306.

Spasm, 539 (see CONVULSIONS); of the cervical muscles, 573; clonic, 540; co-ordinated, 541; of the facial nerves, 572; forms of localized, 540; of the muscles of the lower extremity, 575; of the œsophagus, 371; of the respiratory muscles, 576; tonic, 540, 541; tonic-clonic, 540.

Spasm of the glottis, 142. See GLOTTIS, SPASM OF.

Spastic-paretic gait in multiple sclerosis, 630; in spastic spinal paralysis, 664; in syringomyelia, 678.

Speech, disturbances of, 716; in acute bulbar myelitis, 694; in amyotrophic lateral sclerosis, 652; in athetosis, 787; in bulbar hæmorrhage, 692; in cerebral embolism, 740; in cerebral hæmorrhage, 728, 736; in cerebral syphilis, 759; in cerebral tumors, 750, 752; in chorea, 781; in compression of the medulla, 695; in embolism and thrombosis of the basilar artery, 694; in epilepsy, 772; in focal diseases of the centrum ovale, 720; in general paralysis of the insane, 762, 766; in hæmatoma of the dura, 697; in hereditary ataxia, 649; in meningitis, 704; in multiple sclerosis, 629; in progressive bulbar paralysis, 685, 687.

Sphincter, reflex spasm of, in cystitis, 894.

Spina bifida, 679; complication of, with purulent meningitis, 680; seat of, 679; surgical treatment of, 680; tumor formation in, 680.

Spinal apoplexy, 604; symptoms of, 605; treatment of, 605.

Spinal cord, anæmia of, 604.

Spinal cord, cavity and fissure formation in, 677; in cerebro-spinal meningitis, 104, 105; circulatory disturbances of, 604; consumption of, 632; diffuse diseases of, 615; diseases of, 599; functional disturbances of, 605; functions of, 624; in gout, 988; hæmorrhages into, 604; new growths of, 676; systemic diseases of, 615; traumatic lesions of, 606.

Spinal cord, compression of, 608; origin of, 608; seat of, 609; in spina bifida, 680.

Spinal cord, concussion of. See TRAUMATIC NEUROSES.

Spinal cord, degeneration of, in amyotrophic lateral sclerosis, 650.

Spinal cord, disease of, after sudden lowering of atmospheric pressure, 608.

Spinal cord, hyperæmia of, 604.

Spinal cord, injuries of, 606; complication of, with secondary inflammation, 607; symptoms of, 607; treatment of, 607.

Spinal cord, membranes of, acute inflammation of, 599; hæmorrhages of, 603; new growths of, 676.

Spinal cord, secondary degeneration of, 680; after cerebral hæmorrhage, 732, 734; after cerebral lesions, 680; in compression of the cord, 610; in multiple sclerosis, 628; in transverse affections of the cord, 681; in tumors of the cord, 677.

Spinal cord, softening of, 618.

Spinal cord, tumors of, 676; differential diagnosis of, from transverse myelitis, 677; forms of, 676; origin of, 676; prognosis and treatment of, 677; relation of, to unilateral lesion of the spinal cord, 677.

Spinal irritation, 606; in hysteria, 802; in neurasthenia, 815.

Spinal meningitis, 599, 601; origin of, 599, 601; prognosis of, 600; symptoms of, 600, 601; treatment of, 601.

Spinal muscles, paralysis of, 564; in pseudo-hypertrophic spinal paralysis, 564, 660.

Spinal neurasthenia, 605; diagnosis of, 606; sensitiveness of the vertebræ in, 606; symptoms of, 606.

Spinal paralysis, acute ascending, 674; acute infection in, 675; diagnosis and prognosis of, 676; symptoms of, 674; treatment of, 676.

Spinal paralysis, atrophic, 671.

Spinal paralysis of children, 667 (see INFANTILE PARALYSIS); from acute infection, 667; diagnosis and prognosis of, 670; relation of, to primary neuritis, 668; spinal cord in, 667; symptoms of, 668; treatment of, 670.

Spinal paralysis, spastic, 663; diagnosis of, 666; pathological lesion in the cord in, 665; relation of, to chronic hydrocephalus, 665; treatment of, 666.

Spine, stiffness of, in sinus thrombosis, 707; (tonic) in tetanus, 792; in tubercular meningitis, 703.

Spirilli in relapsing fever, 34.

Spirometer in pulmonary emphysema, 178.

Spleen in acute ascending spinal paralysis, 675; in acute yellow atrophy of the liver, 486; in Addison's disease, 879; in articular rheumatism, 906; in cerebro-spinal meningitis, 106; in cirrhosis of the liver, 480; in erysipelas, 64; in hæmoglobinuria, 956; in hepatic syphilis, 491; in leukæmia, 946; in malaria, 94, 95, 96; in miliary tuberculosis, 239; in pernicious anæmia, 940; in phthisis, 228; in pneumonia, 197; in pseudo-leukæmia, 952; in pylephlebitis, 501; in rachitis, 922; in relapsing fever, 32; in scarlet fever, 37, 43; in scurvy, 959; in septico-pyæmia, 109, 112; in tubercular meningitis, 705; in typhoid fever, 11; in typhus fever, 30; in yellow fever, 101.

Spleen, extirpation of, in leukæmia, 951.

Splenic fever, 117.

Splenization of the lung in atelectasis, 181.

Splint-rest in sciatica, 528.

Spondylitis deformans in chronic articular rheumatism, 914.

Spoonwort in scurvy, 960.

Spotted fever, 28, 103. See TYPHUS FEVER and CEREBRO-SPINAL MENINGITIS.

Stasis, œdema from, in renal diseases, 829.

Status epilepticus, 774, 775.

Stenocardia in hysteria, 761; in valvular disease of the heart, 297.

Stenson's experiment in spinal anæmia, 604.

Stimulants in cholera morbus, 412; in pneumonia, 206; in tetanus, 794.

Stitch in the side in pleurisy, 256; in pneumonia, 192.

Stomacace, 342. See STOMATITIS.

Stomach. See also GASTRIC.

Stomach, adhesions of, 387; in chlorosis, 935; in dysentery, 79; in erysipelas, 63; perforation of, 387, 394; in phthisis, 227; in pulmonary gangrene, 243; in purpura hæmorrhagica, 962; purulent inflammation of, 383; in yellow fever, 101.

Stomach, abscess of, 383.

Stomach-bougie in dilatation, 398.

Stomach, cancer of, 391.

Stomach, dilatation of, 396.

Stomach, hæmorrhage from, 374; in gastric cancer, 392; in gastric ulcer, 386.

Stomach, nervous affections of, 400; diagnosis of, 402; in hysteria, 805; nervous complications of, 400; peristaltic disturbance of the stomach in, 400; prognosis and treatment of, 402.

Stomach-pump in chronic gastric catarrh, 377; in dilatation of the stomach, 399.

Stomach, softening of, 358.

Stomach, ulcer of, 384.

Stomachics in gastric catarrh, 382; in jaundice, 468; in neurasthenia, 818.

Stomatitis, 341; acute, 342; aphthous, 343; chronic, 342; in diabetes, 971; mercurial, 341; in scarlet fever, 39; scorbutic, 958; treatment of, 342, 343, 344; in typhoid fever, 12; ulcerative, 342.

Stonecutters' lung, 245.

Strabismus convergens in abducens paralysis, 556; in sinus thrombosis, 707.

Stramonium cigarettes in asthma, 172.

Strangulation, internal, 433.

Stricture of the intestine, cicatricial, 432.

Stripe-pneumonia, 189.

Stroke, apoplectic, 728.

Strongylus duodenalis, 448.

Strongylus gigas, 875.

Strophanthus in contracted kidney, 864.

Struma in exophthalmic goître, 596; extirpation of, 598; of the supra-renal capsules, 879.

Strychnine in acute bulbar paralysis, 695; in cerebral hæmorrhage, 737; in diphtheria, 73; in facial paralysis, 562; in laryngeal paralysis, 142; in myelitis, 627; in neuritis, 585; in oculo-motor paralysis, 557; in progressive muscular atrophy, 658; in spinal paralysis of children, 671.

Strychnine poisoning, 1008.

Subsultus tendinum in typhoid fever, 14.

Succession of Hippocrates in pyo-pneumothorax, 269.

Sudamina in articular rheumatism, 904.

Sugar formation in diabetes, 970; influence of febrile diseases on, 971; influence of mental excitement on, 970; influence of physical exertion on, 970.

Suggestion in catalepsy, 797; in hysteria, 808.

Sulphonal in chorea, 783; in neurasthenia, 819.

Sulphur in anæmia, 938; in hæmorrhoids, 429.

Sulphur baths in lead paralysis, 570.

Sulphuretted hydrogen poisoning, 1007.

Sulphuric acid in purpura hæmorrhagica, 962.

Sulphuric-acid poisoning, 1003.

Sulphurous-acid poisoning, 1003.

Sunstroke, 746; causes of, 746; symptoms of, 746; treatment of, 747.

Support, mechanical, in spasm of the cervical muscles, 575; for the vertebral column in pressure paralysis of the spinal cord, 614.

Suppositories in dysentery, 80.

Suppuration as a cause of arthritis, 912.

Suppurative fever in small-pox, 55.
Supra-renal capsules, diseases of, 878.
Suspension in tabes, 648.
Swamp fever, 90. See Malaria.
Sweat-glands in jaundice, 467 ; in typhoid fever, 17 ; in uræmia, 832.
Sweating in articular rheumatism, 904 ; in diabetes, 972 ; in hæmoglobinæmia, 954 ; in phthisis, 226, 229 ; in trichinosis, 123.
Sympathetic, irritation of, 591 ; in exophthalmic goître, 597 ; trophic disturbances in, 591 ; paralysis of, 591 ; contraction of the pupils in, 591 ; in exophthalmic goître, 597 ; in hemicrania, 591 ; vasomotor disturbances in, 590.
Synovitis in articular rheumatism, 902 ; scarlatinal, 43.
Syphilis as cause of tabes, 632.
Syphilis of the rectum, 425.
Syphiloma, formation of, in syphilis of the liver, 490.
Syringomyelia, 677 ; extent of, 678 ; origin of, 678.

Tabes dorsalis, 632 ; ataxic stage of, 636 ; cause of anæsthesia in, 641 ; development of, 632 ; diagnosis and prognosis of, 645 ; Friedreich's form of, 648 ; hereditary predisposition to, 632 ; histology of, 633 ; in chronic ergotism, 633 ; initial stage of, 635 ; involving cranial nerves, 642 ; partial paralysis of sensation in, 641 ; peripheral degeneration in, 634 ; peripheral paralysis in, 639 ; persistence of patellar reflex in, 641 ; pupillary changes in, 642 ; suspension in, 648 ; symptoms of, 636 ; syphilis as cause of, 632 ; terminal stage of, 636 ; treatment of, 646 ; trophic disturbances in, 644 ; with regard to progressive general paralysis, 645.
Tabes mesenterica, 459.
Tabes, motor, 654. See Progressive Muscular Atrophy.
Tabes spastica, 663. See Lateral Sclerosis.
Taches bleuâtres, 17.
Taches cérébrales, 705.
Tachycardia, 320.
Tactile circles, 506.
Tactile compasses, 506.
Tænia echinococcus, 494 ; medio-canellata, 441 ; saginata, 441 ; solium, 440.
Tailor's cramp, 579.
Tampons in nasal catarrh, 128.
Tannin in chronic pharyngeal catarrh, 359 ; in nephritis, 846.
Tape-worms, 440 ; cures for, 443.
Taste, disturbances of, 533 ; central, 533 ; diagnosis of, 534 ; in epilepsy, 772 ; in facial paralysis, 559 ; in hysteria, 801 ; partial, 533 ; test of, 533 ; treatment of, 534.
Teeth, anomalies of, in diabetes, 971 ; in rachitis, 923.
Teething, 350 ; convulsions in, 351.
Telegrapher's cramp, 579.
Temperature, sense of, in paralysis agitans, 786 ; partial paralysis of, 508 ; perverse, 508 ; in syringomyelia, 678 ; in tabes, 641 ; test of, 508.
Temporal convolutions, focal diseases of, 716 ; relation of, to deafness, 716 ; relation of, to word-deafness, 716, 719 ; seat of the cortical center for hearing in, 716.
Tendinous spots on the pericardium, 323.
Tendon reflexes, 544 ; absence of, 545 ; in amyotrophic lateral sclerosis, 652 ; in cerebral hæmorrhage, 732 ; in cerebral paralysis of children, 745 ; in chronic hydrocephalus, 768 ; in epilepsy, 773 ; increase of, 545 ; in the lower extremities, 544 ; in multiple sclerosis, 629 ; in myelitis, 620 ; in poliomyelitis of adults, 672 ; in pressure paralysis of the spinal cord, 612 ; in progressive bulbar paralysis, 687 ; in secondary degeneration of the spinal cord, 682 ; in spastic spinal paralysis, 663, 665 ; in spinal paralysis of children, 669 ; in tabes, 641 ; in unilateral lesion of the spinal cord, 684 ; in the upper extremities, 545.
Tendons, sheaths of, in articular rheumatism, 902 ; thickening of, in radial paralysis, 565.
Tenesmus in dysentery, 78 ; in intestinal catarrh, 407.
Terminal phalanges, thickening of, in pulmonary stenosis, 293.
Testicles in genito-urinary tuberculosis, 889.
Tetanilla. See Tetany.
Tetanus, 791 ; bacilli of, 791 ; diagnosis of, 793 ; distinction of, from hydrophobia, 794 ; from meningitis, 794 ; from strychnine poisoning, 794 ; endemic and epidemic, 791 ; hydrophobic, 792 ; idiopathic, 791 ; influence of external conditions on, 791 ; intermittent (see Tetany), 789 ; neonatorum, 791 ; paroxysms of, 792 ; prodromal symptoms of, 791 ; rheumatic, 791 ; symptoms of, 792 ; toxines of traumatic, 791 ; treatment of, 794.
Tetany, 789 ; diagnosis of, 790 ; distinction of, from ergotism, 790 ; from professional neuroses, 790 ; epidemic, 789 ; origin of, 789 ; symptoms of, 789 ; treatment of, 791.
Thermæsthesiometer, 508.
Thermic fever, 746 ; pathology of, 746 ; symptoms of, 746 ; treatment of, 747.
Thermo-cautery in noma, 347 ; in pressure paralysis of the spinal cord, 614.
Thomsen's disease, 795. See Myotonia.
Thoracic aorta, aneurism of, 334 ; causes of, 334 ; diagnosis of, 337 ; prognosis of, 337 ; symptoms of, 335 ; treatment of, 338.
Thoracotomy, 266.
Thorax, barrel-shaped, 177 ; compression of, in pulmonary emphysema, 180 ; deformity of, in osteomalacia, 926 ; in rachitis, 923 ; phthisical, 221 ; in pleurisy, 258 ; rigid dilatation of, 175.
Thrombi in typhoid fever, 16 ; in valvular disease of the heart, 300.
Thrombosis of the portal vein, 502.
Thrush, 344.
Thyroid gland, atrophy of, in myxœdema, 589 ; extirpation of, for exophthalmic goître, 598 ; relation of, to exophthalmic goître, 597.
Tibialis paralysis, 569.
Tic convulsif, 572, 573 (see Spasm, Facial) ; douloureux, 521 (see Trigeminus, Neuralgia of) ; rotatoire, 574 (see Spasm of the Cervical Muscles).
Tissue metamorphosis, anomalies of, 928 ; in anæmia, 933 ; in diabetes mellitus, 970.
Tissue, necrosis of, in gout, 988.
Toe-drop in peroneal paralysis, 569.
Tongue, atrophy of, in amyotrophic lateral sclerosis, 652 ; in progressive bulbar paralysis, 685 ; in progressive muscular atrophy, 657.
Tongue, injuries to, in epilepsy, 773.
Tongue, paralysis of, in acute bulbar paralysis, 694 ; in bulbar hæmorrhage, 692 ; (unilateral) in cerebral hæmorrhage, 731 ; in compression of the medulla, 695 ; in embolism and thrombosis of the basilar artery, 694.

Tongue, spasm of, 573.

Tongue, ulcer on the frænum of, in whooping-cough, 161.

Tonics in scurvy, 960.

Tonsillitis, 351 (see SORE THROAT); diagnosis of, 355; follicular, 353; necrotic, 354; parenchymatous, 353; treatment of, 356.

Tonsils, abscess of, 353; chronic hypertrophy of, 356; treatment of, 356; extirpation of, 356; in leukæmia, 947; in pseudo-leukæmia, 952.

Torpid habitus, 1000.

Torticollis, rheumatic, 574; spastic, 574.

Touch, sense of, 506; condition of, 619; diminished in the tongue in facial paralysis, 559; in tabes, 640; test of, 506.

Tracheal catarrh, 146; stenosis, 166.

Tracheitis, 146.

Tracheotomy in diphtheria, 72; in œdema of the glottis, 136; in perichondritis, 135.

Traction diverticula in the œsophagus, 364.

Transfer in hysteria, 814.

Transudation in diabetes, 974; in leukæmia, 949.

Transverse myelitis, 615. See MYELITIS.

Traube's double sound in aortic insufficiency, 289.

Traumatic hysteria, 820.

Traumatic lumbago, 821, 918.

Traumatic neurasthenia, 821.

Traumatic neuroses, 819; cause of, 821; implication of brain in, 820; local, 820; origin of, after railway accidents, 819; relation of, to hysteria, 821; symptoms of, 819; treatment of, 820.

Tremor, 540; alcoholic, 540; in amyotrophic lateral sclerosis, 651; in epilepsy, 775; essential, 540; in exophthalmic goître, 596; hysterical, 541; intention, 540; mercurial, 1005; in multiple sclerosis, 629; in osteomalacia, 927; in paralysis agitans, 784; senile, 540; in typhoid fever, 14.

Trephining the skull in cerebral abscess, 743; in cerebral tumor, 756; in hydatids, 756; in meningeal hæmorrhage, 603; in pressure paralysis, 614; in traumatic epilepsy, 777; in tumors of the cord, 677; the vertebral column in injuries to the cord, 607.

Triceps paralysis, 565.

Trichina spiralis, 121.

Trichinosis, 121; treatment of, 123.

Tricocephalus dispar, 449.

Tricuspid insufficiency, 291

Trigeminus, anæsthesia of, 513; neuroparalytic ophthalmia in, 513; occlusive bandage in, 515; skin of the face in, 514; in tabes, 641.

Trigeminus, neuralgia of, 521; in compression of the medulla, 695; in diabetes, 973; diagnosis of, 522; epileptiform, 522; infra-maxillary, 522; infra-orbital, 522; ligature of the carotid for, 523; operative treatment of, 523; prognosis of, 522; supra-orbital, 522; symptoms and course of, 521; treatment of, 522.

Trigeminus, paralysis of, 557; in bulbar hæmorrhage, 692; in progressive bulbar paralysis, 687.

Trigeminus, spasm of, 571; treatment of, 571.

Trismus, 571; artificial feeding in, 572; in cerebrospinal meningitis, 105; in tetanus, 792; in tubercular meningitis, 705.

Trochlear paralysis, 556.

Trophic disturbances, 587; in arsenical paralysis, 571; of the bones and joints, 580; in cerebral hæmorrhage, 735; in cervico-brachial neuralgia, 524; in cutaneous anæsthesia, 512; in facial hemiatrophy, 594; of the hair and nails, 589; in intercostal neuralgia, 525; in median paralysis, 566; in myelitis, 622; in neuralgia, 517; in neuritis, 583; in occipital neuralgia, 523; in paralysis, 538; in pressure paralysis of the spinal cord, 612; in progressive muscular atrophy, 655; in pseudo-hypertrophic muscular paralysis, 659; in sciatic paralysis, 569; of the skin, 588; in spinal paralysis of children, 669; in syringomyelia, 678; in tabes, 644; treatment of, 590; in trigeminal anæsthesia, 514; in trigeminal neuralgia, 522.

Trousseau's phenomenon in tetany, 790.

Trousseau's spots in tubercular meningitis, 705.

Tubercle bacilli, 208; detection of, 220.

Tubercula dolorosa of the peripheral nerves, 586.

Tuberculosis, 207. See also MILIARY TUBERCULOSIS.

Tuberculosis of the genito-urinary apparatus, 888; diagnosis of, 890; prognosis and treatment of, 890.

Tuberculosis of the intestines, 423; treatment of, 424.

Tuberculosis of the larynx, 136; diagnosis of, 137; treatment of, 138.

Tuberculosis of the lungs, 207; causes of, 208; complications of, 218; diagnosis of, 229; heredity of, 211; infectiousness of, 209; local, 212; physical examination in, 221; predisposition to, 210; prognosis of, 230; prophylaxis of, 231; symptoms of, 218; treatment of, 231.

Tuberculosis of the peritoneum, 457; of the pharynx, 226; of the serous membranes, 261, 327, 457; of the supra-renal capsules, 879.

Turpentine, inhalations of, in asthma, 172; in whooping-cough, 163.

Turpentine, oil of, in acute phosphorus poisoning, 1006; in cystitis, 896; in fœtid bronchitis, 158; in genito-urinary tuberculosis, 890; in sciatica, 527; in tape-worm, 444.

Turpentine pipes in bronchitis, 154.

Tussis convulsiva, 160. See WHOOPING-COUGH.

Tylosis of the tongue, 346.

Typhlitis, 418; diagnosis of, 420; prognosis of, 421; treatment of, 421.

Typhoid, bilious, 35. See also RELAPSING FEVER.

Typhoid fever, 1; abortive, 18; bacilli of, 1; baths in, 22; in children, 19; contagiousness of, 2; in the corpulent, 19; diagnosis of, 20; disinfection in, 26; disposition to, 4; in drunkards, 19; immunity toward, 4; influence of age upon, 4; influence of season upon, 5; in old people, 19; outcry of children in, 19; peculiarities in the course of, 18; period of incubation of, 5; prodromal symptoms of, 5; prognosis of, 20; prophylaxis of, 26; recurrent fever-attack in, 8; relapses of, 19; relapses of, duration of, 19; relapses of, frequency of, 20; sopor in children in, 19; temperature curve in, 6; treatment of, 20; walking, 16.

Typho-malarial fever, 98.

Typhus abdominalis, 1 (see TYPHOID FEVER); levissimus, 18.

Typhus exanthematicus, 28 (see TYPHUS FEVER); levissimus, 31.

Typhus fever, 28; contagiousness of, 29; diagnosis of, 31; distinction of, from typhoid fever, 31; epidemic occurrence of, 29; immunity toward, 29; period of incubation of, 29; prodromal symptoms of, 29; prognosis of, 31; treatment of, 31.

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